

William J. Benjamin

Borish's Clinical Refraction



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BORISH'S CLINICAL REFRACTION, SECOND EDITION

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Contributors

JOHN F. AMOS, OD, MS

Professor, Dean, School of Optometry
University of Alabama at Birmingham
Birmingham, Alabama

IAN L. BAILEY, OD, DSc, FCO, FAAO

Professor of Optometry and Vision Science
School of Optometry
University of California, Berkeley
Berkeley, California

WILLIAM J. BENJAMIN, OD, MS, PhD

Professor of Optometry and Vision Science
School of Optometry
University of Alabama at Birmingham
Birmingham, Alabama

IRVIN M. BORISH, OD, DOS, LLD, DSc

Professor Emeritus, Indiana University
Former Benedict Professor, University of Houston
Boca Raton, Florida

CHARLES E. CAMPBELL

Consultant
Berkeley, California

FREDDY W. CHANG, MSc, OD, PhD

Professor, Department of Optometry
Southern College of Optometry
Memphis, Tennessee

B. RALPH CHOU, MSc, OD, FAAO

Associate Professor, School of Optometry
University of Waterloo
Waterloo, Canada

MELISSA W. CHUN, OD, FAAO

Associate Clinical Professor
Department of Ophthalmology
Jules Stein Eye Institute
David Geffen School of Medicine at UCLA
University of California, Los Angeles
Los Angeles, California

KENNETH J. CIUFFREDA, OD, PhD

Chair and Distinguished Teaching Professor
Department of Vision Sciences
State College of Optometry
State University of New York
New York, New York

CHARLES D. COE, OD, PhD

Lieutenant Colonel, U.S. Army
Walter Reed Army Medical Center
Washington, D.C.

GEORGE W. COMER, OD, MBA

Associate Professor, Primary Eye Care Service
Department of Clinical Sciences
Southern California College of Optometry
Fullerton, California

CHARLES G. CONNOR, PhD, OD

Professor and Director of Research
Southern College of Optometry
Memphis, Tennessee

KENT M. DAUM, OD, PhD

Associate Professor of Optometry
School of Optometry
University of Alabama at Birmingham
Birmingham, Alabama

DAWN K. DeCARLO, OD, MS

Associate Professor and Director
UAB Center for Low Vision Rehabilitation
Department of Ophthalmology
University of Alabama at Birmingham
Birmingham, Alabama

DAVID B. ELLIOTT, PhD, MCOptom, FAAO

Head of Department, Professor
Department of Optometry, University of Bradford
Yorkshire, United Kingdom

JOSEPH B. FLEMING, OD

Associate Professor and Director of Clinical Programs
School of Optometry
University of Alabama at Birmingham
Birmingham, Alabama

LISA BARNHART FOX, OD

Eye Care Practitioner
Charlotte, North Carolina

MARCELA G. FRAZIER, OD, FAAO

Assistant Professor, School of Optometry
University of Alabama at Birmingham
Birmingham, Alabama

ADAM GORDON, OD, MPH

Clinical Associate Professor
Director, Optical Services
School of Optometry
University of Alabama at Birmingham
Birmingham, Alabama

DAVID A. GOSS, OD, PhD

Professor
School of Optometry
Indiana University
Bloomington, Indiana

CHARLES L. HAINE, OD, MS

Vice President for Academic Affairs
Southern College of Optometry
Memphis, Tennessee
Staff Optometrist
Kennedy VA Medical Center
Memphis, Tennessee

NIKOLE L. HIMEBAUGH, OD

Indiana University
School of Optometry
Bloomington, Indiana

DOUGLAS G. HORNER, OD, PhD, FAAO

Associate Professor
School of Optometry
Indiana University
Bloomington, Indiana

HOWARD C. HOWLAND, MS, PhD

Professor
Department of Neurobiology and Behavior
Cornell University
Ithaca, New York

KARIN JOHNSON, OD, DO

Geriatric Physician
OMNI Medical Group
Tulsa, Oklahoma

JENNIE Y. KAGEYAMA, OD, FAAO

Optometrist
Vision Rehabilitation Center, Retina Division
Jules Stein Eye Institute
University of California, Los Angeles
Los Angeles, California

THOMAS R. KARKKAINEN, OD, MS, FAAO

Manager, Applied Optical Products Clinic
Department of Research and Development
Senior Research Optometrist
Vistakon, a Division of Johnson & Johnson Vision Care, Inc.
Jacksonville, Florida

MARJEAN A. TAYLOR KULP, OD, MS, FAAO

Associate Professor
College of Optometry
The Ohio State University
Columbus, Ohio

RICHARD LONDON, MA, OD, FAAO

Professor, College of Optometry
Pacific University
Forest Grove, Oregon

TERESA A. LOWE, OD, FAAO

Assistant Clinical Professor
Department of Clinical Sciences
State College of Optometry
State University of New York
New York, New York

GERALD E. LOWTHER, OD, PhD

Dean and Professor, School of Optometry
Indiana University
Bloomington, Indiana

WENDY L. MARSH-TOOTLE, OD, MS

Associate Professor, School of Optometry
Scientist, Vision Science Research Center
University of Alabama at Birmingham
Birmingham, Alabama

GLEN L. McCORMACK, OD, PhD

Professor of Physiological Optics
Department of Visual Sciences
New England College of Optometry
Boston, Massachusetts

WILLIAM L. MILLER, OD, MS, PhD

Assistant Professor, College of Optometry
University of Houston
Houston, Texas

DONALD O. MUTTI, OD, PhD

Associate Professor
College of Optometry
The Ohio State University
Columbus, Ohio

JAMES M. NEWMAN, MS, OD, FAAO

Clinical Professor, Director of Academic Support Services
School of Optometry
Southern College of Optometry
Memphis, Tennessee

PAUL L. PEASE, OD, PhD

Professor Emeritus, College of Optometry
University of Houston
Houston, Texas

C. DENISE PENSYL, OD, MS, FAAO

Chief of Optometry
Bakersfield VA Outpatient Clinic
Greater Los Angeles VA Healthcare System
Bakersfield, California

DONALD G. PITTS, OD, PhD

Professor Emeritus
University of Houston
Houston, Texas

MICHAEL POLASKY, OD

Former Assistant Dean and Professor
College of Optometry
The Ohio State University
Columbus, Ohio

THOMAS W. RAASCH, OD, PhD

Associate Professor, College of Optometry
The Ohio State University
Columbus, Ohio

WILLIAM H. RIDDER III, OD, PhD

Professor
Department of Basic and Clinical Science
Southern California College of Optometry
Fullerton, California

MARK ROSENFIELD, MCOptom, PhD

Associate Professor
Department of Vision Sciences
State College of Optometry
State University of New York
New York, New York

J. JAMES SALADIN, OD, PhD

Professor, Michigan College of Optometry
Ferris State University
Big Rapids, Michigan

THOMAS O. SALMON, OD, PhD, FAAO

Associate Professor, Oklahoma College of Optometry
Northeastern State University
College of Optometry
Tahlequah, Oklahoma

LISA L. SCHIFANELLA, OD, MS

Clinical Associate Professor
School of Optometry
University of Alabama at Birmingham
Birmingham, Alabama

LEO P. SEMES, OD

Associate Professor, Department of Optometry
School of Optometry
University of Alabama at Birmingham
Birmingham, Alabama

JOHN B. SIEGFRIED, PhD

Professor of Physiological Optics
Pennsylvania College of Optometry
Elkins Park, Pennsylvania

P. SARITA SONI, OD, MS

Associate Vice President for Research
Co-Director, Borish Center for Ophthalmic Research
Professor of Optometry
School of Optometry, Indiana University
Bloomington, Indiana

GREGORY L. STEPHENS, OD, PhD

Associate Professor, College of Optometry
University of Houston
Houston, Texas

MARK W. SWANSON, OD, MS, FAAO

Associate Professor
School of Optometry
University of Alabama at Birmingham
Birmingham, Alabama

LORETTA SZCZOTKA-FLYNN, OD, MS

Associate Professor
Department of Ophthalmology
Case Western Reserve University
Cleveland, Ohio
Director
Contact Lens Service
University Hospitals of Cleveland
Cleveland, Ohio

LARRY N. THIBOS, OD, PhD

Professor, School of Optometry
Indiana University
Bloomington, Indiana

BARRY A. WEISSMAN, OD, PhD, FAAO (Dip CL)

Professor, Chief Contact Lens Service
Department of Ophthalmology and Jules Stein Eye Institute
David Geffen School of Medicine
University of California, Los Angeles
Los Angeles, California

BRUCE WICK, OD, PhD

Professor Emeritus, College of Optometry
University of Houston
Houston, Texas

GEORGE C. WOO, OD, PhD

Former Chair, Professor of Optometry
School of Optometry
Hong Kong Polytechnic University
Hong Kong SAR, China
Professor Emeritus, School of Optometry
University of Waterloo
Waterloo, Canada

STANLEY WOO, OD, MS

Assistant Professor, College of Optometry
University of Houston
Houston, Texas

KARLA ZADNIK, OD, PhD

Glenn A. Fry Professor in Optometry and Physiological
Optics
College of Optometry
The Ohio State University
Columbus, Ohio

To

Patricia C. and Daniel J. Benjamin

for their support and endurance of the countless hours, irrecoverable, which were spent on a good cause.

Through their sacrifice of precious time, this work was finished and not abandoned.

Preface

to the Second Edition of Borish's Clinical Refraction



There has been a shift in overall tone regarding refractive eye care since the First Edition of *Borish's Clinical Refraction* was published. At that time the educational establishment and professional associations were becoming less astute with respect to clinical optics. The optical skills of eye care practitioners were receiving less attention than was necessary for provision of optimum vision in the population. It appeared as if the casual or approximate refraction and reliance on the automated refraction were to become the rule rather than the exception. The trend was promoted by corrections of presbyopia with soft contact lenses and of ametropia with refractive surgery that were not capable of exploiting an expert refraction, and by reimbursement systems favoring medical aspects of eye examinations. This situation conspired against the traditional role of practitioners to provide the best optical correction for the individual patient. However, most members of the eye care professions have by now realized the limitations of presbyopic correction with soft contact lenses and of ametropic correction with refractive surgery. The vast majority of those seeking eye care do so for refractive reasons and associated routine eye conditions. In most accounts the revenues generated by the clinical refraction and corrective devices in the general eye

practitioner's office overshadow those generated by the treatment of medical eye conditions. These factors and the expectations of wavefront refraction and wavefront correction have rekindled a general interest in optical performance. Thus, determination and correction of ocular optical deficiencies are being re-emphasized and refined. The First Edition of *Borish's Clinical Refraction* likely played a role in this resurgence. The Second Edition will help ensure that the eye care professions won't lose what they once valued so highly.

Hence, it is my pleasure to introduce the Second Edition of *Borish's Clinical Refraction*. Each chapter has been thoroughly updated from those published before. The chapters with the most alteration or addition are Chapter 14 (Posterior Segment Evaluation), Chapter 16 (Clinical Electrophysiology), Chapter 22 (Analysis, Interpretation, and Prescription for the Ametropias and Heterophorias), Chapter 25 (Prescription of Absorptive Lenses), Chapter 30 (Infants, Toddlers, and Children), Chapter 34 (Patients with Keratoconus and Irregular Astigmatism), and Chapter 36 (Patients with Low Vision). The previous chapter on the optics of contact lenses was expanded into two chapters, Chapter 26 (Applied Optics of Contact Lens Correction) and Chapter 27 (Clinical Optics of Contact Lens Prescription). Chapters entirely new to the Second Edition are Chapter 19 (Wavefront Refraction), Chapter 29 (Optical Correction with Refractive Surgery and Prosthetic Devices), and Chapter 37 (Refractive Effects of Ocular Disease).

I'm told that the second edition of a book is always better than the first, and I sincerely believe this to be the case with *Borish's Clinical Refraction*. The relationship between visual acuity and refractive error and the physiology behind the development of myopia are more extensively reviewed. Recent information on the effects of pupillary dilation and the fundus appearance in eyes of different axial lengths were added. Electrophysiology has been covered in a more clinical manner. The potential for monitoring of the cortical response to visual stimuli during the refraction, and the possibility for future reduction of the subjectivity of the subjective

refraction, is better demonstrated. The analysis of optometric data in the examination is more comprehensive in terms of the zone of single clear binocular vision. Short-corridor and free-form progressive-addition multifocal spectacle lenses are covered, as are the increased variety of occupational progressive lenses. The absorption characteristics of new spectacle and contact lens materials have been displayed. A special section on solar viewing and solar eclipses has been added. Important new information on the optical correction of our youthful patients and its effect on development of ametropia have been incorporated. In terms of contact lenses, some of the added features are the Coroneo Effect, Rizzuti Phenomenon, the optics underlying corneal refractive therapy (orthokeratology) and piggyback contact lenses. The busy practitioner will appreciate how the optical quirk known as the lacrimal lens allows the ready application of these latter two modalities in practice, and how the former two optical entities are correlated with ocular surface abnormalities. New information on keratoconus and the use of rigid optic zones, the centration of which is key to a successful result, are more fully explained given the proliferation of contact lens designs resulting from the quest for hyper-oxygen transmissibility. Moreover, the advent of wavefront refraction and wavefront correction is anticipated with the new Chapter 19. The clinician will understand the major current limitation with wavefront analysis, as it is only just being applied to the visible spectrum in comparison to the monochromatic assessments that are now the norm. Those interested in the refractive outcomes and consequences of refractive surgery or prosthetic optical devices other than contact lenses, can now turn to the new Chapter 29 for a frank discussion of these modes of correction. And, the Second Edition of *Borish's Clinical Refraction* concludes with Chapter 37 on the refractive effects of ocular disease, such that the primary eye care practitioner may better diagnose and manage these eye conditions from a refractive standpoint.

The First and Second Editions were both recipients of knowledge acquired over the careers of the many chapter authors. Several of the authors have retired over the last 8 years: Charles Campbell and Drs. Paul Pease, Donald Pitts, Michael Polasky, James Saladin, and

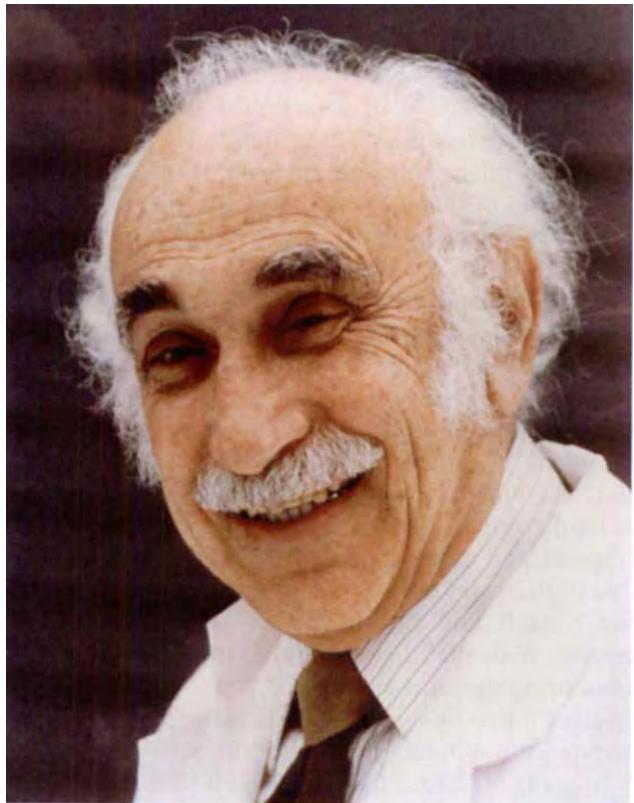
George Woo. Some others have intimated that they may also do so before any third edition of this book is contemplated. Two have become deans of schools of optometry: Drs. John Amos and Gerald Lowther. Yet, they all still worked diligently to place what could be their final and most comprehensive distillations into the Second Edition of *Borish's Clinical Refraction*. It was certainly an honor and privilege for me to have worked with each one of them. Their material is, I believe, an immense educational value for readers of the Second Edition.

The Second Edition of *Borish's Clinical Refraction* is now the Seventh Iteration of his venerable book, as the initial *Outline of Optometry* by Irvin M. Borish was printed over 68 years ago. Irv Borish is nearly as active as he ever was, and is now past the 93-year mark. Among several awards received after publication of the First Edition, he was recognized in 2002 for his creativity, perseverance, and intrepid spirit with the Herman B. Wells Visionary Award from the Indiana University Foundation and Board of Trustees. However, his lifelong companion, Bea Borish, passed away in April of 2001. The 2005 meeting of the American Academy of Optometry was the first in over 70 years not to feature the presence of at least one of the (now) great grandparents of optometry, whose name became synonymous with that of the clinical refraction in the last half of the 20th century. Irv has developed quite a reputation within the American Academy of Optometry as an amateur painter—an image of one of his paintings, a gift received years ago by the editor, adorns this Preface to the Second Edition. Dr. Borish continues to lead by example and remains an inspiration to us all. He changed the eye care professions and the world.

The reader is encouraged to review the Preface to the First Edition, on the following pages, which concluded with the naming of the book for the purpose of keeping Irv Borish associated with clinical refraction well into the 21st century. With the printing of this Second Edition, the Seventh Iteration, that aforementioned goal has been achieved.

William J. Benjamin
Editor

Preface to the First Edition



DR. IRVIN M. BORISH

In a photo reprinted with permission of the *Review of Optometry*, March 15, 1982.

The procedures comprising the clinical refraction and much of the modern eye examination were introduced over the last 150 years or so, during the formative period of the eye care professions. After substantial modification, revision, and collection into the examination routine, incremental refinements of these techniques and their interpretations built up periodically to the point that, occasionally, they required updating and restatement taking into account the new findings over time. So it was that the *Outline of Optometry* by Dr. Irvin M. Borish was published in 1938 by V. J. Le Gros & Co.,

Chicago, under a copyright by the Northern Illinois College of Optometry. This book was to become the ancestor of a series of three subsequent volumes that oriented the basic eye examination around the clinical refraction. Essentially an elaboration of Irv Borish's teaching notes into chapters on different topics, this original 266-page book was expanded to 431 pages when published in 1949 as the first edition of *Clinical Refraction* by Professional Press, Inc., Chicago. Being virtually the only optometric text that was fully annotated and grounded in clinical science, this book became the standard clinical text in the field. The first edition was introduced at a time when literally thousands of World War II soldiers were being retrained in optometry programs under the "G.I. Bill," and so became immediately a huge success. The second edition appeared in 1954. It was, however, the third edition published as a single 1381-page volume in 1970 that became the standard reference text in the field. Its hallmark was the thoroughness with which it chronicled each routine examination procedure and the underlying physiological principles. The text helped substantially to educate, as well as to train, a generation of eye care clinicians and clinical scholars, and was probably the first optometric book to transcend the professional boundary between ophthalmology and optometry. Written in outline form over a span of 7 years by Dr. Borish, 6 chapter coauthors, and 14 collaborating editors, the very successful third edition underwent five printings and a redacted two-volume version was released in 1975. Even today, the third edition serves as the resource or reference book when one needs to look up the derivation of a particular fact or article about the eye examination from the era prior to Index Medicus and computerized library search methodologies.

However, time has now overtaken even the third edition of *Clinical Refraction*. New understandings of many of the examination procedures have been accompanied by revolutions in the electronic, computer, and optics industries. Eye care now includes many new pharmacological and surgical interventions, and increasing numbers of automated or semiautomated

technical procedures are often used in clinical practice. Therefore, it became time to refresh the knowledge base and procedures of the routine eye examination and, in particular, the clinical refraction. The new *Borish's Clinical Refraction* is a completely new volume, although the goals of this work are similar to those of *Clinical Refraction* (1970). The new book is written in text, not in outline form as was the 1970 edition, by respected experts in the individual topics represented by the chapter titles. It is extensively illustrated and has much color reproduction, both features unlike the old book. There were many changes that have occurred over the intervening 28 years, particularly with respect to objective, electronic, and computerized testing. The work is highly practical in nature, though it also takes the time to educate the reader as he or she consumes each chapter. With an "eye out" for the future, it explains the direction and potential eventual outcome of future developments in the clinical refraction.

Borish's Clinical Refraction provides an encyclopedic coverage of objective and subjective refractive techniques at far and near, and the associated elements of the eye examination that impact upon them. The volume includes the complete clinical application of modern methods of examination, with descriptions and estimates of recently introduced potential additions and alterations to the clinical refraction. The reader is instructed in the precise techniques, supplied with premises and principles underlying the techniques, informed of incidents common to ophthalmic practice, and shown anticipated patient responses. This is the first volume to provide comprehensive coverage of refractive techniques in a variety of special patient populations, that is, the elderly, presbyopes, children, amblyopes and strabismics, anisometropes and aniseikonics, and those patients with irregular astigmatism, low vision, high ametropia, or contact lenses. The book is particularly well illustrated and color figures are presented when necessary, especially for color vision testing and corneal topography. Using a great breadth of authority and knowledge, the text reflects many years of highly successful practical experience, the academic bases for the various examination techniques, and coordinates the various elements, objective and subjective, which compose the basic eye examination.

Borish's Clinical Refraction was specifically written for the ophthalmic practitioner and the advanced student of refractive and eye examination procedures. Hence, it has a clinical orientation and practical hands-on approach to match the clinical situation. The number and quality of the illustrations and photos make the written text easier to understand. It is hoped that these features will make it possible to link principles with clinical practice and help the practitioner to faster assimilate the material into his or her clinical routine. The text is also meant to help bridge the gap between

practitioners and educators. Few stones were left unturned in providing the clinician with the background knowledge and education necessary to learn or update his or her techniques and patient management as future advances become clinically relevant.

Borish's Clinical Refraction conveys an optimum capacity per page by slight minification of the figures so as to leave room for more print, slight reduction of the font size, and slimming of the page margins to 0.5". This dense informational content allows the text to encompass less than 1250 pages for a comfortable binding into a single volume. It is common in a multi-authored book to have significant redundancy, but I have tried to minimize the overlaps by editing out obvious or substantial redundancy and by personal authorship of several of the potentially overlapping chapters. In some cases I felt that some overlap was of benefit in the transfer of complicated basic knowledge to clinical application and I allowed these redundancies to stand.

In this endeavor I must sincerely thank the chapter authors (45 of them, including Dr. Borish) for submitting such comprehensive and thorough initial draft manuscripts, for allowing me to heavily critique their submissions, and for working their initial documents into final manuscripts while enduring pesty phone calls, newsletters, and reminders. I also thank Mr. Kenneth Norris, Ms. Debra Brewer, and Mr. Timothy Hays for their expertise in producing many of the diagrams and photographs in those chapters that I authored or coauthored. I am indebted to Ms. Hazel Hacker and Mr. Richard Lampert of W.B. Saunders Company for their diligence and understanding in pursuing this considerable project, and to Dr. Irvin M. Borish for his guidance and consultation throughout the long and arduous process. With their help, the time from author recruitment to publication was halved to 3½ years—from the 7 years for the 1970 classic—achievable only with more than twice the number of contributors!

In using his name, the new *Borish's Clinical Refraction* also serves to celebrate the contributions of Dr. Irvin M. Borish to the field of eye care, and to commemorate the legacy he has left to the eye care professions. Born in Philadelphia on January 21, 1913, the son of a Russian immigrant worker who passed away just after his high school years, Irvin Max Borish went to Temple University. He moved to Chicago and lived with an uncle in order to attend the Northern Illinois College of Optometry (NICO), from which he graduated as its first "straight A" student. He was Chief of the Eye Clinic at the NICO and became acquainted with biophysicist Dr. Charles Sheard, who had founded the original full-scale university program in optometry at the Ohio State University, and who was then a Distinguished Professor at the University of Minnesota working at the Mayo Foundation in Rochester. Always extremely energetic and a good strategist, Dr. Borish became convinced that the

future of an eye care profession was with an enhanced university education, and that the quality of eye care could only be upgraded in concert with excellent clinical research. He constantly sought to further the schools and colleges of optometry in terms of the levels of education and clinical research. Indeed, this issue resulted in the termination of his career at the private Northern Illinois College of Optometry, when he took the position that the NICO should upgrade its level of education by affiliation with the University of Illinois. Then, during the slow building phase of his 30-year practice in Kokomo, Indiana, Dr. Borish used the slack time to write the first edition of his book and began a crusade with two others to institute a university-level optometry school in Indiana. The results were, of course, *Clinical Refraction* (1949) and, eventually, the founding of the School of Optometry at the Indiana University in Bloomington. He was a founding member of the Association of Schools and Colleges of Optometry and coauthored the first *Manual of Accreditation for the Council on Accreditation of the American Optometric Association*. The "Irvin M. Borish Chair in Optometric Practice" was established at the University of Houston and, recently, the "Borish Center for Ophthalmic Research" was dedicated in his honor at the Indiana University.

An overview of the awards that he has received, his many important career activities, and his publications would be too long to be recounted in this Preface. These are, however, contained within the "Irvin M. Borish Reading Room" in the library of the University of Houston, College of Optometry. Dr. Borish is now a Professor emeritus of the Indiana University and a past Benedict Professor of the University of Houston, where it was my good fortune to have been assigned an office across the hall from his. My wife, Pat, and I were given considerable doses of the Borish philosophy on numerous occasions spent with Irv and his wife, Bea, who now reside in Boca Raton, Florida. As one would expect knowing the two of them, theirs is an active retirement spent pushing those premises and goals honed over a lifetime. Truly, he is the primary architect of ophthalmic education and practice, and his name was synonymous with clinical refraction for the post-WW II generation of ophthalmic clinicians. On the sixtieth anniversary of the publication of his original *Outline of Optometry*, it is my hope and, I believe, the hope of the other 44 authors of Borish's *Clinical Refraction*, that his name will now remain associated with clinical refraction well into the twenty-first century.

William J. Benjamin

Editor

1

Refractive Status of the Eye

Mark Rosenfield

The ocular refractive status refers to the locus within the eye conjugate with optical infinity during minimal accommodation.* Under these conditions:

- In an *emmetropic* eye, incident parallel rays of light are brought to a focus upon the retina.
- In a *hyperopic* (or *hypermetropic*) eye, incident parallel rays of light are brought to a focus behind the retina.
- In a *myopic* eye, incident parallel rays of light are brought to a focus in front of the retina.

These differences are illustrated in Figure 1-1.

Ametropia, or the absence of emmetropia, may be produced by variations in:

- The relative location of the optical elements of the eye with respect to the retina.
- The relative refractive power of the optical elements with respect to the location of the retina.

For example, if the total refractive power of an eye remains constant but the axial length (i.e., the distance from the anterior corneal surface to the retina measured along the visual axis) increases, a myopic shift in refractive error will result. Similarly, if the axial length of an eye remains constant but the refractive power of one or more of its optical elements increases, a myopic shift in refractive status will occur. These two forms of myopia may be referred to as *axial myopia* and *refractive myopia*, respectively.

In a myopic eye, the *punctum remotum (PR)*, or *far point*, that is, the point conjugate with the retina during

minimal accommodation, lies in front of the principal point of the eye. The distance in meters from the far point to the principal point (denoted k) is the reciprocal of the refractive error in diopters (denoted K). If the depth of focus of the eye is discounted, the far point of a myopic eye represents the farthest distance that can be seen clearly. In a hyperopic eye, the far point lies behind the principal point. These far-point locations are illustrated in Figure 1-2. A refractive error may be corrected by introducing a supplementary or correcting lens whose second principal focus coincides with the far point of the eye.

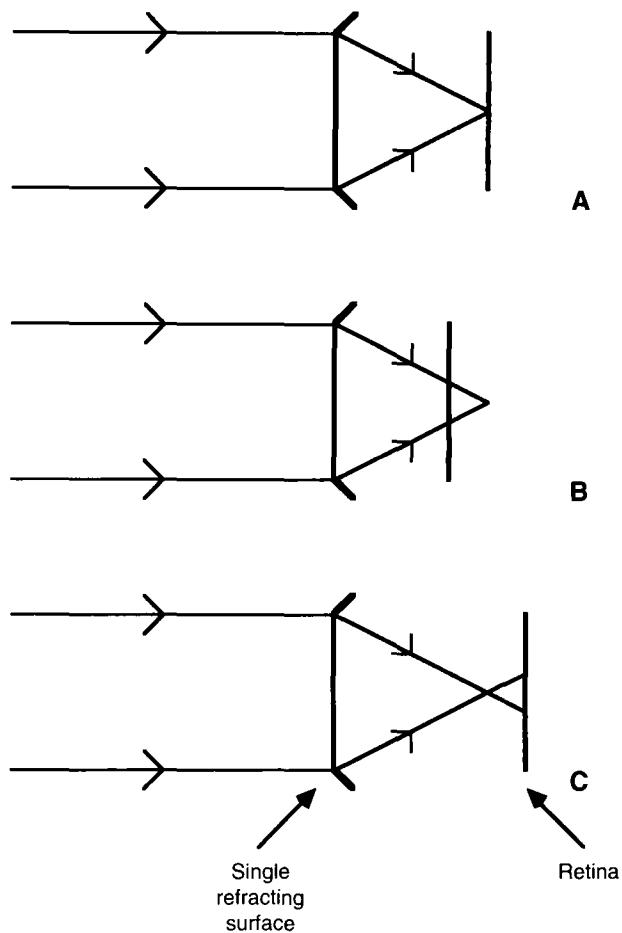
MYOPIA

Myopia results from an eye having excessive refractive power for its axial length. This may be due either to the eye having a relatively long axial length or to increased dioptric power of one or more of the refractive elements. Aristotle (384–322 BC) is credited with first distinguishing nearsightedness.⁷ However, the term *myopia* was derived by Galen (131–201 AD) from the words *myein* ("to close") and *ops* ("eye").⁸ Galen observed that nearsighted people partially closed their eyes to see better. Galen's theory of vision involved visual spirits called *pneuma*, which originated in the brain and filled the anterior chamber. In normal vision, the pneuma passed from the eye to distant objects so that they might be observed. A deficiency of pneuma reduced the ability to perceive distant objects, resulting in myopia. The Romans considered myopia to be a permanent visual handicap called *vicium perpetuum*, which if found in a servant considerably lessened his value.⁹

In addition to dividing myopia into axial and refractive types according to etiology, several other classifications have been suggested. In a review of methods for the classification of myopia, Grosvenor¹⁰ observed that a wide range of systems have been devised in the last 150 years. The proposed classifications may be grouped under the following broad headings:

- Rate of myopic progression
- Anatomical features of myopia

*Most definitions of refractive error refer to the location of the point conjugate with the retina when the eye is either at rest or not accommodating. It is now established that when the eye is viewing a distant object of regard, that is, a 0 D accommodative stimulus, there is typically an accommodative response of approximately 0.40 D^{1–4} that is also referred to as the "lead of accommodation." Even in the absence of an optical stimulus to accommodation, for example, in total darkness, an accommodative response of 0.50 to 1.00 D is usually observed.^{5,6} Thus, the situation of zero accommodation that is theoretically required for the assessment of refractive error is almost never obtained. In practice, the refractive state of the eye is typically assessed under conditions designed to minimize the accommodative response. See Chapter 4 for a full treatment of the typical relationship between the accommodative stimulus and response.

**Figure 1-1**

When parallel rays of light are incident upon emmetropic (A), hyperopic (B), and myopic (C) eyes, they are focused on, behind, and in front of the retina, respectively. It should be noted that for ease of illustration, in this and subsequent figures a simplified eye is adopted that comprises a single refracting surface converging the incident rays toward the retina.

- Degree of myopia
- Physiological and pathological myopia
- Hereditary and environmentally induced myopia
- Theory of myopic development
- Age of myopia onset

Classification by Rate of Myopic Progression

Donders¹¹ classified myopia on the basis of its rate of progression, describing three categories of myopia: stationary, temporarily progressive, and permanently progressive. *Stationary myopia* is generally of low degree (-1.50 to -2.00 D) and arises "in the years of development." The degree of myopia remains stationary during

adulthood and may occasionally diminish with the approach of old age. However, Donders incorrectly suggested that the apparent reduction in myopia with increasing age was probably due to age-related pupillary miosis with an associated increase in the depth of focus of the eye.

Temporarily progressive myopia generally arises in the early teens and progresses until the late 20s. After this age, the rate of myopia progression approaches zero. Interestingly, Donders reported that it was rare for myopia to develop after 15 years of age in previously normal eyes and, falsely, that it never developed after the 20th year of life (see Late-Onset Myopia).

Permanently progressive myopia ascends rapidly until around 25 to 35 years of age, and thereafter advances more slowly. Subsequent increases in myopia are said to occur in jumps, rather than in a smooth progression. Donders observed that because of pathological conditions such as retinal detachment and macular degeneration, in these cases it was rare at 60 years of age "to find a tolerably useful eye."

Classification by the Anatomical Features of Myopia

Borish⁸ stated that myopia could be:

- *Axial*, whereby the eye is too long for its refractive power.
- *Refractive*, whereby the refractive system is too powerful for the axial length of the eye.

An increase in axial length may occur in the anterior or posterior portions of the globe individually, or may occur throughout the eye. The site of elongation may have implications for determining the etiology. For example, it has been suggested that expansion of the posterior portion of the globe may be related to the actions of the superior and inferior oblique muscles during vergence.¹²

Borish⁸ further divided refractive myopia into:

- *Index myopia*, in which one or more of the refractive indices of the media are anomalous.
- *Curvature myopia*, in which the reduced radius of curvature of one or more refractive surfaces produces increased dioptric power.
- *Anterior chamber myopia*, in which a decrease in anterior chamber depth increases the refractive power of the eye.

Classification by Degree of Myopia

Classification of myopia on the basis of degree is frequently associated with other factors, such as the age of myopia onset.⁷ Hirsch¹³ examined the refractive error of 562 eyes having at least -1.00 D of myopia in patients between 18 and 60 years of age. He divided the population into three groups on the basis of the degree of

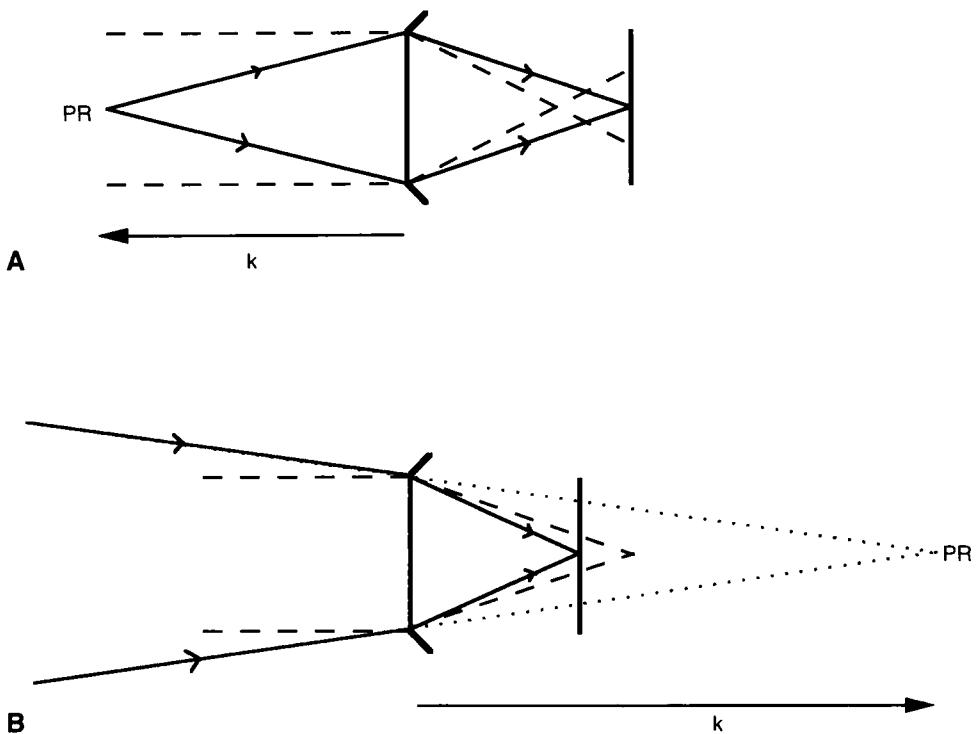


Figure 1-2

Location of the far point, or punctum remotum (*PR*), in a myopic (A) and hyperopic (B) eye, respectively. In a myopic eye, rays diverging from the far point are focused on the retina, whereas in a hyperopic eye, rays converging toward the far point are imaged on the retina. The dashed lines represent the path taken by parallel incident rays from a distant object of regard. Under the standard sign convention, the distance *k* is negative for the myopic eye and positive for the hyperopic eye.

myopia, which he designated the *alpha*, *beta*, and *gamma* groups, respectively. Using inferential statistics, he determined that the alpha group followed a normal distribution curve, with a theoretically assumed peak of +0.50 D. The beta group was represented by a second normal distribution curve, with its peak around -4 D. Hirsch suggested that the myopia in this group may be hereditary in origin. The gamma group ranged from -9 to -15 D, and this degree was described by Hirsch as malignant, pathological, degenerative, or congenital. Sorsby et al.,¹⁴ in an investigation of 341 eyes between 20 and 60 years of age, concluded that 95% of refractive errors fell within ± 4 D. They also suggested that the etiology of myopia of less than 4 D differed from that myopia exceeding 4 D, noting that the range of biometric component values for refractive errors up to ± 4 D was essentially the same as that found in an emmetropic eye. Sorsby et al.¹⁴ suggested that these relatively low refractive errors resulted from a breakdown of correlated growth of the ocular components, rather than the dimensions of any individual refractive component lying outside the normal range.

Classification into Physiological and Pathological Myopias

Physiological myopia was defined by Curtin¹⁵ as myopia in which each component of refraction lies within the normal distribution for that population. Thus, the myopia arises from a failure of correlation between the refractive components. However, physiological myopia may be defined as normal as opposed to pathologic myopia.¹⁶ Therefore, physiological myopia might simply and more accurately be defined as nonpathological myopia.

Duke-Elder and Abrams¹⁷ defined pathological refractive errors as "those refractive anomalies determined by the presence in the optical system of the eye of an element which lies outside the limits of the normal biological variations." *Pathological myopia* may also be described as malignant or degenerative myopia.¹⁷ These authors adopted the term *degenerative myopia* to describe myopia that is accompanied by degenerative changes, particularly in the posterior segment of the globe. This is most frequently found in high (>6 D) degrees of myopia, but Duke-Elder and

Abrams suggested that a classification merely by degree of ametropia is inappropriate because degenerative changes may also occur in cases of low myopia. Moreover, in 1913 Harman¹⁷ described a case of more than 17 D of myopia without any pathological changes.

Classification into Hereditary and Environmentally Induced Myopia

The debate of hereditary versus environmental influences on the development of myopia has persisted for more than 400 years and is still unresolved.¹⁸ Kepler, writing in 1604, was the first to have suggested an association between the development of myopia and the performance of sustained near-vision tasks.⁷ However, Rosenfield¹⁹ noted that the case for such an association remains unproven. It is frequently impossible to distinguish between environmental and hereditary influences, and hence other means of classification have been adopted (e.g., age of onset or degree of myopia) in an attempt to provide additional information regarding the etiology of refractive error development.

Classification According to Theory of Myopic Development

In a review of the etiology of refractive error, McBrien and Barnes²⁰ described three major theories of myopic development:

- The biological-statistical theory
- The use-abuse theory
- The theory of emmetropization

The biological-statistical theory²¹ considered variations in refractive error as forming a biological continuum ranging from high myopia to high hyperopia. Thus, ametropia simply represented the normal biological variation of a physiological component. However, data by both Stenstrom²² and Sorsby et al.¹⁴ clearly demonstrated that the distribution of refractive error was not normal (see Components of Refraction and Their Correlation).

The so-called use-abuse theory proposed by Cohn²³ suggested that myopia onset was an adaptation to use or abuse of the eyes during sustained near vision. Cohn examined the prevalence of myopia in more than 10,000 German schoolchildren. He observed that in the youngest children there was little myopia, but the prevalence increased with age. Cohn concluded that because the increased prevalence of myopia occurred during the educational process, a substantial portion of which entailed reading and other close work, the onset of myopia was related to increased near-vision activities.

Numerous investigators have reported a higher prevalence of myopia among people whose occupations involve substantial amounts of close work.^{7,24-26} In addition, Young²⁷ demonstrated that when adolescent monkeys are restricted to a near-vision environment,

they exhibit significant increases in myopia. Other studies indicated an increased prevalence of myopia in an Eskimo population after the introduction of formal education, with its increased near-vision requirement.²⁸⁻³¹ However, other factors, such as intelligence and changes toward a Western diet, may also have been at least partly responsible for the change in refractive error distribution in this population.^{7,32}

In view of the higher prevalence of emmetropia than might be predicted on purely statistical grounds (see Figure 1-10), it would appear that the components of the eye do not grow independently, but rather undergo a process of coordinated growth. This proposed correlated growth of the ocular biometric components has been referred to as *emmetropization*.^{33,34} Van Alphen³⁵ suggested that emmetropization was achieved by a negative-feedback, self-focusing control system. Variations in ciliary muscle tone could produce changes in refractive error by interfering with this self-focusing mechanism. This process is discussed further in Correlation of the Ocular Components and Emmetropization and in Chapters 2 and 3.

Classification Based on Age of Onset

Several studies have classified myopia on the basis of the subjects' age at the time of reported myopia onset. In a review of this topic, Grosvenor¹⁰ classified myopia into the following categories:

- *Congenital myopia*—Myopia is present at birth and persists through infancy.
- *Youth-onset myopia*—The onset of myopia occurs between 6 years of age and the early teens.
- *Early adult-onset myopia*—The onset of myopia occurs between 20 and 40 years of age.
- *Late adult-onset myopia*—Myopia onset occurs after 40 years of age.

The prevalence of each of these categories of myopia is illustrated in Figure 1-3.

Clearly, a major difficulty in any attempt to classify myopia in terms of age of onset is that recall by people of their myopic development will probably relate to their first refractive correction, whereas symptoms of reduced distance vision may have occurred at a previously undetermined time. Rosenberg and Goldschmidt³⁶ investigated the development of myopia in 280 Danish schoolchildren and found that the premyopic period and initial myopia development were so variable in terms of duration, symptomatology, and progression that it was not possible to determine the exact onset of myopia. They observed that marked differences existed in adaptation to reduced distance visual acuity, noting that some children had symptoms when only 0.50 D of myopia or less was present, whereas others had between 1 and 2 D of myopia and yet failed to report any visual symptoms.

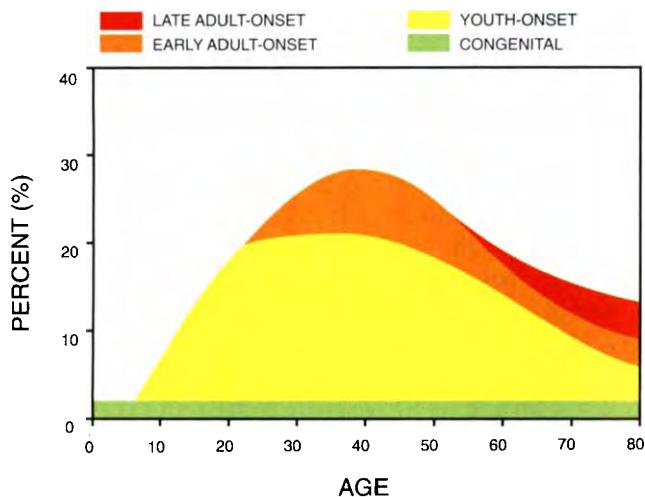


Figure 1-3

Prevalence of myopia with age, classified as congenital, youth-onset, early adult-onset, and late adult-onset myopia. (From Grosvenor T. 1987. A review and a suggested classification system for myopia on the basis of age-related prevalence and age of onset. Am J Optom Physiol Opt 64:550.)

Several researchers have used the age of myopia onset in an attempt to differentiate between environmentally induced myopia and myopia that relates to inherited factors.³⁷⁻⁴⁰ The rationale behind this differentiation is that studies on the growth of the ocular components have indicated that the eye reaches its adult axial length by 13 years of age.⁴¹⁻⁴³ Furthermore, it has been demonstrated that the other refractive components of the eye have attained their adult values by 13 to 15 years of age.^{44-45a} Investigations of the development of refractive error in children have indicated that stabilization of the refractive error normally occurs around 15 years of age.⁴⁶⁻⁴⁹ Morgan observed that, by the age of 16 years, most children have attained their adult refraction, which will remain nearly constant for the next three decades.⁵⁰

However, Hirsch⁵¹ noted that a small percentage of people exhibit changes in refractive error after 16 years of age, and investigations of military cadets support this proposal, indicating that either the first development or the progression of myopia is often observed in these older individuals (see Late-Onset Myopia).

Late-Onset Myopia

Goldschmidt⁷ described a type of myopia that develops after the cessation of bodily growth, adopting the term *Spätmyopie* (literally, "late myopia" in German) to describe this form of ametropia. Goldschmidt stated that this myopia may be environmentally determined and that its onset was likely to be related to high levels of near vision. He also observed that subjects who become myopic during the period of bodily growth may

develop late-onset myopia in addition to their initial myopia, should they choose an occupation that requires high levels of close work. Goldschmidt stated that whereas "common low myopia," which developed during periods of bodily growth, had its etiology "in the genetic substance," another type of myopia may exist that develops after the cessation of bodily growth and is principally found in individuals undertaking fatiguing close work. Accordingly, Goldschmidt concluded that this type of myopia was environmental in origin. However, Rosenfield¹⁸ noted that it may not be appropriate to assign etiology on the basis of age of onset. He observed a number of systemic conditions that first manifest themselves in mid- or late-adulthood, such as Huntington's disease and Parkinson's disease, have a clear hereditary basis.^{52-53a}

Goss and Winkler⁴⁸ examined the refractive records of 299 patients, all of whom had at least four examinations between 6 and 24 years of age and developed at least 0.50 D of myopia during this period. They observed that although there was a great deal of individual variability, the mean age of myopia cessation was 15.53 years. They also noted that myopia cessation occurred earlier in female subjects. Later, Goss et al.⁵⁴ examined longitudinal data on 559 myopic patients, 108 of whom had been examined on three or more occasions. They categorized the change in myopia during adulthood (i.e., beyond 18 years of age) into the following three groups:

- **Adult stabilization**—Rapid increases in myopia during early adolescence were followed by stabilization during early adulthood. Minor adjustment of the refractive error sometimes occurred after stabilization, but this change was generally small, on the order of ± 0.25 D. Sixty-eight percent of male subjects and 87% of female subjects fell into this category.
- **Adult continuation**—The rapid myopic progression seen during adolescence continued through adulthood. This pattern represented 25% of male subjects and 13% of female subjects.
- **Adult acceleration**—Myopic progression increased after adolescence. This was the least common pattern, representing 6.3% of male subjects and no female subjects.

Goss et al.⁵⁴ reviewed a series of reports of cases of myopia onset during young adulthood. Several investigators observed an increase in myopia among students of military academies that varied with the amount of time spent undertaking near-vision tasks.⁵⁵⁻⁵⁹ Riffenburgh⁶⁰ presented nine cases of myopia onset after 20 years of age. He suggested that the myopia that manifests itself in adulthood has a form different from that which appears in childhood, and concluded that young adult-onset myopia was associated with near work. Young⁶¹ stated that approximately 8% of the myopic

subjects in graduate and professional schools became myopic in their 20s. Furthermore, Zadnik and Mutti,⁶² in an investigation of refractive error changes in law students, reported that in a 6-month period, 37.5% ($n = 12$) of the eyes examined became at least 0.50 D more myopic. Stevenson⁶³ described two types of myopia—developmental myopia, in which the age of onset is between 6 and 9 years, and environmental myopia, which develops between 15 and 17 years of age.

Goss et al.⁵⁴ and Goss and Erickson⁶⁴ described cases of late-onset myopia that were induced by a decrease in the radius of corneal curvature. Goss et al. observed increases in both myopia and corneal steepening in 32 subjects, with an overall correlation coefficient of +0.58. Goss and Erickson also reported a significant correlation between changes in refractive error and corneal steepening in 37 patients who had three or more refractions at age 18 or older. However, it should be noted that in Goss and Erickson's study, the mean changes in refractive error were small (for male subjects, -0.06 D; for female subjects, -0.02 D), and some subjects actually showed increased hyperopia during the test period.

Adams⁶⁵ presented his own case of late-onset myopia in which his refractive correction changed from -0.25 D at 19 years of age to -4.75 D at 42 years of age. Although there was no significant change in corneal curvature, his axial length at 42 years of age was 25.8 mm, 1.8 mm longer than the mean given by Stenstrom²² for an adult population. Taking 0.234 D of myopia for each additional 0.10 mm of axial length, this equates to 4.20 D of myopia accounted for by the increased length of the globe.⁶⁶

McBrien and Millodot⁴⁵ compared the ocular biometric components of 30 late-onset (after 15 years of age) myopes and 30 emmetropic subjects who were age and sex matched. Using A-scan ultrasonography and keratometry, they observed that late-onset myopes had a significantly increased axial length. Both anterior and vitreous chamber depths were significantly longer in the late-onset myopes, although there was no significant difference in corneal curvature. These findings indicate that late-onset myopia results from axial elongation, rather than corneal or lenticular changes. This conclusion was supported by Bullimore et al.⁶⁷ and Grosvenor and Scott,⁶⁸ whereas Rosenfield and Gilmartin⁶⁹ also observed no significant differences in corneal curvature between early-onset myopes, late-onset myopes, and emmetropes.

A series of studies by Grosvenor⁷⁰ and Grosvenor and Scott^{44,71,72} have suggested that the onset of young adult-onset myopia may be predicted by examination of the ratio between the axial length and the corneal radius of curvature (AL/CR ratio). They noted that using the Gullstrand schematic eye, one would predict an AL/CR ratio in an emmetropic eye of 24.0/8.0, or 3.0. However, in

a comparison of the magnitude of this ratio between British and Melanesian emmetropic schoolchildren, Grosvenor reported that the AL/CR ratio was significantly greater in the British subjects. Any increase in this ratio, regardless of whether it is due to an elongation in axial length or to a steepening corneal radius, would tend to lead to myopia. Accordingly, Grosvenor suggested that in the British children, the increased ratio was accompanied by a reduction in crystalline lens power in order to maintain emmetropia. In a cross-sectional study, Grosvenor and Scott^{44,45a} reported an extremely high correlation ($r = -0.92$) between the AL/CR ratio and refractive error. Indeed, this correlation was higher than that observed between axial length alone and refractive error ($r = -0.76$). Furthermore, in a longitudinal investigation, Goss and Jackson⁷³ found that eyes that became myopic over a 3-year period had higher AL/CR ratios than did those that remained emmetropic. This difference in ratio was due to a decreased corneal radius of curvature at the initial examination while the axial lengths were equivalent. This latter finding appears to conflict with the observations of Zadnik et al.,⁷⁴ who reported that children who had two myopic parents (and were therefore at greater risk of developing myopia) had longer eyes than did children with one or no myopic parents, even before any myopia had become manifest. Indeed, Zadnik et al.⁷⁵ reported that the best single predictor of future myopia onset for a group of third-grade children (mean age at baseline was 8.6 years) was a cycloplegic spherical equivalent refractive error of less than +0.75 D.

Other Myopias

Night Myopia

The phenomenon of increased myopia under low-luminance conditions was first reported in 1789 by the Reverend Nevil Maskelyne, the Astronomer Royal. He found that his astronomical observations at night were facilitated by the use of concave spectacle lenses. Maskelyne reported, "To see day objects with most distinctness, I require a less concave lens by 'one degree' (between 0.37 and 1 D) than for seeing the stars best by night."⁷⁶ Almost a century later, in 1883, the same effect was observed by Lord Rayleigh. For a review of other historical papers relating to this condition, see Levene.⁷⁶

More recent evidence has demonstrated that night myopia is produced by an increased accommodative response (typically on the order of 0.50 to 1.00 D) under degraded stimulus conditions.^{5,6,80} However, there is also some suggestion that changes in chromatic aberration may also be involved in this myopic shift. The chromatic aberration of the eye results in blue light being refracted more than red light.⁷⁷ Furthermore, as the eye transfers from photopic to scotopic luminance

levels, its peak sensitivity shifts from approximately 555 nm to around 510 nm. This change in sensitivity is termed the *Purkinje shift*.⁷⁸ Thus, at extremely low luminance levels, the eye becomes most sensitive to those wavelengths undergoing a greater degree of refraction, and therefore appears to be more myopic than it is under photopic viewing conditions. To determine the extent to which chromatic aberration could account for the myopic shift under reduced illumination, Wald and Griffin⁷⁹ used a spectral stigmatoscope to measure the refractive state of the eye under monochromatic light. They assessed axial chromatic aberration by measuring the eye's refractive state under nine narrow monochromatic conditions over a range of 365 to 750 nm. They concluded that the Purkinje shift in spectral sensitivity would produce a myopic shift in refractive power of approximately 0.35 to 0.40 D. However, the mean magnitude of the refractive error shift observed under reduced illumination in their study was -0.59 D (range = -1.40 to +3.40 D). Thus, chromatic aberration may account for a significant proportion of the increased myopia observed under degraded stimulus conditions.

Direct evidence that night myopia is primarily produced by ocular accommodation comes from researchers who examined variations in the form of the third Purkinje image (reflected from the anterior surface of the crystalline lens) to assess the accommodative response under very low illumination levels.⁸¹⁻⁸⁴ These investigators all reported mean changes in accommodation of approximately 0.75 D. This confirmed that the change in the dioptric power of the eye resulted directly from a shift in accommodation, that is, a change in the refractive power of the crystalline lens. Indirect evidence that changes in accommodation must be the primary source comes from the observation of equivalent levels of tonic accommodation under a number of widely varying test conditions. For example, in the measurement of tonic accommodation, the accommodative loop may be opened by having the subject view a Ganzfeld field, a low spatial frequency difference of Gaussian (DOG) grating, a distant target through a 0.5 mm pinhole, or by placing the subject in total darkness.⁸⁵⁻⁸⁹ Clearly, the magnitude of spherical and/or chromatic aberration will exhibit wide variations under these different conditions, and yet equivalent values of tonic accommodation have been recorded.⁹⁰ Therefore, both chromatic aberration and tonic accommodation appear to be the main determinants of the relative myopic shift observed under degraded stimulus conditions.⁵

Pseudomyopia

Pseudomyopia has been defined as a reversible form of myopia that results from a spasm of the ciliary muscle.⁹¹

It is apparent that this does not meet the standard definition of a refractive error, that is, one that occurs under conditions of minimal accommodation. The excessive accommodative response produces an apparent myopic shift that will disappear when a cycloplegic agent is administered to produce relaxation of accommodation. These patients are frequently detected by the presence of a significantly greater (more than 1 D) amount of relative plus power (i.e., more hyperopia or less myopia) on retinoscopy compared with the subjective refractive findings, or by the observation of either an eso shift in oculomotor balance or a reduction in distance visual acuity, particularly toward the end of a working day.

HYPEROPIA (OR HYPERMETROPIA)

Hyperopia results when the eye has insufficient refractive power for its axial length. The term *hypermetropia* comes from *hyper*, meaning "in excess"; *met*, meaning "measure"; and *opia*, meaning "of the eye." This refractive error may be the result of an eye having a relatively short axial length, or reduced dioptric power of one or more of the refractive elements. Early writers frequently failed to differentiate between hyperopia and presbyopia, and Donders¹¹ appears to have been the first worker to clarify the differences between these two refractive conditions. Interestingly, Levene⁷⁶ noted that in 1623, while discussing presbyopia, Daça de Valdes, a licentiate and notary public of the Court of the Holy Office in Seville, Spain, observed, "Sometimes the sight of old people is so greatly weakened that they are even unable to see far away and many need to have glasses to see at a distance." In addition, in 1696, Hamberger¹⁷ taught that "presbyopia" could occur in the young and even congenitally. These citations appear to be some of the earliest references to hyperopia. For further reviews of the history of this refractive condition, see Duke-Elder and Abrams¹⁷ and Levene.⁷⁶

Grosvenor⁹² observed that hyperopia has received considerably less attention than myopia, possibly because its etiology is generally believed to be almost entirely due to genetic or hereditary factors, with environmental influences having no more than a minimal role. It may produce reduction in both far and near visual acuity, depending on the patient's accommodative ability, although the greatest symptoms typically occur at near.

Hyperopia in children has been associated with poor reading ability, low intelligence test scores, learning difficulties, and delay in visual perceptual skills development.⁹²⁻⁹⁹ However, the reason for these associations is unclear. Hirsch⁹⁶ suggested the following four hypotheses to account for the weak but statistically significant correlation between refractive error and intelligence test

scores, with myopes performing significantly better than hyperopes:

1. Hyperopia and myopia may represent underdevelopment and overdevelopment of the eye, respectively, with ocular and cerebral development being related.
2. Intelligence test scores may be associated with the amount of reading done, with myopes reading more than hyperopes.
3. The more intelligent child might read more, with the result that he or she becomes myopic. Conversely, the less intelligent child might read less and avoid becoming myopic; that is, he or she might remain hyperopic.
4. Many intelligence tests require the child to perceive fine detail at near vision for a prolonged period of time. This may place the myopic child at an advantage, because the accommodative requirement is reduced, particularly if the individual is uncorrected. A subsequent study by Grosvenor⁹⁴ using an intelligence test that did not require any reading indicated no significant difference between myopes and hyperopes, suggesting that there is a relationship between reading ability and refractive error, rather than between intelligence and refractive error.

Borish⁸ listed a number of systems for classifying hyperopia:

- Anatomical features
- Degree of hyperopia
- Physiological and pathological hyperopias
- Action of accommodation

Classification by Anatomical Features

Borish⁸ indicated that, like myopia, hyperopia could be:

- *Axial*, in which the axial length is too short for the refractive power of the eye.
- *Refractive*, in which the refractive system is underpowered with respect to the axial length of the eye.

Borish further divided refractive hyperopia into:

- *Index hyperopia*, in which one or more of the refractive indices of the media are anomalous.
- *Curvature hyperopia*, in which the increased radius of curvature of one or more refractive surfaces produces a decrease in refractive power.
- *Anterior chamber hyperopia*, in which decreased anterior chamber depth decreases the refractive power of the eye.

Additional anatomical factors that would produce hyperopia include the absence of a refractive element (e.g., aphakia) and the displacement of a refractive element (e.g., lateral displacement of the crystalline lens, producing partial aphakia).

Classification by Degree of Hyperopia

Hyperopia may be classified as:

Low	(0.00 to +3.00 D)
Medium	(+3.12 to +5.00 D)
High	(>+5.00 D)

However, this method of classification provides little information unless accompanied by knowledge of the patient's accommodative ability (see Classification by the Action of Accommodation).

Classification into Physiological and Pathological Hyperopias

As noted earlier, physiological ametropias may be defined as nonpathological, whereas pathological refractive errors are anomalies that lie outside the limits of normal biological variation. A reduction in axial length may occur as a result of the presence of a space-occupying lesion within the eye, such as a tumor, hemorrhage, edema, or pathological flattening of the cornea (e.g., *cornea plana*).

Classification by the Action of Accommodation

Because hyperopia results from a relatively underpowered eye with respect to its axial length, an increase in accommodation (i.e., a temporary increase in the dioptric power of the crystalline lens) may serve to compensate, at least partially, for this refractive error. For example, if a young, healthy patient with +2.00 D of hyperopia wishes to view a distant object of regard, accommodating, that is, increasing the refractive power of the lens by +2.00 D, will allow the distant object to be imaged upon the retina. (For simplicity, the depth of focus is assumed to be zero.) However, if this same patient were to view a near object located 50 cm in front of the eye, he or she would have to accommodate another +2.00 D (i.e., a total of +4.00 D) to view this near stimulus. In comparison, an emmetrope or -2.00 D myope would have to accommodate only 2 D or 0 D, respectively, to view the near target. Because this extra accommodation will be accompanied by accommodative convergence, hyperopia is frequently accompanied by near-vision asthenopia and either esophoria or esotropia resulting from the excessive accommodation and vergence at near.

Accordingly, hyperopia may be classified with regard to the action of accommodation as follows:

- *Latent hyperopia*—Hyperopia that is masked by accommodation and is not revealed by noncycloplegic refraction. A cycloplegic agent is necessary to uncover the full amount.
- *Manifest hyperopia*—Hyperopia indicated by the maximum plus lens that provides the optimum distance visual acuity.

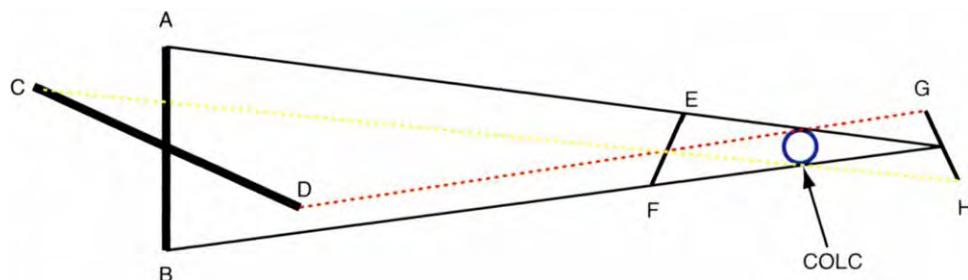


Figure 1-4

Example of against-the-rule astigmatism. Light from a point source is incident upon refracting surface ABCD, where the horizontal meridian (CD) has greater dioptric power than the vertical meridian (AB). Accordingly, a vertical focal line is formed at EF in the plane where the horizontal rays (dotted lines) are brought to a focus, and a horizontal focal line is formed at GH, where the vertical rays (solid lines) are brought to a focus. The circle of least confusion (COLC) occurs at the dioptric midpoint between the two focal lines.

- **Total hyperopia**—The sum of latent and manifest hyperopia. Total hyperopia may be further divided into facultative and absolute hyperopia.
- **Facultative hyperopia**—Hyperopia that is masked by accommodation but can be revealed by noncycloplegic refraction.
- **Absolute hyperopia**—Hyperopia that cannot be compensated for by accommodation, that is, the portion of the refractive error that exceeds the amplitude of accommodation. For example, an 8.00 D hyperope with an amplitude of accommodation of 5.00 D has 3.00 D of absolute hyperopia.

ASTIGMATISM

The term *astigmatism* (from *a*, meaning “privative” or “lacking,” and *stigma*, meaning “a point”) was suggested to describe this anomaly by Dr. William Whewell (1794–1866), Master of Trinity College, Cambridge.⁷⁶ Sir Isaac Newton appears to have been the first to describe this anomaly in his *Lectiones Opticae*, a treatise based on a series of lectures presented in Cambridge between 1670 and 1672.¹⁰⁰ In discussing obliquely incident rays of light upon a refracting surface, Newton noted that “there are principally two centers of radiation” (or foci).¹⁰⁰ Newton also made reference to what was to become known as the *circle of least confusion*, the dioptric midpoint between the two foci, noting, “We ought to take for the sensible image some single point in that it occupies the middle of all the light proceeding from there toward the eye, and that lies approximately midway between the points D and ø” (the two foci).¹⁰⁰

Although Bennett¹⁰¹ patriotically claimed that astigmatism was a British invention that remained practi-

cally a British monopoly for nearly 150 years,* there were early references to this phenomenon by writers of other nationalities. For example, in an article published in Paris in 1694, de La Hire⁷⁶ noted the effects of tilting the crystalline lens and later correctly reported that the image of a circular object would appear as an oval. Thomas Young, writing in 1800, appears to have been the first to consider the concept of line foci. Furthermore, he measured his own astigmatism using an optometer and verified that it was not corneal in origin by neutralizing his cornea’s refractive power.⁷⁶ For a full review of the history of the discovery of astigmatism, see Levene,⁷⁶ Bennett,¹⁰¹ and Donders.¹¹

Astigmatism may be classified as follows:

- As regular or irregular
- With respect to the contributing ocular component
- By orientation
- With respect to the refractive error

Classification into Regular and Irregular Astigmatism

In regular astigmatism, the meridians having the maximum and minimum refractive powers are separated by an angle of 90 degrees (Figure 1-4). Hence, the primary meridians are perpendicular or orthogonal. In irregular (also called bi-oblique)¹⁷ astigmatism, the maximum and minimum powered meridians are separated by an angle other than 90 degrees. Significant irregular astigmatism, which fortunately is relatively

*Indeed, Trevor-Roper¹⁰² noted that on the continent of Europe, astigmatism was described as the “English disease” because of the tendency of English practitioners to prescribe weak correcting lenses. However, he suggested that this was probably due to the enthusiasm of the vendor, rather than to any frailty or oversensitivity of the English eye!

uncommon, may be found in conditions such as a scarred cornea or keratoconus (see Chapter 34).

Classification with Respect to Contributing Ocular Component

The Anterior Cornea

Astigmatism is most frequently produced by the toricity of the anterior corneal surface. Because the air/tear film interface represents the largest change in refractive index, variations in radii of curvature at this interface produce the greatest dioptric effect (see also Chapter 17). Several workers have demonstrated that external pressure either from the eyelids or from pathological structures (e.g., chalazia or tumors) can produce anterior corneal astigmatism.^{17,103}

The Posterior Cornea

Tscherning¹⁰⁴ and Bannon and Walsh¹⁰⁵ indicated that the posterior corneal surface may also contribute significantly to astigmatism. Bannon and Walsh provided the example of a cornea that, when measured with conventional keratometry, gave readings of 43.00 D horizontally and 46.00 D vertically. Assuming that the anterior and posterior surfaces remained parallel to each other, they calculated that this cornea would have +3.35 D of anterior surface astigmatism and -0.45 D of posterior surface astigmatism. However, because the toricity of the posterior cornea is difficult to measure in the clinical setting, its relatively small contribution is generally ignored (see Chapters 17 and 26 concerning keratometric readings and their derivation).

The Crystalline Lens

Astigmatism may be produced by the toricity of the lens surfaces or tilting of the lens.¹⁰⁶ Duke-Elder and Abrams noted that both the anterior and posterior lenticular surfaces frequently exhibited astigmatism.¹⁷ However, the magnitude was typically small and in the direction opposite to that of the corneal astigmatism. Tscherning¹⁰⁴ observed that a small amount of astigmatism results from physiological tilting of the crystalline lens axis, with 3 to 7 degrees of rotation about the vertical axis and up to 3 degrees of tilt about the horizontal axis, with the top of the lens lying anteriorly to the inferior portion. This would result in approximately 0.25 D of against-the-rule astigmatism (see Classification by Orientation).

Other Possible Causes

Gullstrand observed that the fovea is not normally located on the optic axis (a line passing approximately through the centers of curvature of the refractive surfaces of the eye) but is usually displaced both temporally and inferiorly.¹⁰⁷ Taking a value of 5 degrees for angle

alpha, that is, the angle between the optic axis and the visual axis (a line passing from the point of fixation to the fovea), this would produce 0.10 D of oblique astigmatism.

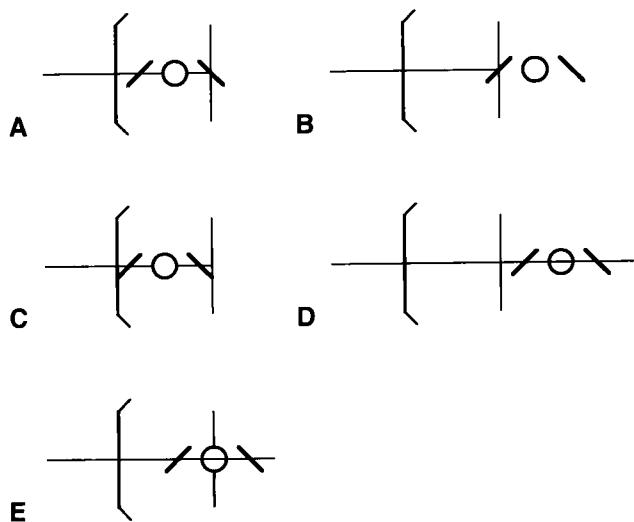
Flüeler and Guyton¹⁰⁸ verified that a tilted retina does not cause astigmatism. Although the optical imagery from this condition appears to resemble astigmatic blur, the effect is best described as induced field curvature. For example, if a retina is tilted about its vertical meridian, a vertical line image will be focused on the retina, whereas the horizontal line image will be defocused. However, in contrast to an astigmatic eye, the size of the horizontal blur circle away from the vertical line image will vary with increasing eccentricity.

Classification by Orientation

If the corneal meridian that has the least refractive power is horizontal (± 20 degrees), that is, between 160 and 20 degrees, this is described as *with-the-rule astigmatism*. If the corneal meridian that has the least refractive power is vertical (± 20 degrees), that is, between 70 and 110 degrees, this is described as *against-the-rule astigmatism*. If the corneal meridian that has the least refractive power lies either between 20 and 70 degrees or between 110 and 160 degrees, this is described as *oblique astigmatism*. It should be noted that, in regular astigmatism, the corneal meridian that has the least refractive power is the meridian that has the larger or flatter radius of curvature and the same orientation as the axis of the minus correcting cylinder. The meridian having the most refractive power is that with the smaller or steeper radius of curvature and is oriented perpendicular to the axis of the minus correcting cylinder.

Classification with Respect to the Refractive Error

Astigmatism may also be classified with respect to the relative position of the retinal images of a distant object under conditions of minimal accommodation (Figure 1-5). If one image is located in the retinal plane, this is referred to as *simple astigmatism*. Depending on the relative location of the image that is focused away from the retina, the ametropia may be classified as *simple myopic astigmatism* (Figure 1-5, A) or *simple hyperopic astigmatism* (Figure 1-5, B). If neither image is located in the plane of the retina, but both are either in front of or behind the retina, this is referred to as *compound astigmatism*. Depending on the location of the two images, the ametropia may be classified as *compound myopic astigmatism* (Figure 1-5, C) or *compound hyperopic astigmatism* (Figure 1-5, D). If one image lies in front of the retina and the other lies behind it, this is referred to as *mixed astigmatism* (Figure 1-5, E).

**Figure 1-5**

Classification of astigmatism based upon the refractive error. A, Simple myopic astigmatism. The front focal line is anterior to the retina, whereas the back focal line coincides with the retina. B, Simple hyperopic astigmatism. The front focal line coincides with the retina, whereas the back focal line lies posterior to the retina. C, Compound myopic astigmatism. Both focal lines lie in front of the retina. D, Compound hyperopic astigmatism. Both focal lines lie behind the retina. E, Mixed astigmatism. One focal line lies in front of and the other behind the retina.

ANISOMETROPIA

Anisometropia is a difference between the refractive states of the two eyes that occurs in one or both principal meridians.¹⁰⁹ This becomes clinically significant when its magnitude reaches approximately 1 D in either or both of the principal meridians. Levene⁷⁶ noted that this condition was recognized by De Valdez in 1623, who attributed it to the use of a single eyeglass rather than spectacles. Furthermore, in 1743, Buffon suggested that this condition would result in strabismus because of the differences in visual acuity. Clinical management of anisometropia is covered in Chapter 32.

Optical difficulties in anisometropia may result from three principal factors¹¹⁰:

- A difference in induced prism (by decentration) through the correcting spectacle lenses between the two eyes when the gaze is directed away from the optical centers of the lenses.
- A difference in the stimulus to ocular accommodation between the two eyes (when corrected with spectacles).
- A difference in spectacle magnification between the two eyes.

Anisometropia may be classified as follows^{8,111}:

- By refractive error

- By magnitude
- By etiology
- By the contributing ocular components

Classification by Refractive Error

On the basis of refractive error, anisometropia may be categorized as:

- Isoanisometropia: Both eyes are either hyperopic (*anisohyperopia*) or myopic (*anisomyopia*).
- Antimetropia: One eye is myopic and the other is hyperopic.

Classification by Magnitude

Gettes¹¹² reported that patients' symptoms typically vary with the magnitude of the dioptric difference between the two eyes, as indicated below:

- 0 to 2 D (*low*): The patient usually tolerates full spectacle correction with little difficulty.
- 2 to 6 D (*high*): The patient is likely to have binocular problems.
- >6 D (*very high*): The patient is typically asymptomatic, frequently because of the presence of central suppression.¹¹³

Table 1-1 presents results from a study by Rayner¹¹⁴ on the prevalence of degrees of anisometropia using data drawn from spectacle prescription orders.

Classification by Etiology

On the basis of etiology, anisometropia may be classified as:

TABLE 1-1 Prevalence of Degrees of Anisometropia from a Survey of 5444 Spectacle Prescription Orders

Dioptric Range	Spherical Anisometropia (%)	Cylindrical Anisometropia (%)
0–0.5	79.8	86.8
0.62–1.00	11.8	8.1
1.12–1.50	3.4	2.3
1.62–2.00	1.9	1.3
2.12–2.50	1.0	0.6
2.62–3.00	0.7	0.3
3.12–3.50	0.4	0.2
3.62–4.00	0.3	0.1
>4.00	0.7	0.3

From Rayner AW. 1966. Aniseikonia and magnification in ophthalmic lenses. Problems and solutions. Am J Optom Physiol Opt 43:619. © The American Academy of Optometry, 1966. Note that the prevalence of spherical anisometropia exceeding 1 D is approximately 8.4%.

- **Hereditary.** Hereditary anisometropias include those due to congenital glaucoma, congenital cataracts, and conditions causing eyelid closure, such as congenital third nerve palsy, ptosis, and soft-tissue swelling of the periorbital tissues after obstetric trauma.^{115,116}
- **Acquired.** Acquired anisometropias include those following trauma; space-occupying lesions in and around the globe; and iatrogenic factors such as monocular lens extraction (unilateral aphakia), refractive surgery, penetrating keratoplasty, craniocerebral erosion (growing skull fracture).^{117–119}

Classification by Contributing Ocular Component

Sorsby et al.¹²⁰ measured the ocular components of 68 anisometropic patients and concluded that axial length was the most significant contributing factor to anisometropia. Their results are summarized below.

Axial Length. Differences in axial length between the two eyes were observed in 97% of the cases examined, particularly in patients who had greater than 5 D of anisometropia. In 86% of these high and very high anisometropes, differences in axial length contributed more than 80% of the refractive difference. In some subjects, the difference in axial length actually exceeded the refractive difference, and this effect was counterbalanced by the cornea. Reanalyzing data from Sorsby et al.¹²⁰ and van der Torren,¹²¹ Laird¹²² found correlation coefficients of 0.94 and 0.71, respectively, when comparing axial length with anisometropia.

Crystalline lens. Lenticular anisometropia was typically observed in individuals who had between 3 and 5 D of anisometropia. In only one individual did the lenticular anisometropia counter the axial differences.

Cornea. Sorsby et al.¹²⁰ noted that in general the cornea was not a significant factor in anisometropia. Indeed, in 10 subjects, corneal anisometropia was in the opposite direction to the axial anisometropia. In these individuals, the corneal power tended to reduce the anisometropia rather than contribute to it.

COMPONENTS OF REFRACTION AND THEIR CORRELATION

Hirsch¹²⁶ noted that variability in any of 13 individual elements—six refractive surfaces, five indices of refraction, and two linear distances—could influence the refractive status of the eye. However, Curtin¹⁵ observed that four of these variables were the most influential, namely corneal and crystalline lens power, anterior chamber depth, and axial length of the eye. The combined refractive power of the cornea and crystalline lens, in conjunction with the physical separation of these elements (i.e., the anterior chamber depth) determines

the refractive power of the eye and, accordingly, the location of the second principal focus. If the axial length of the eye does not correspond with this secondary focal length, ametropia is the result.

Corneal Power

Steiger²¹ conceptualized emmetropia as a locus occupying a position between myopia and hyperopia along a biological continuum. He measured the radii of curvature of 5000 corneas using the Javal–Schiøtz ophthalmometer, and observed that the values of corneal radii were normally distributed. On the basis of these findings, Steiger suggested that all of the ocular components of the eye—such as axial length, corneal curvature, and anterior chamber depth—would be characterized by their own frequency distribution curve. Because the refractive state of the eye results from the interaction of these components, the distribution of refractive error should reflect the variability of the individual components. This became known as the biological variability theory (or, more simply, the biological theory) for development of refractive error.

Although subsequent studies have clearly demonstrated that neither the axial length of the eye nor its refractive error are normally distributed (see Figures 1-9 and 1-10), Steiger's observations on the normal distribution of corneal power have been supported by other investigations.^{14,22,124,125} The distribution of corneal powers reported by Stenstrom is illustrated in Figure 1-6.²²

A number of investigators have reported that the cornea generally reaches its adult dioptric power around 4 years of age, although Keeney¹²⁶ suggested that this may occur even earlier, at approximately 2 years of age. Sorsby et al. indicated that changes in corneal power were trivial between 3 and 13 years of age, and this finding was reproduced by Zadnik et al.^{42,127} It has also

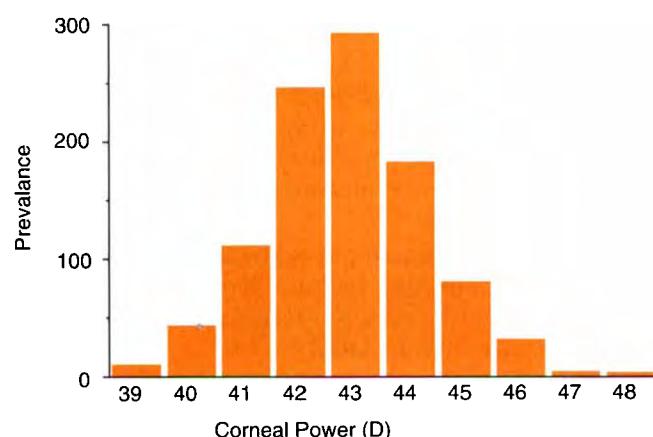


Figure 1-6

Distribution of corneal refractive power reported by Stenstrom.²² The observation of a normal distribution of this refractive component is consistent with the findings of other studies (e.g., Sorsby et al.¹⁴ and Tron¹²⁵).

been demonstrated that variations in the dioptric power of the cornea do indeed contribute to the development of refractive error in some individuals. For example, Sorsby et al.¹⁴ reported mean corneal powers in populations of hyperopes (+0.50 to +4.00 D), emmetropes, and myopes (-0.50 to -4.00 D) of 42.86, 43.25 and 44.04 D, respectively. In addition, Goss and Jackson⁷³ recorded keratometry findings in a 3-year longitudinal study of refractive error development. Subjects were divided into those who became myopic during the course of the study and those who remained emmetropic. The mean horizontal corneal powers for the two groups were 44.22 D and 43.51 D, respectively. This difference was statistically significant.

Although other studies have supported this observation of increased corneal power in myopes, it has not been a consistent finding across investigations.^{11,21,71,128,129} This variability is consistent with the notion that changes in corneal power contribute only to a portion of all refractive errors. Indeed, Stenstrom²² observed a weak negative correlation between corneal power and refractive error ($r = -0.18$), although further analysis by Hirsch and Weymouth¹³⁰ indicated that if axial length and anterior chamber depth measurements were held constant, the coefficient of correlation improved to -0.70. Accordingly, it is apparent that variations in corneal curvature may play a significant role in the development of refractive error in at least a limited number of individuals.

Crystalline Lens Power

Zadnik et al.¹²⁷ noted that few investigators have actually measured all parameters of the crystalline lens and most have calculated lens power or curvatures from the measurements of the other ocular components. For example, Stenstrom²² measured corneal curvature, anterior chamber depth, axial length, and refractive error, and calculated the crystalline lens power from these directly measured parameters. Stenstrom reported no significant correlation ($r = 0.00$) between refractive error and lens power. Sorsby et al.⁴² measured both the anterior and posterior radii of curvature and calculated the lens thickness from these data in both cross-sectional and longitudinal investigations of children between 3 and 16 years of age. In their cross-sectional study, they demonstrated that the mean crystalline lens power declined from around 20.8 D at age 3 years to approximately 20.0 D at 15 years of age. This was later confirmed by Zadnik et al.,¹²⁷ who showed that the reduced lens power resulted from a flattening of both the anterior and posterior radii of curvature (Figure 1-7). Interestingly, Zadnik et al. also observed a decline in lens thickness (measured directly using A-scan ultrasound), particularly between 6 and 8 years of age. This observation of lens thinning had been alluded to earlier by Sorsby et al.,⁴² and was also observed by Larsen (Figure

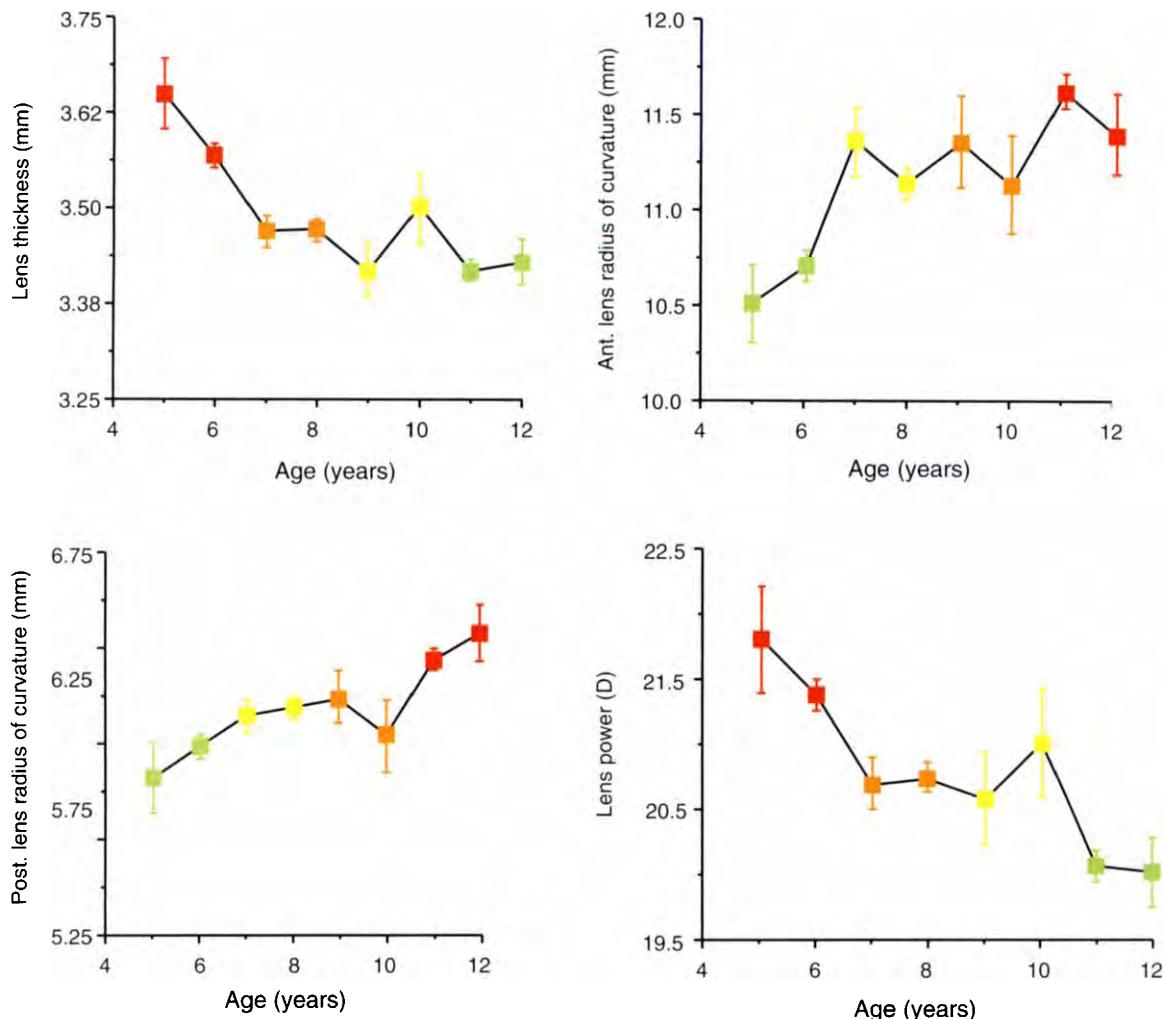
1-8).¹³¹ From 10 years of age onwards, the anterior radius of curvature steepens and the central thickness increases throughout adulthood.^{132,133}

One parameter that has received less attention because of the difficulty of measurement *in vivo* is the refractive index of the lens. The refractive index varies within this tissue because of the variation in protein density, which increases toward the center of the lens structure.^{134,135} Pierscionek¹³⁶ stated that increased lens power during adulthood might be predicted to produce a steady increase in myopia with age. However, this is not typically observed, and the absence of a myopic shift might be due, at least in part, to subtle changes in the cortical refractive index gradient.^{137,135} Borish⁸ noted that a change in the assumed single value of crystalline lens refractive index of ± 0.004 would result in a shift in the ocular refraction of ± 0.85 D. Therefore, relatively small changes in the refractive index could produce substantial variations in ametropia. In an investigation of the equivalent refractive index of the crystalline lens in children, Mutti et al.¹³⁸ compared three different refractive index profiles: (1) the Gullstrand-Emsley schematic index, (2) a 10-shell gradient index model, and (3) the equivalent refractive index required to produce agreement between the measured refractive error and the ocular components.^{107,134,135,138,139} Mutti et al.¹³⁸ observed that the Gullstrand-Emsley value of refractive index ($n' = 1.416$) gave significantly lower values of crystalline lens power than did the other two techniques, suggesting that higher values would be more appropriate in children.

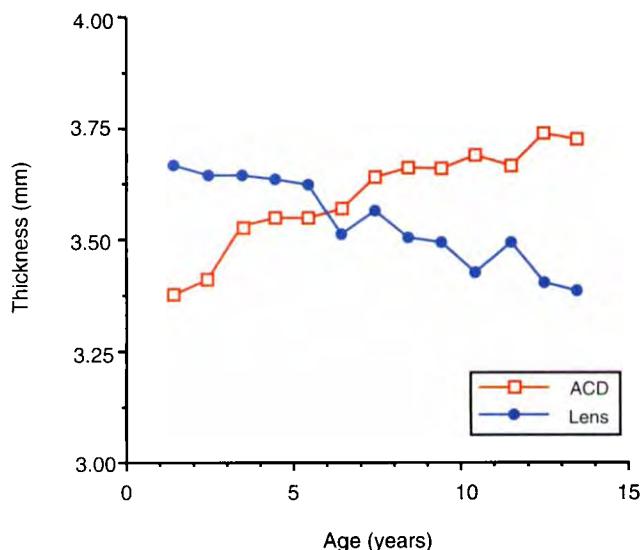
Examinations of the relationship between the overall crystalline lens power and refractive error have yielded conflicting findings. In 1911, Zeeman¹⁵ reported flatter anterior and posterior lens radii of curvature in myopic eyes compared with emmetropic eyes. Garner et al. also found significantly decreased crystalline lens power in myopic eyes.¹²⁹ However, in line with Stenstrom,²² Grosvenor and Scott⁷ observed no significant difference in either lens power or lens thickness among emmetropes, early adult-onset myopes, and youth-onset myopes. Similar findings were reported by Bullimore et al.⁶⁷ Although McBrien and Millodot⁴⁵ found a significant difference in lens thickness between emmetropes and late-onset myopes, with the late-onset myopic group having thinner lenses, they also observed that a group of early-onset myopes, who had a higher mean refractive error than the late-onset group, actually had thicker lenses than did the emmetropes. Indeed, Tron¹²⁵ noted the high variability of crystalline lens measurements, with the range of observed lens refractive powers exceeding that of the cornea, even though the mean absolute power of the lens was less than 50% of the corresponding corneal value.

Anterior Chamber Depth

Tron¹²⁵ and Stenstrom²² observed that the variation in the depth of the anterior chamber is normally distrib-

**Figure 1-7**

Mean changes in crystalline lens thickness, anterior (*Ant.*) and posterior (*Post.*) radii of curvature, and lenticular refractive power between 5 and 12 years of age. Data are from a cross-sectional study by Zadnik K, Mutti DO, Friedman NE, Adams AJ. 1993. Initial cross-sectional results from the Orinda Longitudinal Study of Myopia. *Optom Vis Sci* 70:750–758.

**Figure 1-8**

Mean changes in lens thickness (*closed circles*) and anterior chamber depth (*ACD, open squares*) in 465 boys between 6 months and 13 years of age. Data are from Larsen JS. 1971. The sagittal growth of the eye. Pt. II. Ultrasonic measurement of the axial diameter of the lens and the anterior segment from birth to puberty. *Acta Ophthalmol* 49:427–440. The lens thinning is also apparent here (see Figure 1-7), as reported by Zadnik K, Mutti DO, Friedman NE, Adams AJ. 1993. Initial cross-sectional results from the Orinda Longitudinal Study of Myopia. *Optom Vis Sci* 70:750–758.

uted. This is consistent with the normal distributions of anterior corneal radius of curvature and crystalline lens power. Up to approximately 13 years of age, the anterior chamber depth appears to increase.^{42,131} However, Larsen¹³¹ observed that this increase is accompanied by lens thinning, so that the distance from the cornea to the posterior pole of the crystalline lens remains relatively constant. This is illustrated in Figure 1-8. Between 20 and 70 years of age, the anterior chamber depth decreases from approximately 4.0 mm to around 3.5 mm, because of the age-related increase in lens thickness.¹⁴⁰ Koretz et al.¹⁴⁰ verified that the reduced chamber depth was due entirely to the increased lens thickness by demonstrating that the cornea to posterior lens distance showed no significant change with age.

Hirsch and Weymouth¹³⁰ and Borish⁸ indicated that increased anterior chamber depth should decrease the refractive power of the eye, because it has the effect of increasing the separation between the two major ocular refractive elements. Accordingly, one might predict that refractive myopia is associated with decreased anterior chamber depth. However, Erickson¹⁴¹ pointed out that the effect of changes in chamber depth on the refractive error depends on the causative structure. If an increase in anterior chamber depth is produced by the anterior lens shifting posteriorly by 0.1 mm (with the overall axial length of the eye remaining constant), this will produce a 0.13 D increase in hyperopia. Conversely, if a 0.1-mm increase in chamber depth results from the growth of the cornea away from the lens, which will also produce a 0.1-mm increase in the axial length of the globe, then a 0.14 D increase in myopia results.

These observations indicate that variations in anterior chamber depth cannot be considered in isolation, but rather must be examined in conjunction with the resulting changes in axial length.¹⁴² For example, myopia may result from a decrease in anterior chamber depth with no accompanying change in axial length, an increase in anterior chamber depth with an increase in axial length, or an increase in axial length with no change in anterior chamber depth. This is confirmed by the finding that myopia (or less hyperopia) has been associated with both increased and decreased anterior chamber depth. Hirsch and Weymouth¹³⁰ concluded that only 7% of the variance in the refractive state can be accounted for by variations in the depth of the anterior chamber.^{68,71,143-145}

Axial Length

Duke-Elder and Abrams¹⁷ cited Plempius in 1632 as being the first to demonstrate that the myopic eye had a greater axial length than its emmetropic counterpart. Subsequently, Arlt in 1856 observed that enucleated myopic eyes were long and pear shaped, with thinning

of the posterior segment of the sclera. Steiger²¹ and Tron¹²⁵ were unable to measure the axial length in living eyes directly but calculated this parameter from measurements of the other components. Using such calculations, Tron indicated that the frequency distribution of axial lengths was not normal, but had a high peaked curve (i.e., leptokurtotic) and was asymmetric, including a larger number of longer eyes. However, if eyes having more than 6 D of myopia were excluded, a normal distribution of axial lengths was observed. Using Tron's data, Wibaut¹⁴⁶ reported a high correlation ($r = -0.76$) between the axial length and refractive power of the eye.

Stenstrom²² used the roentgenographic (x-ray) method described by Rushton¹⁴⁷ to measure axial length directly, and also reported a leptokurtotic and skewed distribution (Figure 1-9). Stenstrom observed a correlation of +0.61 between the reciprocal of the axial length (refractive index divided by axial length is equal to the required vergence of the emergent ray bundle after refraction to be imaged upon the retina) and the total refractive power of the eye. Subsequent reanalysis of Stenstrom's data by Hirsch and Weymouth¹³⁰ indicated that if the corneal radius and anterior chamber depth measurements were held constant, the correlation improved to +0.87. However, Borish⁸ pointed out that Stenstrom's data included a significant number of high refractive errors (53 subjects greater than ± 5 D), which might have falsely increased the correlation coefficient.

Keeney¹²⁶ observed that the axial length of the fetal eye increases from approximately 14 mm up to 17 mm during the third trimester in utero, and this finding of 17.0 to 17.5 mm axial length at birth was supported by the observations of Scammon and Wilmer,¹⁴⁸ Sorsby et al.,⁴² and Sheridan,¹⁴⁹ although Ellerbrock¹⁵⁰ quoted a range of 15.8 to 17.5 mm. Sorsby et al.⁴² noted a period of very rapid growth up to 3 years of age, with the axial length increasing around 5 mm to approximately 23 mm. The rate of growth then slowed dramatically, with axial elongation of about 1 mm occurring between 3 and 13 years of age. Similar results were reported by Larsen⁴¹ in measurements of the vitreous chamber. He observed a mean increase of 3.67 mm during the first 3 to 4 years of life, with a subsequent mean increase in vitreous chamber depth of 1.94 mm between 4 and 14 years of age. Although a number of studies have suggested that the length of the globe reaches its adult size and stabilizes around 15 years of age (i.e., concurrent with the cessation of bodily growth), more recent investigations have verified that young adult-onset myopia, that is, myopia that first manifests after 18 years of age following a period of refractive stability, results from an increase in axial length.* However, it is unclear whether axial growth never ceased in these individuals,

*References 7, 24, 45, 48, 65, 67, 151.

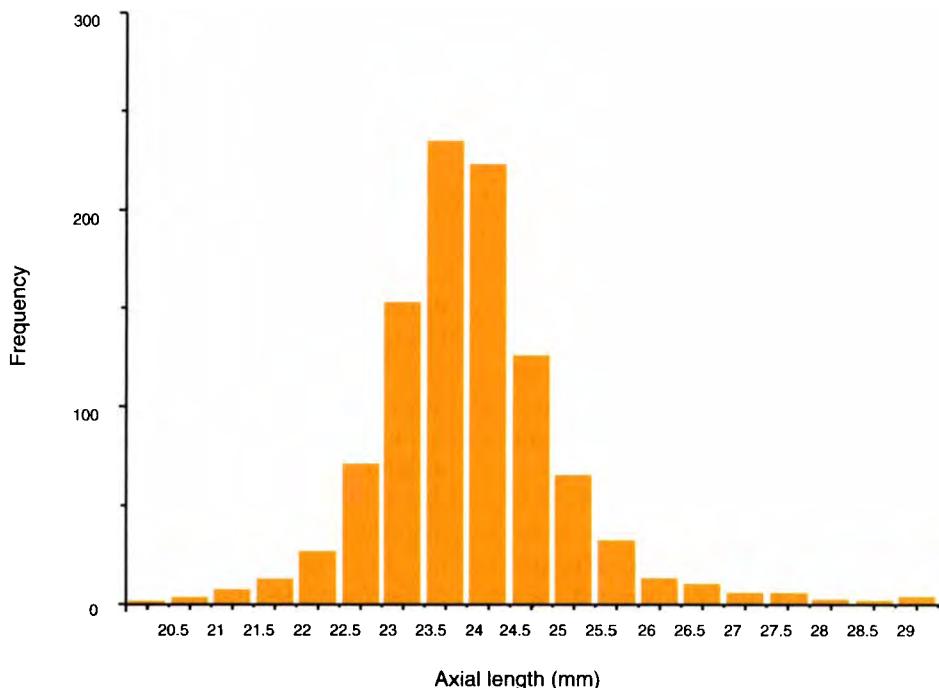


Figure 1-9

Distribution of the axial length of 1000 eyes measured using a radiographic technique. The range of axial lengths is not normally distributed but rather is leptokurtotic and skewed toward increased axial diameter. Data are from Stenstrom S. 1948. Investigation of the variation and correlation of the optical elements of human eyes (translated by Woolf D). *Am J Optom Arch Am Acad Optom* 48:218–232, 286–299, 340–350, 388–397, 438–449, 496–504. Similar findings were reported by Tron EJ. 1940. The optical elements of the refractive power of the eye. In Ridley F, Sorsby A (Eds), *Modern Trends in Ophthalmology*, pp 245–255. New York: Paul B. Hoeber. and Sorsby A, Benjamin B, Davey JB, et al. 1957. *Emmetropia and Its Aberrations* (Special Report Series Medical Research Council No. 293). London: Her Majesty's Stationery Office.

or whether elongation of the globe occurred after a period of component stability.

CORRELATION OF OCULAR COMPONENTS AND EMMETROPIZATION

Although it is of interest to consider each of the ocular components and their relation to the development of refractive error individually, it is clear that the components' combined interactive effect must also be examined. For example, both an eye with a relatively short axial length and high total refractive power, and an eye with a relatively long axial length and low total refractive power may be emmetropic. Thus, the refractive error of the eye cannot necessarily be predicted from knowledge of the dimensions of a single biometric component. Evidence for this observation comes from examination of the wide range of axial lengths in emmetropic eyes. In 1895 Schnabel and Herrnheiser,¹⁵ using postmortem material, obtained axial length measurements ranging from 22 to 25 mm in emmetropic eyes. Similar broad distributions were also reported by Tron,¹²⁵ Deller et al.,¹⁵² Stenstrom,²² and Sorsby et al.¹⁴

Upon examining the typical range of refractive errors, several investigators have observed that this parameter is not normally distributed, but rather is markedly leptokurtotic and skewed toward myopia (Figure 1-10). The apparently excessive prevalence of emmetropia (compared with a statistically "normal" distribution) has led to the proposal of an active emmetropizing process in which the growth of one or more ocular biometric components can compensate for variations in the dimensions of another component.

Stenstrom²² and Sorsby et al.¹⁴ examined the correlations between the ocular components, and their findings are presented in Table 1-2. However, both Van Alphen³⁵ and Zadnik et al.¹²⁷ noted that because some of these parameters were not actually measured, but rather were computed from the other components, a number of these correlations were spurious. Stenstrom⁷⁰ did not measure crystalline lens power and Sorsby et al.⁷¹ failed to measure axial length. In addition, it is of interest that Stenstrom observed a high correlation ($r = -0.84$) between refractive error and the AL/CR ratio. This observation supports the findings of Grosvenor⁶⁸ and Grosvenor and Scott.⁴⁴

Accordingly, there is some suggestion that during the period of ocular growth, an increase in the axial length of the globe may be accompanied by a reduction in the

power of either the cornea or crystalline lens in order to maintain an emmetropic refractive error. For example, Hirsch¹⁵³ noted that between birth and 3 years of age, the axial length increases approximately 5 to 7 mm. Such an increase could produce a myopic shift on the order of 15 to 20 D. However, the fact that refractive error remains relatively stable during this period supports the notion of coordinated growth, whereby the

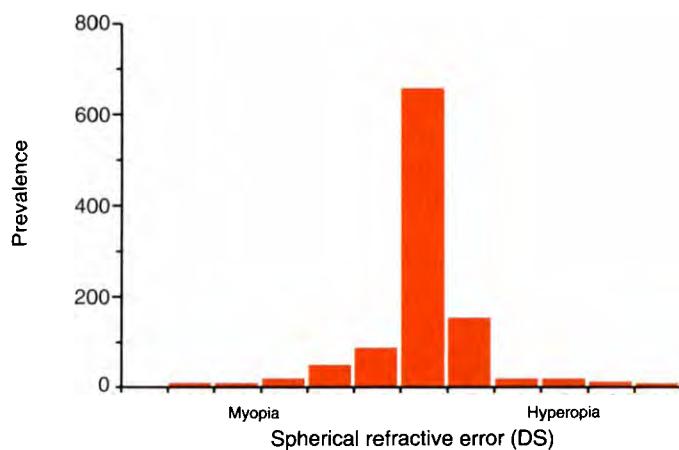


Figure 1-10

Distribution of the refractive errors of 1000 eyes. The range of ametropia is not normally distributed but rather is leptokurtotic and skewed toward increased myopia. Data are from Stenstrom S. 1948. Investigation of the variation and correlation of the optical elements of human eyes (translated by Woolf D). *Am J Optom Arch Am Acad Optom* 48:218–232, 286–299, 340–350, 388–397, 438–449, 496–504.

ocular components do not develop independently of one another.¹⁵⁴

Studies of the growth of the cornea (see Corneal Power) have indicated that this structure generally reaches its adult dioptric power during the first 4 years of life. This suggests that after 4 years of age, only the crystalline lens is available to compensate for any axial length changes.⁷¹ However, other studies have observed increased corneal power in early adult-onset myopia, implying that the cornea may in fact continue to change shape beyond early childhood. Alternatively, in young adulthood, the eye may no longer be able to compensate for an already increased corneal power.^{54,64} Myopia ensues when changes in refractive power fail to compensate for an increase in the axial length of the globe. Interestingly, Hirsch and Weymouth¹⁵⁵ suggested that the growth rate of the lens might be connected with development of the axial length of the globe, with both elements possibly being under the control of the same chemical mediators (e.g., growth hormones) or mechanical factors.

The observation that the growth of the cornea is completed by 4 years of age might suggest that any association between axial length and corneal curvature must develop at this early stage. Accordingly, Hirsch and Weymouth¹⁵⁵ proposed that an eye that will go on to develop a relatively long axial length will be large at this early stage in ocular development. This was confirmed by Zadnik et al.,⁷⁴ who observed that children who had two myopic parents tended to have significantly longer axial lengths even when their eyes were still emmetropic or hyperopic. Myopia may therefore ultimately develop from the normal growth of these larger eyes. These strong familial effects on refractive

TABLE 1-2 Correlations Between Ocular Biometric Parameters

For Refractive Errors Between:	Stenstrom ²² ±10 D	Sorsby et al. ¹⁴ ±8 D	Stenstrom ²² ±3 D	Sorsby et al. ¹⁴ ±3 D
Refraction and axial length	-.75	-.77*	-.45	-.59*
Refraction and ACD	-.34	-.46	-.40	-.50
Refraction and corneal power	-.19	-.30	-.21	-.26
Refraction and lens power	-.02*	+.28	+.13	+.42
Axial length and ACD	+.44	+.46*	+.45	+.39*
Axial length and corneal power	-.31	-.28*	-.52	-.51*
Axial length and lens power	-.39*	-.49*	-.60	-.60*
ACD and corneal power	+.09	+.19	+.09	+.14
ACD and lens power	-.26*	-.46	-.32*	-.44
Corneal power and lens power	-.10*	-.10	-.09*	-.09

Adapted from Van Alphen GWHM. 1961. On emmetropia and ametropia. *Ophthalmologica (Suppl)* 142:7. Reproduced with permission of S Karger AG, Basel.

Asterisks (*) indicate coefficients resulting from calculated parameters rather than from actual measurements of components. Data are from Stenstrom S. 1948. Investigation of the variation and correlation of the optical elements of human eyes (translated by Woolf D). *Am J Optom Arch Am Acad Optom* 48:218–232, 286–299, 340–350, 388–397, 438–449, 496–504; and Sorsby A, Benjamin B, Davey JB, et al. 1957. *Emmetropia and Its Aberrations (Special Report Series Medical Research Council No. 293)*. London: Her Majesty's Stationery Office. ACD, Anterior chamber depth.

error development were also observed by Pacella et al.¹⁵⁶ and Liang et al.¹⁵⁷

In a substantial investigation into the process of emmetropization, Van Alphen³⁵ reviewed and reanalyzed the data of both Stenstrom²² and Sorsby et al.¹⁴ Based on multiple regression analysis, he considered that the multiple correlations were essentially the result of a few independently acting variables. He suggested that in emmetropic subjects at least two independent factors are relevant:

- A factor (denoted S) determining the relationship between corneal power and axial length.
- A factor (denoted P) grouping axial length, lens power, and anterior chamber depth.

Van Alphen proposed that because of factor S there is a trend for larger eyes to have flatter corneas, and this association is essentially independent of refractive error. Factor P represents an underlying influence that tends to produce deeper anterior chambers and flatter lenses in larger eyes. However, in consideration of ametropia, Van Alphen introduced a third factor (denoted R). Factor R is associated with the resistance to intraocular pressure offered by the ciliary muscle–choroid layer. Van Alphen suggested that intraocular pressure was of significance in the determination of both corneal curvature and the axial length of the eye. If intraocular pressure is countered by both choroidal tension and scleral elasticity, the degree of choroidal tension could be a factor in the determination of the axial length. Van Alphen considered the ciliary muscle–choroid combination as a functional unit that could behave physiologically as a continuous sheet of smooth muscle. Therefore, high ciliary muscle tone will lower the tension on the sclera, whereas low ciliary muscle tone will result in scleral stretch. Van Alphen proposed that the process of emmetropization is achieved by a negative-feedback, self-focusing control system. Variations in ciliary muscle tone could produce changes in refractive error by interfering with this self-focusing mechanism. Thus, eyes of any size (factor S) that are hyperopic at birth will have to stretch (factor P) to become emmetropic. In this process, axial length is adjusted to the total refractive power. The degree of adjustment (factor R) determines the refraction and the shape of the globe. Accordingly, factor R represents the degree of ametropization, or the degree of adjustment of factor P with respect to factor S.

METHODS OF MEASURING THE OCULAR COMPONENTS

Optical Methods

Keratometry and Corneal Topography

These procedures are used to assess the radius of curvature, relative astigmatism, and integrity of the anterior

corneal surface. The optical principles and clinical techniques involved with these procedures are discussed fully in Chapter 17.

Ophthalmophakometry

Ophthalmophakometry determines the radii of curvature and relative positions of the cornea and crystalline lens surfaces. Two techniques of ophthalmophakometry are Tscherning's technique and the comparison procedure.

Tscherning's Technique. This method was first reported by Tscherning¹⁰⁴ and was described further by Emsley.¹⁰⁷ Tscherning's ophthalmophakometer (Figure 1-11) consisted of a telescope, a fixation device, and a series of lamps mounted on an arc around the axis of the telescope in a manner similar to a perimeter arc.

To Determine the Anterior Chamber Depth. Consider Figure 1-12. A bright lamp (L_1) positioned away from the visual axis and the telescope (T) are adjusted until the third Purkinje image of L_1 , formed by reflection at the anterior crystalline lens (A_2), is centered in the telescope. A fixation target is then placed at the bisector of L_1 and T. A second, dim lamp (L_2) is subsequently introduced so that an image of this lamp, formed by reflection at the corneal surface (at E), is also centered in the telescope and is therefore aligned with the crystalline image of L_1 . Because this second lamp is dim, the image of L_2 formed at the anterior crystalline lens surface cannot be detected. In $\Delta A_2' C_1 E$,

$$\frac{A_2' C_1}{C_1 E} = \frac{\sin \beta}{\sin s}$$

Therefore

$$A_2' C_1 = C_1 E \frac{\sin \beta}{\sin s} = r \frac{\sin \beta}{\sin s'}$$

where r = the corneal radius of curvature.

Now

$$\begin{aligned} A_1 A_2' &= A_1 C_1 - A_2' C_1 \\ &= r - A_2' C_1 \\ &= r - \frac{r \sin \beta}{\sin s} \end{aligned}$$

Therefore the apparent anterior chamber depth

$$(A_1 A_2') = r \left[1 - \frac{\sin \beta}{\sin s} \right]$$

where r = the corneal radius of curvature; β = half the angle between the telescope and the second, dim lamp (L_2); and s = half the angle between the telescope and the first, bright lamp (L_1).

The corneal radius of curvature may be determined using keratometry. If the apparent anterior chamber

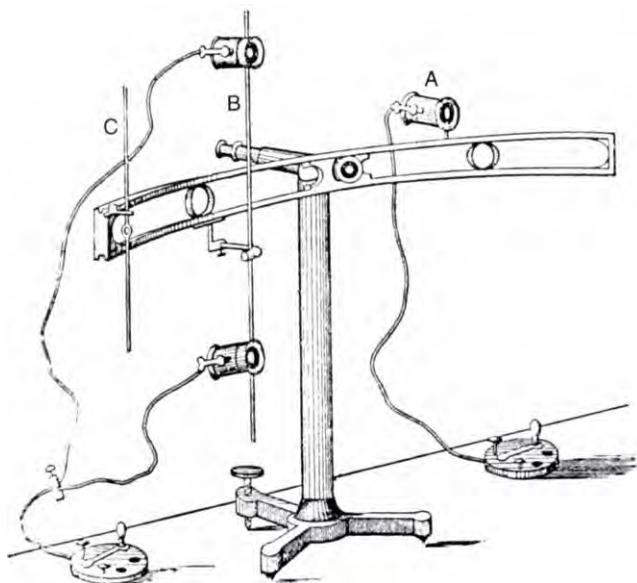


Figure 1-11

Ophthalmophakometer described by Tscherning.¹⁰⁴ The instrument is composed of a telescope, a fixation target, and a series of lamps of varying intensity that are mounted on an arc around the axis of the telescope. *A*, Cursor carrying a single lamp. *B*, Cursor carrying two lamps on the same vertical rod. *C*, Cursor carrying fixation target. (From Tscherning M. 1900. Physiologic Optics, p 64. Philadelphia: Keystone.)

depth = d' , the true anterior chamber depth (d) may be found from the following equation

$$\frac{n_2}{d} = \frac{1}{d'} + F_1$$

where n_2 = refractive index of the aqueous and F_1 = refractive power of the cornea.

To Determine the Radius of Curvature of the Crystalline Lens Surfaces. The procedure for determining the anterior lens radius of curvature is described here; a similar protocol may be used for the posterior surface.

Consider Figure 1-13. A bright lamp (L_1) is set immediately above the telescope (T) to one side of the fixation target. The second, dim lamp (L_2) is positioned so that the image formed by reflection at the cornea is aligned with the image of L_1 . In ΔC_1C_2E ,

$$\frac{C_1C_2}{C_2E} = \frac{\sin i'}{\sin c} = \frac{C_1C_2}{r_2 + 1}$$

Therefore

$$C_1C_2 = (r_2 + 1) \frac{\sin i'}{\sin c}$$

Now

$$\frac{\sin i}{\sin i'} = n$$

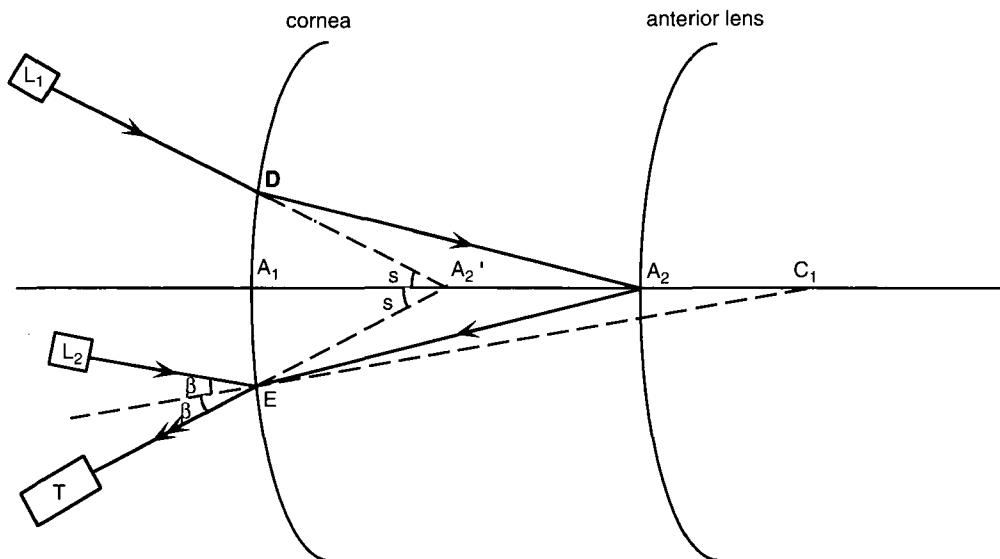


Figure 1-12

Determination of the anterior chamber depth using Tscherning's ophthalmophakometer. A bright lamp (L_1) and a dim lamp (L_2) are positioned so that their images formed by reflection at the anterior crystalline lens and cornea, respectively, are centered within the telescope (T). C_1 is the center of curvature of the cornea. A ray from L_1 , incident upon the cornea at D and directed toward A_2' , is refracted to intercept the anterior crystalline lens surface at A_2 . A ray from L_2 , incident upon the cornea at E , is reflected toward T . Angles $2s$ and 2β indicate the angles between L_1 and T and L_2 and T , respectively.

Therefore

$$C_1 C_2 = (r_2 + 1) \frac{\sin i}{n \sin c}$$

In addition,

$$C_1 C_2 = r_2 + 1 - r_1$$

Thus

$$r_2 + 1 - r_1 = (r_2 + 1) \frac{\sin i}{n \sin c}$$

which may be rearranged as

$$(r_2 + 1) = r_1 \left[\frac{n \sin c}{n \sin c - \sin i} \right]$$

where r_2 = the radius of curvature of the anterior lens surface, l = the anterior chamber depth, r_1 = the radius of curvature of the cornea, i = half the angle between the telescope and lamp L_2 , c = the sum of angle i and the angle between the telescope and optical axis, and n = the refractive index of the aqueous. Thus, if the anterior chamber depth has already been determined, the radius of curvature of the anterior crystalline lens surface may be calculated.

Comparison Method. This method compares the size of the image formed by reflection at the anterior cornea with that from an alternative refractive surface (e.g., the anterior lens surface).¹⁰⁷ Fletcher¹⁵⁸ described this technique as the ophthalmophakometry method of choice, and the clinical procedure has been detailed by Van Veen and Goss.¹⁵⁹ The principles behind this technique were fully described by Bennett.⁴² Briefly, for a relatively distant object, the height of an image formed by reflection at a spherical surface is approximately proportional to the radius of curvature of the reflecting surface. Accordingly, the ratio of the height of the images formed by reflection at each of the eye's four reflecting surfaces will be proportional to the ratio of their radii of curvature (Figure 1-14). However, in the case of the images from the crystalline lens surfaces, these result from both reflection at the lens surface and refraction at the cornea. Bennett noted that the optics of this combined reflection/refraction system are most simply approached if the rays are considered to emerge from an "equivalent mirror," as illustrated in Figure 1-15 and indicated below.

Consider Figure 1-15. The reflecting surface (A_2) and refracting (A₁) surfaces are optically equivalent to a single reflecting surface (A₂') having a radius of curvature of r_2' . If the height of the image formed by reflection at this equivalent mirror is h_3' and the height

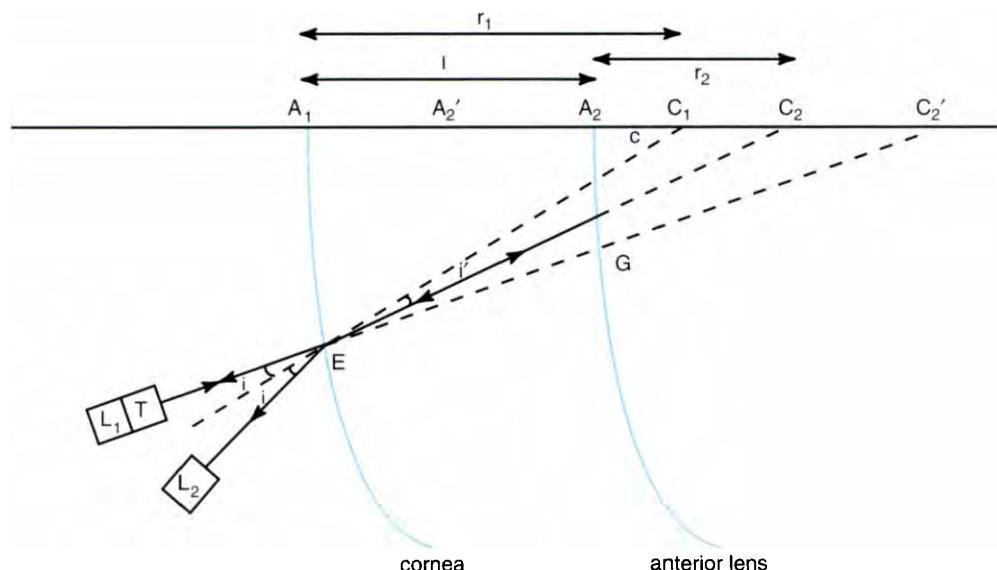
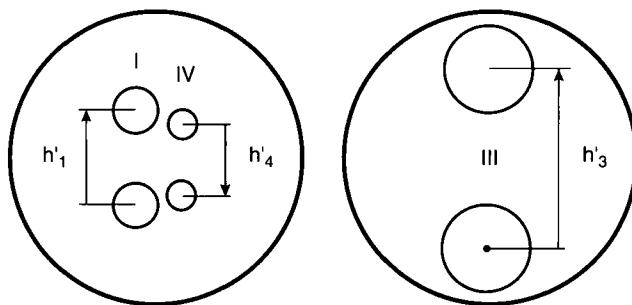


Figure 1-13

Determination of the anterior crystalline lens radius of curvature using Tscherning's ophthalmophakometer. A bright lamp (L_1) is set immediately above the telescope (T) to one side of the fixation target, and a second, dim lamp (L_2) is positioned so that its image formed by reflection at the cornea is centered within the telescope. C_1 and C_2 represent the centers of curvature of the cornea and anterior crystalline lens, respectively. A ray from L_1 , incident upon the cornea at E and directed toward G , is refracted toward C_2 . Angles i and i' represent the angles of incidence and refraction at the cornea, respectively. r_1 , Radius of curvature of the cornea; r_2 , radius of curvature of the anterior lens surface; l , anterior chamber depth.

**Figure 1-14**

Comparison ophthalmophakometry. Two vertically displaced light sources are arranged to provide twin Purkinje images I, III, and IV by reflection at the cornea, anterior crystalline lens, and posterior crystalline lens, respectively. For example, the ratio h'_3/h'_1 is equal to the ratio of the respective radii of curvature, that is, r'_3/r_1 , where r'_3 is the radius of curvature of the equivalent mirror (see Figure 1-15) and r_1 is the radius of curvature of the anterior cornea. Thus if r_1 is determined by keratometry, r'_3 can be calculated. (From Bennett AG, Rabbets RB. 1989. Clinical Visual Optics, 2nd ed, p 477. London: Butterworth-Heinemann.)

of the image formed by reflection at the cornea is h_1 , then

$$\frac{r'_2}{r_1} = \frac{h'_3}{h_1}$$

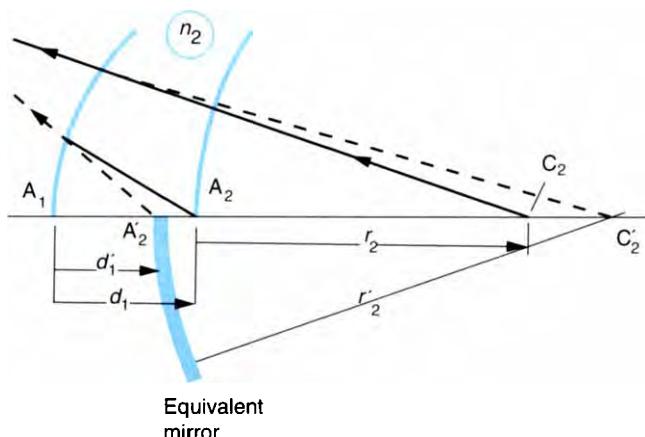
Once r_1 is determined by keratometry, r'_2 , the radius of curvature of the equivalent mirror, may be found. Now, $d_1 + r_2 = A_1 C_2$ and $d'_1 + r'_2 = A_1 C'_2$. Also C_2 and C'_2 are conjugate points following refraction at the cornea. Therefore

$$\frac{n_2}{d_1 + r_2} - \frac{n_1}{d'_1 + r'_2} = F_1$$

where n_1 and n_2 are the refractive indices of the aqueous and air, respectively.

If the anterior chamber depth is determined using an alternative technique (e.g., ultrasound or Tscherning's ophthalmophakometry), then the radius of curvature of the anterior crystalline lens (r_2) may be calculated.¹¹⁰

Mutti et al.¹⁶⁰ described a video ophthalmophakometry procedure. The use of a video camera rather than conventional still photography is valuable in the assessment of children, whose unsteady fixation and limited attention span can produce methodological difficulties. In addition, they suggested that the ocular component curvatures could be determined directly from the ophthalmophakometer readings by calibrating the instrument using steel balls of known radius, rather than obtaining readings by comparison with the keratometry

**Figure 1-15**

Comparison ophthalmophakometry. The anterior crystalline lens reflecting surface (A_2) and the refracting surface, or cornea (A_1), are optically equivalent to a single reflecting surface (A'_2) of distance d'_1 from the cornea having a radius of curvature of r'_2 . C_2 and C'_2 are the centers of curvature of the anterior crystalline lens and the equivalent single refracting surface, respectively. d_1 , Anterior chamber depth; n_2 , refractive index of the aqueous humor. (From Bennett AG, Rabbets RB. 1989. Clinical Visual Optics, 2nd ed, p 478. London: Butterworth-Heinemann.)

findings. This direct measurement procedure showed slightly improved repeatability when compared with the keratometry comparison technique.

X-ray Methods

The use of x-rays to measure the axial length of the eye was first described by Rushton,¹⁴⁷ and this technique was adopted in the extensive studies performed by Stenstrom.²² X-rays have also been used to determine the refractive power of the eye.¹⁶¹ If two narrow beams are directed into the eye, the subject will perceive two luminous vertical lines whose separation indicates the size of the retinal image. Because the size of the object and the distance between the object and the image are known, the total refracting power of the eye may be calculated. For safety reasons, x-ray methods are no longer generally used.

Ultrasound

Ultrasound waves are sound waves having a frequency above 20 KHz, or beyond the audible range of the human ear.^{162,163} When such high-frequency waves are incident upon a boundary between two media of differing acoustic density, a proportion of the energy is reflected while the rest is transmitted. Thus, their behavior is somewhat analogous to that of light waves; however, they are completely reflected at a tissue/gas

interface and are unhindered by opacification.¹⁶⁴ Accordingly, as an ultrasonic pulse is directed through the eye, reflected echoes are generated at each change in media density. The separation between each boundary can be determined from the time taken for the echoes to return to the source, providing that the velocity of the sound waves within the various media is known.¹⁶⁵

Ultrasound has been used to examine the anatomical structures of the eye *in vivo*, and to determine the biometric dimensions of the eye. Previous studies have indicated that this technique provides measurements to an accuracy of ± 0.1 mm.¹⁶⁶⁻¹⁶⁸ For a full review of the principles of ultrasonic examination, see Coleman et al.,¹⁶² Guthoff,¹⁶⁹ and Byrne and Green.¹⁷⁰ The history of the ophthalmological applications of ultrasound was reviewed by Thijssen.¹⁷¹ In addition to providing biometric information, amplitude-mode (a-mode) ultrasound has also been used for assessment of the ocular changes that take place during accommodation.^{172,173}

With regard to determining the biometric dimensions of the globe, a-mode ultrasound has been used widely for the measurement of corneal thickness, anterior chamber depth, crystalline lens thickness, and vitreous chamber depth. In a comprehensive review of this topic, Coleman et al.¹⁶² noted that there are four principal areas of concern in making accurate ultrasonic measurements of the eye: (1) transducer alignment, (2) distortion-free measurement, (3) frequency and width of the beam, and (4) measurement system standardization.

Transducer Alignment

It is critical that the transducer beam be aligned along the desired axis of measurement during depth determination. Jansson¹⁷⁴ demonstrated that a misalignment of 5 degrees produces an error in axial length measurement of 0.1 mm. Accordingly, the most commonly adopted procedure has been the inclusion of a fixation light within the transducer probe itself.^{175,176} Byrne and Green¹⁷⁰ noted that patients with lenticular opacities may have difficulty in fixating the probe's light. An alternative technique was described by Coleman and Carlin¹⁷⁷ whereby the patient viewed an optotype via a front surface mirror while the transducer was directed toward the eye through a hole in the mirror. This technique also allowed objective assessment of axis alignment by allowing the practitioner to view the macula through the optical system using a direct ophthalmoscope.

Steele et al.¹⁷⁸ compared a-mode ultrasonic measurements obtained using a number of fixation targets and observed greater variability of anterior chamber depth and lens thickness findings using the fixation light contained within the probe when compared with a distant (6 m) fixation target. They suggested that the internal fixation target may have induced variations in the

accommodative response, and it would seem likely that subjects' awareness of the apparent nearness of the target may have caused proximally induced accommodation.^{88,179} Steele et al.¹⁷⁸ therefore recommended that either a distant light or an acuity letter (optotype) viewed at a distance of 6 m be used as a fixation target.

Distortion-free Measurement

Obviously, it is critical that application of the probe does not flatten or distort the globe in any way. For this reason, many workers have used a "water standoff probe," a water-filled probe covered with a soft membrane, to avoid corneal applanation.¹⁶⁴ However, small air bubbles may become trapped within the water chamber, which will produce erroneous readings if the bubbles adhere to the surface of the transducer.¹⁷⁰ Most modern, commercially available units generally use solid probes that contact the cornea directly. However, care must be taken to ensure that minimal pressure is applied. Hofmann¹⁸⁰ demonstrated that ultrasonically determined measurements of axial length decrease with increasing applanation pressure, with mean values of approximately 23.10 and 23.00 mm being reported for applanation pressures of 8 and 24 mmHg, respectively. Furthermore, several researchers have observed shorter axial dimensions when using a contact probe in comparison with the corneal immersions or standoff procedures.¹⁸¹⁻¹⁸³

Frequency and Width of the Ultrasound Beam

The frequency of the examining beam is a critical parameter in determining the accuracy of measurement. Increasing the frequency of the transducer improves resolution; however, it also results in an increase in tissue absorption. Accordingly, frequencies of 10 to 20 MHz have been found to allow accurate measurement of axial length, whereas higher frequencies may be used for assessment of the anterior segment alone.^{162,168,170} In addition, the beam should be as narrow as possible to facilitate the measurement of curved surfaces. Coleman et al.¹⁶² used the analogy of trying to measure the depth of a cup with a broad ruler to illustrate the error induced when an excessively broad beam is used.

Measurement System Standardization

To convert the time intervals between the echoes into linear distances, the velocity of the ultrasound beam within the ocular media must be known. The following values are typically used^{174,184}:

Aqueous and vitreous	1530–1532 m/sec
Cornea, retina, and choroid	1550 m/sec
Sclera	1600 m/sec
Lens	1640 m/sec

However, Giers and Epple¹⁸¹ observed increased variability when measuring the lens thickness in cataractous

lenses and suggested that this may result from changes in ultrasound velocity. Coleman et al.¹⁸⁵ indicated that a velocity of 1629 m/sec should be adopted for the crystalline lens of cataract patients to account for the reduced density when compared with normal lenses.

Calculation Method

A calculation method for determining the equivalent powers of both the eye and the crystalline lens without the need for ophthalmophakometry was described by Bennett.¹³⁹ This technique requires measurement of the refractive error and anterior corneal curvature (i.e., keratometry) and ultrasonic determination of the anterior chamber depth, lens thickness, and vitreous chamber depth. With the use of refractive indices from the Gullstrand-Emsley schematic eye, the equivalent powers could be calculated. Dunne et al.¹⁸⁶ compared findings obtained using this procedure with measurements recorded using ophthalmophakometry. They reported agreement between the calculation technique and both ocular and lens powers obtained using ultrasound and phakometry measurements.¹⁸⁷ However, Mutti et al.¹⁶⁰ noted that the 95% limit of agreement between the two techniques for the equivalent eye and lens powers were ± 0.22 and ± 0.37 D, respectively. This level of agreement was at least partially produced by the fact that the same axial length measurement was used in both procedures. In a subsequent report, Royston et al.¹⁸⁸ detailed a method for calculating the crystalline lens radii without ophthalmophakometry.

Mutti et al.¹⁶⁰ compared the repeatability of crystalline lens power measurements obtained using the calculation and ophthalmophakometry procedures. They reported that the repeatability of ophthalmophakometry using a video camera was significantly better than that of the calculation method, although there was no significant difference between ophthalmophakometry using still photography and the calculation technique. They indicated that the direct measurement procedure was preferable, because of its independence from cumulative bias induced from measurement of other ocular components. In addition, direct observation allows the component changes responsible for variations in lens power over time (e.g., changes in anterior or posterior curvature or lens thickness) to be identified.

Other Techniques for Measuring the Dimensions of the Globe

Lieb et al.¹⁸⁹ and Hitzenberger¹⁹⁰ recently proposed the use of laser Doppler interferometry to measure the axial length of the eye *in vivo*. The major advantage of this technique over conventional ultrasound is that no contact between the cornea and sensor is required, eliminating the need for a local anesthetic and possible erroneous findings resulting from corneal applanation. A

commercially available instrument, the Zeiss Meditec IOLMaster (Carl Zeiss Meditec, Dublin, CA), uses partial coherent interferometry to measure the axial length of the globe with a resolution of 0.01 mm. For a fuller description of the instrument, see Santodomingo-Rubido et al.¹⁹¹ Several previous investigations have demonstrated the validity of this instrument in comparison with conventional ultrasound biometry.^{192,193} Kielhorn et al.¹⁹⁴ observed 95% limits of repeatability for axial length measurement using this instrument of ± 0.07 mm.

Charman¹⁶⁵ reviewed the possible use of x-ray computed tomography, magnetic resonance imaging (MRI), emission computed tomography, and positron emission tomography for imaging the eye. A number of recent investigations have used MRI to determine the shape of both the eye and its internal structures *in vivo*.¹⁹⁵⁻¹⁹⁷

REFRACTIVE ERROR DETERMINATION

Subjective Optometers

The history of subjective refraction has been comprehensively reviewed by Bennett and Lang.^{76,101,198} The earliest optometer appears to have been Scheiner's multiple pinhole shown in Figure 1-16.¹⁰¹ A distant object (typically, a small spot of light) is viewed through a double pinhole. An emmetropic patient will see a single spot, whereas an ametropic individual will see two spots. The

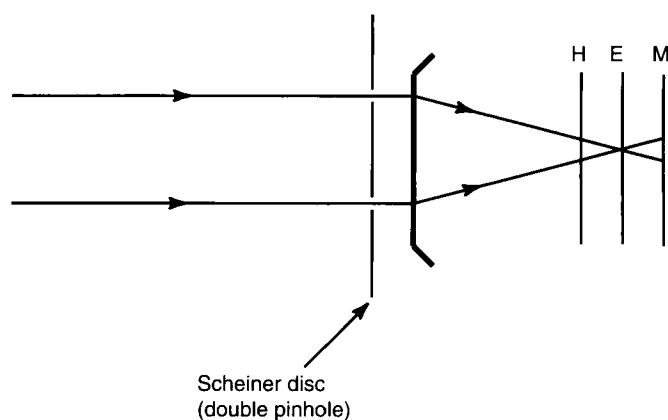


Figure 1-16

The Scheiner disc, or multiple pinhole. H, E, and M represent the relative locations of hyperopic, emmetropic, and myopic retinas, respectively. When viewing a distant point object of regard, an emmetropic observer will report seeing a single image, whereas an ametropic individual will report two images. If the top hole is subsequently occluded, a myope will report that the top image disappears (because of retinal inversion), whereas a hyperope will indicate that the bottom image disappears.

type of ametropia can be determined by covering one of the pinholes and asking the patient which spot disappeared. If the top hole is covered, the hyperope will report that the bottom image disappeared (because of retinal inversion), whereas a myope will report that the top image disappeared. This can be easily remembered by an aide-mémoire for this test (and maybe throughout life!)—*myopes never lie*. Scheiner¹⁰¹ also described the use of a triple pinhole in the equilateral triangular formation. With this arrangement, the form of ametropia could be determined on the basis of whether the patient reports an erect or inverted triangle. The Scheiner principle can also be used in the design of automated objective refractors (see Chapter 18).

The Simple Optometer

The refractive state of the eye may also be determined by assessing the physical location of the far point. This is relatively easy in treating myopic patients, entailing moving a fine target slowly away from the patient. The farthest distance at which it can be seen clearly represents the far point. Indeed, this technique was described in 1623 by Benito Daza de Valdés,¹⁰¹ who drew out a handful of mustard seeds into a row and instructed the subject to

count the seeds until they became indistinct. The distance from the bridge of the nose to the farthest seed counted provided a measure of the required lens power in "grados," with 1 grados being approximately 1.16 D.

This procedure will not work for hyperopic patients, for whom the far point is located behind the eye. However, if a high-powered convex lens is introduced before the eye, the point conjugate with the retina will now be located in front of the eye for the majority of patients (excluding very high hyperopes). This is the principle behind the simple optometer (Figure 1-17). Patients view a near target through a convex lens. For a clear image without accommodating, they will position the object so that the image formed by the convex lens lies at their far point. If the object's distance from the lens is measured, the image position, and hence the far-point location, can easily be determined.

Two significant problems with the simple optometer are that the scale is nonlinear (i.e., the distance that the object must be displaced for a unit dioptric change is greatest for high hyperopia and least for high myopia) and the apparent size of the test object increases as the target approaches the eye. The latter observation, combined with subjects' knowledge of the nearness of the

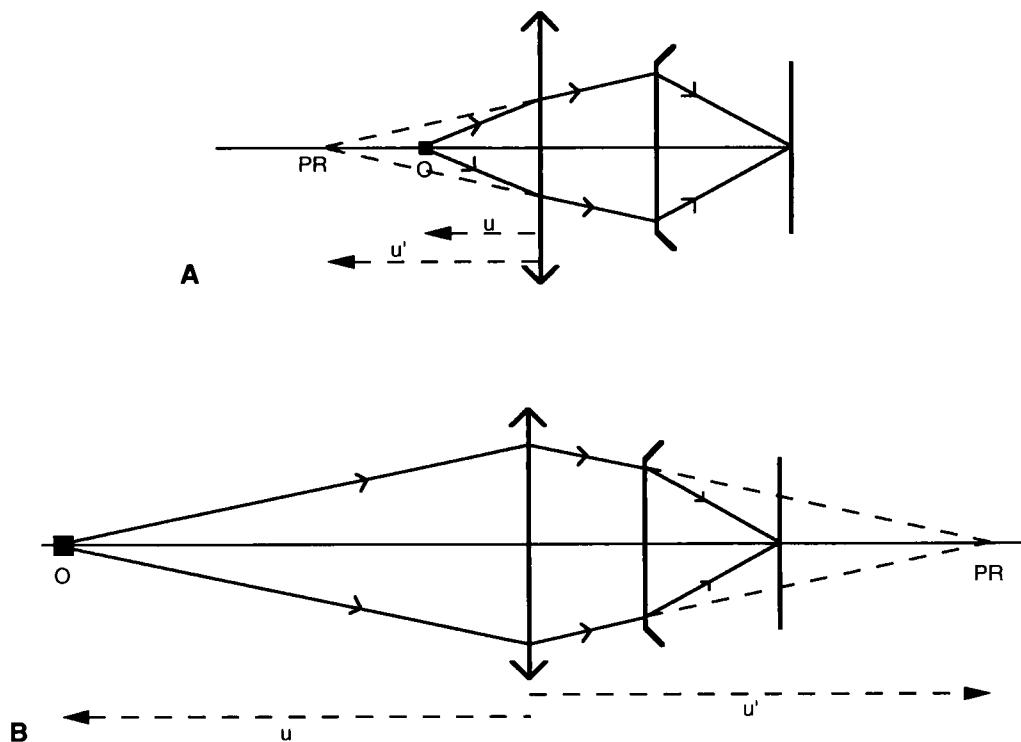


Figure 1-17

The simple optometer. The object of regard (*O*) is positioned so that the image formed by refraction at the convex optometer lens is located at the observer's far point, or punctum remotum (*PR*). *A* illustrates a myopic observer, and *B* shows a hyperopic observer. Because both the optometer lens power and the distance from *O* to the optometer lens (*u*) are known, the location of the image (*u'*), which corresponds with the observer's far point, may readily be calculated from the equation $1/u' - 1/u = F$.

target, is likely to provide a powerful stimulus for proximally induced accommodation.

Young's Optometer

Young's optometer was essentially a simple optometer combined with a Scheiner disc.¹⁰⁷ Rather than using a double pinhole, this optometer was based on the design of Porterfield comprising two vertical slits.⁷⁶ However, the principle behind the Young optometer is identical to that of the Scheiner disc. In Young's original instrument, the test object was a line engraved along the axis of the instrument, extending from the midline between the slits away from the eye in an anteroposterior direction. The line is viewed through each aperture from a different position. Hence, two lines are perceived though only one line actually exists. The two lines appear as an elongated X, with the point of intersection of the X corresponding to the point conjugate with the retina. A small cursor is moved to locate the position where the two apparent lines cross.¹⁰⁷

Badal Optometer

To overcome some of the difficulties with the simple optometer, namely the nonlinear scale and increasing

angular subtense of the image as the target approached the observer, Badal¹⁷ designed an optometer in which the second principal focus of the optometer lens coincided with either the nodal point or the first principal focus of the patient's eye.¹⁹⁹ This is illustrated in Figure 1-18, in which it may be seen that the angular subtense of the image remains constant irrespective of the target location. Furthermore, the relationship between the distance from the target to the optometer lens and the observer's refractive error is now linear.

In practice, the practitioner is not aware of the refractive power of the eye, and hence the exact location of either the first principal focus or the nodal point of the eye is unknown. Accordingly, it is not possible to place the Badal optometer lens in these precise locations. However, if the optometer lens is positioned so that its second principal focus coincides with either the midpoint of the entrance pupil or spectacle plane, then the magnitude of the resulting error in calculating the degree of refractive error will be small in the majority of cases. The Badal optometer is a key optical component of most automated objective refractors (see Chapter 18).

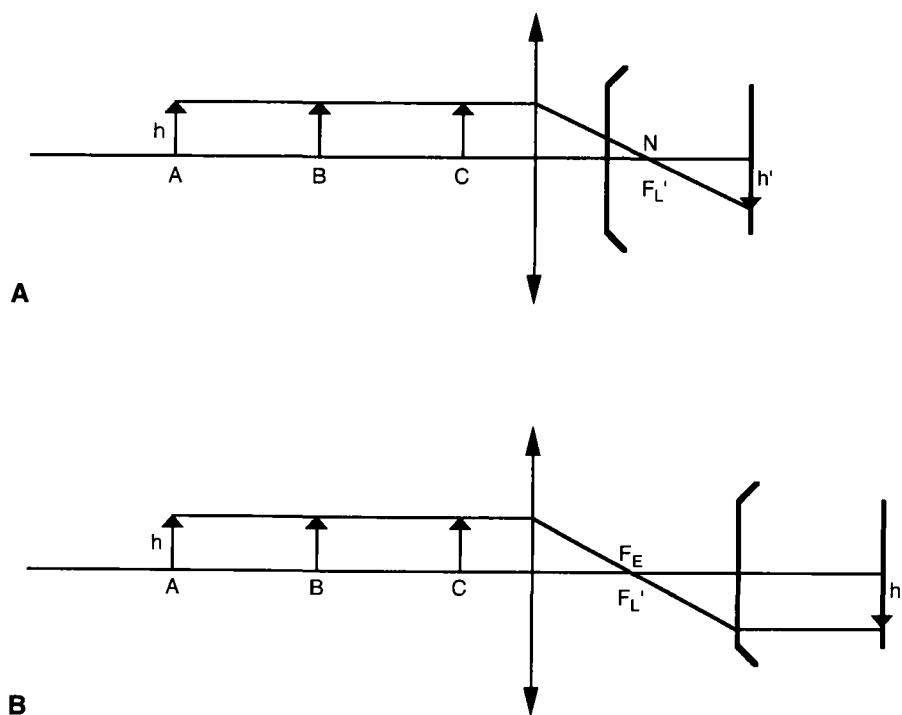


Figure 1-18

Badal optometer. The second principal focus of the optometer lens (F_L') coincides with either the nodal point of the eye (N) (A) or the first principal focus of the eye (F_E) (B). Thus, irrespective of its location (A, B, or C), an object of regard of height (h) will always produce a retinal image of height (h'). In addition, the relationship between the target distance and the observer's refractive error is now linear, thereby overcoming two of the major problems of the simple optometer. (Adapted from Southall JPC. 1993. Mirrors, Prisms and Lenses, 3rd ed, p 422. New York: Macmillan.)

Telescope Optometers

Both Galilean and astronomical (terrestrial) telescopes may be used to determine the refractive state of the eye.¹⁹⁸ The separation of the eyepiece and objective lenses varies with the observer's refractive error, being shorter for a myopic eye and longer for a hyperopic eye (Figure 1-19).

Chromatic Optometers

Chromatic optometers such as the cobalt disc and the more conventional duochrome (bichrome) test use the chromatic aberration of the eye to determine the presence of spherical ametropia.²⁰⁰ For an unaccommodating emmetropic eye, light having a wavelength around

570 nm is focused accurately on the retina, whereas wavelengths shorter and longer than 570 nm are focused in front of and behind the retina, respectively (Figure 1-20).¹⁹⁹ One standard on duochrome filters recommends a green filter having a peak luminosity of approximately 535 nm and a red filter of approximately 620 nm.²⁰¹ These wavelengths provide red and green foci that are located approximately 0.25 D on either side of the retina.²⁰² Thus an emmetropic subject viewing the duochrome chart should observe objects on the red and green backgrounds equally clearly. However, uncorrected myopic observers will indicate increased contrast and clarity for the targets on the red background, and uncorrected hyperopes may indicate a preference for the

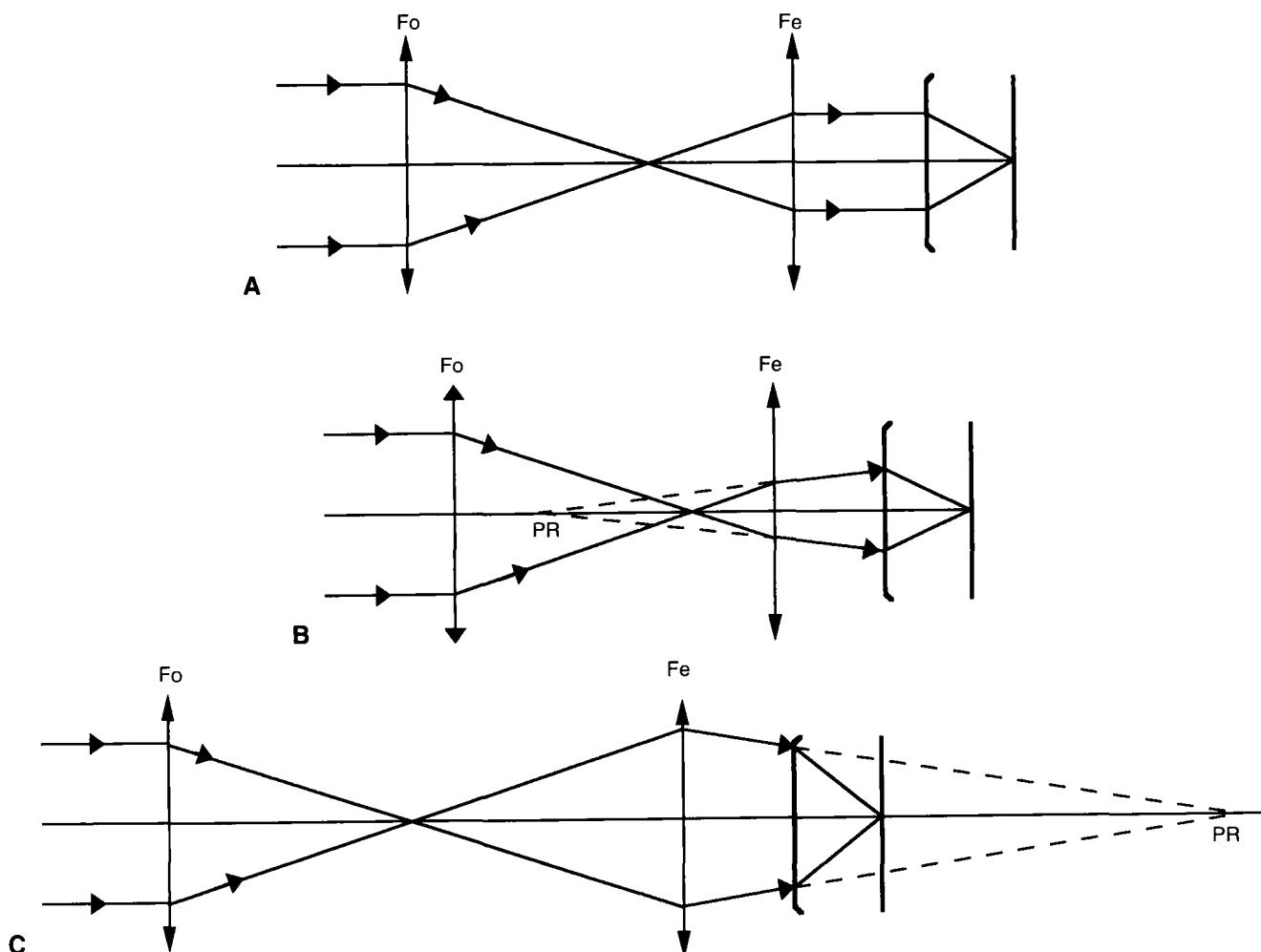


Figure 1-19

Both Galilean and astronomical (terrestrial) telescopes may be used to determine the refractive state of the eye. This figure shows an astronomical telescope. A illustrates the setting for an emmetropic observer with parallel light emerging from the eyepiece (Fe). B and C show the lens positions for a myopic observer and hyperopic observer, respectively, with divergent or convergent light emerging from the eyepiece. Thus a myopic observer will move the objective (Fo) and eyepiece lenses closer together for a clear image of a distant object without accommodating, while a hyperopic observer will move the lenses farther apart. PR, Punctum remotum.

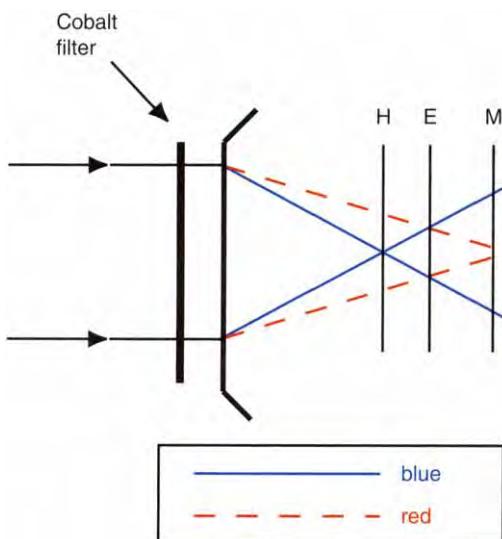


Figure 1-20

Principle of chromatic optometers. Shorter wavelengths (e.g., blue light) are refracted more than longer wavelengths (e.g., red light). Accordingly, when a distant object is viewed, blue (or green) rays of light will be focused in front of the emmetropic retina, whereas red rays will be focused behind it. *H*, *E*, and *M* represent the relative locations of hyperopic, emmetropic, and myopic retinas, respectively. Accordingly, when viewing a duochrome (or bichrome) target, a hyperopic observer will indicate that the target on the green background appears clearer or darker, whereas a myopic observer will report that the target on the red background is clearer or darker.

targets on the green background. Chromatic optometry can be used as a key component of the subjective refraction (see Chapter 20). The principle can be used for the determination of the monocular and binocular spherical endpoints and for equalization of the refraction between the two eyes.

Objective Optometers

Despite the development of highly sophisticated computerized autorefractors, the most commonly used objective optometer in the clinical setting remains the retinoscope. Retinoscopy was developed from ophthalmoscopy, and in 1859, Sir William Bowman described how the shadow movements could be used to detect keratoconus and corneal astigmatism.¹⁰¹ In 1873, Ferdinand Cuignet¹⁷ described more fully this objective procedure for determining the refractive state of the eye. Chapter 18 reveals the theory and practical manner in which retinoscopy is used by the clinician.

The direct ophthalmoscope has also been used for assessment of the refractive error, although it is reported to be somewhat inaccurate.¹⁷ Richman and Garzia²⁰³

compared the ophthalmoscopically determined refractive error assessed under cycloplegia with noncycloplegic subjective refraction and reported that 82% of the findings differed by less than 1 D.⁸ An optometer based on the indirect ophthalmoscope was described by Schmidt-Rimpler in 1877.²⁰⁴ The patient's refractive state could be derived from the separation between the condensing lens and the patient's eye. Electrophysiological techniques have also been used for estimation of the refractive status of the eye.^{205–207} For example, Regan used visually evoked potentials, in conjunction with a stenopalic slit, for objective determination of the refractive error.²⁰⁸

More recently, a number of automated refracting instruments have been described.^{209–212} These instruments are faster than retinoscopy, may be used by support personnel, and can be used with some patients (e.g., keratoconics) for whom retinoscopy is difficult. However, they are also considerably more expensive than a retinoscope, fail to work with relatively small pupils, and often fail to control accommodation adequately. In particular, the use of internal fixation targets provides strong cues for proximally induced accommodation. Discussion of the practical optics of autorefractors and the clinical use of automated objective refraction appear in Chapter 18.

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2

Incidence and Distribution of Refractive Anomalies

Karla Zadnik, Donald O. Mutti

The eye's refractive error has been studied for decades. Many of these investigations have focused on associations between the distribution of refractive error and a wide variety of factors. These factors include—but are not limited to—age, gender, ethnicity, geographical location, diet, intelligence, socioeconomic status, performance of near work, and genetic factors. Many of these associations are statistically strong and have led to intellectually compelling theories on the etiology of myopia as groups of people with markedly different distributions of refractive error are compared.

This chapter first presents the classical notion of emmetropization, which creates the common distribution of refractive errors seen in the United States today. The focus then turns to the areas of the distribution where clinically significant refractive errors occur and how the aforementioned factors influence the distribution. Finally, the classical factors associated with—and, intriguingly, perhaps leading to—the onset of clinically important refractive errors are discussed.

CLASSICAL NOTIONS OF REFRACTIVE ERROR DISTRIBUTIONS

A fascinating process occurs in ocular development between birth and puberty to produce a leptokurtic distribution that overwhelmingly favors emmetropia and is skewed toward myopia, with more moderate to high myopes than moderate to high hyperopes. As seen in Figure 2-1, the distribution of refractive errors at birth closely resembles a normal distribution, with some skew toward hyperopia. Reports of myopia during cycloplegic retinoscopic examination of newborns are variable as to prevalence, with estimates ranging from 0% to 25% (Table 2-1). Between infancy and childhood (as detailed in Chapter 3), the eye grows in such a way that the distribution of refractive errors shifts toward emmetropia, narrows considerably (with most children being emmetropic to slightly hyperopic), and shows a shift in skew toward myopia. This process—whereby the

average refractive error shifts toward emmetropia and the entire distribution of refractive errors decreases its variability—is termed *emmetropization*. Recent cross-sectional and longitudinal studies agree that the vast majority of emmetropization is completed rapidly in infancy during the first year of life.^{1,2}

When does emmetropization stop? Between the ages of 5 and 15 years, ocular component development slows. During this decade, anterior chamber depth increases by only 0.10 to 0.20 mm and vitreous chamber depth and axial length by about 1.0 mm.³⁻⁷ Lens thinning seems to continue its earlier trend by continuing to thin another 0.15 to 0.20 mm. Many textbooks describe the lens as a unique part of the body in that it grows throughout life, continually laying new fibers onto the lens cortex.⁸ Studies reviewed by Larsen⁹ show that the lens weighs about 65 mg at birth and doubles its weight during the first year of life, growing very slowly after age 1 year. A redoubling of crystalline lens weight to 258 mg does not occur until age 80 years. Although it continues to grow in the sense of laying down new fibers, it does not continually grow in the sense of thickening. The cornea is remarkably stable throughout childhood, on average. Lens power decreases about 2.00 D. The average hyperopia decreases about 1.00 D. The interesting feature of ocular development is that, during this time of relatively slow average growth compared with earlier in life, the prevalence of myopia increases by over 7 times to 15%.

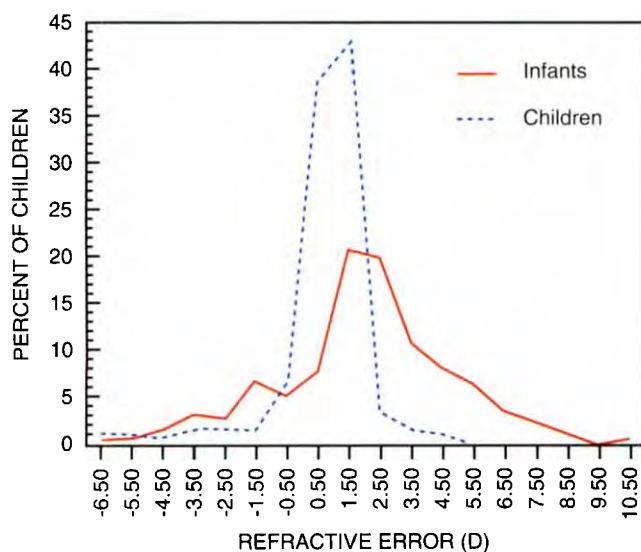
The prevalence of myopia remains low, under 2%, until about the age of 7 or 8 years, when there is a sudden rise that begins to level off only in the early teens (Figure 2-2). Myopia that has its onset during these years can be termed *juvenile-onset myopia*. This myopia typically progresses after its onset, with an average rate of increase of about $-0.50 (\pm 0.25)$ D per year. The eye continues to grow throughout the teen years, suggesting that myopia progresses in these children because the ability of the crystalline lens to compensate for increases in axial length is reduced. All millimeters of axial length increase translate directly

TABLE 2-1 Refractive Error in Infancy and Toddlerhood

Author	N	Age	Method	Mean Refraction (D)	Myopia
Goldschmidt ²⁵	356 infants	2–10 days	Atropine 0.5%	+0.62 (± 2.24)*	24.2%
Santonastaso ²⁸	34 infants	0–3 mo	Atropine retinoscopy	+1.67 (± 2.54)	8.0%
Luyckx ²⁶	104 eyes	0–1 wk	Cyclopentolate 1%	+2.4 (± 1.2)	0.0%
Cook & Glasscock ²⁴	1000 eyes	After post-delivery care	Atropine ointment 1% 4x	+1.54†	25.1%
Mohindra & Held ²⁷	48 infants	0–4 wk	Near retinoscopy	-0.70 (± 3.20)	Not given
Zonis & Miller ¹⁷⁹	600 eyes	48–72 hr	Mydriaticum	+1.10 (± 1.60)	14.5%
Mayer et al. ¹	32 infants	1 mo	Cyclopentolate 1%	+2.20 (± 1.60)	3%
	42 infants	12 mo		+1.57 (± 0.78)	
Mutti et al. ²	262 infants	3 mo	Cyclopentolate 1%	+2.13 (± 1.31)	Not given
	243 infants	9 mo		+1.32 (± 1.07)	

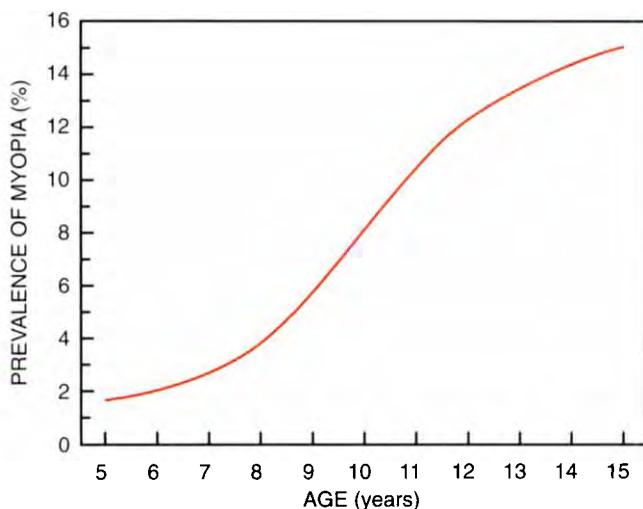
*Standard deviations are in parentheses.

†See Figure 2-1 for distribution.

**Figure 2-1**

Comparison of refractive error distribution among newborns²⁴ with that among children.¹⁷⁸ The distribution of refractive errors narrows and its peak becomes closer to emmetropia between infancy and childhood as the process of emmetropization takes place. (Reprinted from Zadnik K. 1997. The Ocular Examination, p 55. Philadelphia: WB Saunders.)

into diopters of myopia. Sorsby et al.⁶ found that the average values of components of children 13 to 14 years of age were not different from those of young adult male recruits 19 to 22 years old. They concluded that the eye does not grow appreciably beyond the age of 13 to 14 years. The age of cessation of the progression of myopia is 14.6 to 15.3 years for girls and 15.0 to 16.7 years for

**Figure 2-2**

Increases in the prevalence of myopia (at least -0.50 D by noncycloplegic retinoscopy) as a function of age. Data are from Blum et al.³⁹ (Reprinted from Zadnik K. 1997. The Ocular Examination, p 58. Philadelphia: WB Saunders.)

boys, depending on the method of estimation of progression,¹⁰ and it is consistent with the cessation of ocular growth. This age is obviously an average because many patients' myopia progresses well into adulthood.¹¹ In addition, many patients, perhaps another 10% of the population, will become myopic for the first time after the teen years.¹¹ As noted in Chapter 1, this type of myopia is termed *adult-onset myopia*. These two forms of myopia bring the total prevalence of myopia in the adult population to about 25%.¹² The development of

ametropia and its progression throughout the human lifespan are detailed in Chapter 3.

Sorsby et al.¹³ attempted to categorize myopia on the basis of values of and associations between the ocular components. They examined the range of axial lengths in emmetropes, finding that eyes anywhere from 21.0 to 26.0 mm in length could be emmetropic. Emmetropia was not the product of having the correct axial length but rather of having the right match between axial length and primarily—according to Sorsby et al.—corneal power. For ametropias up to ± 4.00 D, the cause was not an incorrect axial length, but rather a mismatch with corneal power they called *correlation ametropia*. Errors greater than ± 4.00 D were called *component ametropia*, being primarily due to excessive axial length; the corneas of these patients fell within a range similar to that of emmetropes. A third classification, for myopia typically greater than -6.00 D and accompanied by degenerative fundus changes, was termed *pathological myopia*.

FACTORS THAT AFFECT REFRACTIVE ERROR DISTRIBUTIONS

One of the difficulties in characterizing refractive error distributions across many associated factors is the effect of the criterion used to define the various refractive error distributions. In Table 2-2, the Working Group on Myopia Prevalence and Progression¹¹ demonstrated the marked effect of a criterion change on the prevalence of myopia in a school-age, population-based sample.¹⁴ The prevalence of myopia changes by an order of magnitude if the operational definition of myopia is altered from "any minus refraction" to a clinically important " -1.00 D or more myopia." Other important factors are the variety of methods used to measure refractive error

across studies, as shown in Tables 2-1 and 2-3 through 2-6, and the wide range of reproducibility obtainable with these subjective and objective refraction measurement techniques.¹⁵

Age

Age is the single most important determinant of the distribution of refractive error in a given group (see Chapter 3). The onset and development of myopia occur in well-established yet poorly understood patterns. Only a very small proportion of infants are myopic at birth, and much of this neonatal myopia is associated with prematurity.¹⁶ Likewise, babies and toddlers exhibit a low prevalence of myopia.¹⁷ Even by the time they enter formal schooling at age 6 years, children are generally not myopic. During the ensuing 6 to 8 years, however, low to moderate myopia is first observed and progresses.^{18,19} For juvenile-onset myopia, onset is typically between the ages of 7 and 14 years,¹¹ the rate of progression is -0.40 (± 0.25) D on average,²⁰ and the age of cessation is 14 to 15 years for females and 15 to 16 years for males.¹⁰ The prevalence of myopia in older age groups increases^{18,22-23} to as high as 25% of the U.S. adult population.¹²

Table 2-1 lists major studies of the prevalence and distribution of refractive error in newborns.²⁴⁻²⁸ Overall, it can be seen that the distribution of infant refractive error is centered somewhere in low to moderate hyperopia with a moderate spread (standard deviations on the order of 1.00 to 2.00 D) and that, not surprisingly, noncycloplegic measures yield distributions with more myopic average values than appear in distributions yielded by cycloplegic measures.²⁷

Astigmatism in infancy and toddlerhood has been well documented, but its purpose in visual development is still unknown. Table 2-3 presents the results from several large-scale studies of infants' and preschoolers' astigmatism with a variety of measurement techniques and a large range of ages.²⁸⁻³⁵ Overall, astigmatism presents in infancy (<1 year of age) with anywhere from one-quarter to one-half of infants showing significant astigmatism (>1.00 DC); the story on the orientation of that astigmatism and its changes with age is less clear-cut. Several recent studies suggest that early infant astigmatism may well be a mix of both with-the-rule and against-the-rule.^{1,2,36} With time, the prevalence of astigmatism decreases toward that seen in school-aged children. There is a general against-the-rule shift with time making early with-the-rule astigmatism resolve more often and early against-the-rule astigmatism the more persistent orientation.^{1,2,36} With respect to the origin of infant astigmatism, one group has reported that infant astigmatism up to age 1 year is primarily corneal in origin,³⁷ but another has reported that the astigmatism prevalent in infancy disappears by age 18 months, consistent with the time course

TABLE 2-2 Effect of Myopia Criterion Definition on Prevalence of Myopia in School-Age Children

Age (yr)	MYOPIA	
	-0.12 D or More	-1.00 D or More
<5-6	6.8%	0.6%
7-8	10.4%	1.0%
9-10	16.7%	2.0%
11-12	21.2%	4.5%
13-14	24.0%	5.4%

Reprinted with permission from Myopia: Prevalence and Progression. Copyright 1989 by the National Academy of Sciences. Courtesy of the National Academy Press, Washington, D.C.

TABLE 2-3 Astigmatism in Infancy and Toddlerhood

Author	N	Cycloplegic Agent/Method	Age	Prevalence of Astigmatism	Orientation
Ingram & Barr ³⁴	296 eyes	Atropine/retinoscopy	1 yr 3.5 yr	29.7% (>1.00 DC) 7.8% (>1.00 DC)	Not given Not given
Fulton et al. ³⁰	145 children	Cyclopentolate 1%/ retinoscopy	40–50 wk 1–2 yr 2–3 yr	23.5% (≥ 21.00 DC) 16% (≥ 1.00 DC) 14% (≥ 1.00 DC)	71% ATR (for children 0–3 yr 21% WTR 8% Oblique
Dobson et al. ²⁹	46 infants 187 infants	Cyclopentolate 1%/ retinoscopy	<6 mo 1 yr (midpoint)	17% (≥ 1.00 DC) 19% (≥ 1.00 DC)	100% ATR 70% ATR 18% ATR 2% Oblique
Howland et al. ³³	93 infants	No cycloplegia/ photorefraction	0–12 mo	86% ≥ 1.00 DC	70% "Horizontal and vertical"
Gwiazda et al. ³¹	521 infants	No cycloplegia/ near retinoscopy	0–12 mo	53% ≥ 1.00 DC	41% ATR 41% WTR 18% Oblique
Howland & Sayles ³²	117 infants	No cycloplegia/ photorefraction	0–12 mo	63%	55% ATR 3% WTR 42% Oblique
Mohindra et al. ³⁵	276 infants	No cycloplegia/near retinoscopy	0–12 mo	45%	40% ATR 40% WTR 20% Oblique
Santonastaso ²⁸	34 infants under 3 mo of age	Atropine/ retinoscopy	0–12 mo	52.4% ≥ 1.00 DC	15% ATR 85% WTR
Ehrlich et al. ³⁶	254 infants	Cyclopentolate 1%/ retinoscopy	9 mo 20 mo	35% ≥ 1.00 DC 13% ≥ 1.00 DC	17% ATR 81% WTR 36% ATR 58% WTR
Mayer et al. ¹	43 infants 33 infants	Cyclopentolate 1%/ retinoscopy	4 mo 48 mo	49% ≥ 1.00 DC 12% ≥ 1.00 DC	56% ATR 29% WTR across age groups
Mutti et al. ²	262 infants	Cyclopentolate 1%/ retinoscopy	3 mo 36 mo	42% ≥ 1.00 DC 4% ≥ 1.00 DC	6% ATR 89% WTR 78% ATR 22% WTR

ATR, Against the rule; WTR, with the rule.

TABLE 2-4 Mean Refractive Error in Childhood

Author	N	Cycloplegic Agent/Method	Age	Mean Refraction (D)
Ingram & Barr ³⁴	296 eyes	Atropine/retinoscopy	1 yr	+0.62 (± 1.11) ^a
			3.5 yr	+0.95 (± 1.11)
Mohindra & Held ²⁷	39 children	No cycloplegia/near retinoscopy	65–128 wk 129–256 wk	0.43 (± 1.32) 0.59 (± 0.85)
Blum et al. ³⁹	1163 children	No cycloplegia/retinoscopy	5–15 yr	Slow decline from mean of +0.62 at age 5 to +0.12 at age 15
Hirsch ¹⁴	9552 children	No cycloplegia/retinoscopy	5–14 yr	Slow decline from mean of +0.80 at age 5 to +0.35 at age 14
Zadnik et al. ⁷	133 children 143 children 129 children	Tropicamide/ autorefraction	6 yr 8 yr 11 yr	+0.73 (± 0.87) +0.37 (± 0.89) +0.30 (± 1.34)

*Standard deviations are in parentheses.

TABLE 2-5 Prevalence of Refractive Errors in White Children

Author	N	Cycloplegic Agent/Method	Age (yr)	Prevalence of Hyperopia	Prevalence of Myopia
Laatikainen & Erkkilä ²¹	162 children	Cyclopentolate 1%/ retinoscopy	7–8	19.1%	1.9%
	218 children		9–10	6.9%	6.4%
	222 children		11–12	11.7%	7.2%
	220 children		14–15	3.6%	21.8%
Sperduto et al. ¹²	NA	No cycloplegia/health survey review	12–17	Not measured	23.9%
Blum et al. ³⁹	1163 children	No cycloplegia/ retinoscopy	5	6% for all ages	2%
			6		2.25%
			7		2.5%
			8		4%
			9		5.5%
			10		8%
			11		10.5%
			12		12.25%
			13		13.25%
			14		14.5%
			15		15%
Hirsch ¹⁹	605 children	No cycloplegia/ retinoscopy	13–14	11.4%	15.2%
Kempf et al. ⁴⁰	333 children	Homatropine/ retinoscopy	6–8	35.4%	1.2%
	495 children		9–11	25.2%	3.4%
	1001 children		≥12	15.2%	4.8%

Laatikainen and Erkkilä defined hyperopia as $\geq +2.00$ D and myopia as ≤ -0.50 D; Sperduto et al. defined myopia as ≤ -0.00 D; Blum et al. defined hyperopia as $\geq +1.50$ D and myopia as ≤ -0.50 D; Hirsch defined hyperopia as $>+1.00$ D and myopia as <-0.50 D; Kempf et al. defined hyperopia as $>+1.00$ D and myopia as ≤ -0.75 D.

TABLE 2-6 Astigmatism in Childhood

Author	N	Cycloplegic Agent/Method	Age (yr)	Astigmatism	ORIENTATION
Fabian ⁴¹	1200 children	Cyclopentolate/ retinoscopy	2	≥ 1.00 DC = 1.6% ≥ 1.00 DC = 0.7%	WTR ATR
Hirsch ⁴²	333 eyes	No cycloplegia/ retinoscopy	5.5	0.75–1.24 DC = 2.4% 0.75–1.24 DC = 0.0% ≥ 1.25 DC = 1.8% ≥ 1.25 DC = 0.0%	WTR ATR WTR ATR
			8.5	0.75–1.24 DC = 2.7% 0.75–1.24 DC = 0.6% ≥ 1.25 DC = 2.7% ≥ 1.25 DC = 0.0%	WTR ATR WTR ATR
			10.5	0.75–1.24 DC = 2.4% 0.75–1.24 DC = 0.6% ≥ 1.25 DC = 2.7% ≥ 1.25 DC = 0.0%	WTR ATR WTR ATR
			12.5	0.75–1.24 DC = 3.0% 0.75–1.24 DC = 0.3% ≥ 1.25 DC = 3.0% ≥ 1.25 DC = 0.0%	WTR ATR WTR ATR
Dobson et al. ²⁹	98 children	Cyclopentolate 1%/ retinoscopy	2	11% ≥ 1.00 DC	35% ATR
	97 children		3	32% ≥ 1.00 DC	55% ATR
	105 children		4	37% ≥ 1.00 DC	10–30% ATR
	108 children		5	50% ≥ 1.00 DC	50–60% WTR
	87 children		6	41% ≥ 1.00 DC	15–30%
	93 children		7	45% ≥ 1.00 DC	Oblique age 4 and older
	90 children		8	18% ≥ 1.00 DC	
	68 children		9	13% ≥ 1.00 DC	
Howland & Sayles ³²	61 children	No cycloplegia/ photorefraction	1.5	42% ≥ 1.00 DC	50–78% ATR for all ages
	29 children		2.5	20% ≥ 1.00 DC	<10% WTR
	60 children		3.5	10% ≥ 1.00 DC	10–50% oblique
	70 children		4.5	12% ≥ 1.00 DC	
Gwiadza et al. ³¹	63 children	No cycloplegia/ near retinoscopy	1–2	43% ≥ 1.00 DC	55–65% ATR up to age 4.5
	86 children		2–3	30% ≥ 1.00 DC	
	137 children		3–4	22% ≥ 1.00 DC	20–40% ATR and 60–80%
	140 children		4–5	18% ≥ 1.00 DC	
	53 children		5–6	24% ≥ 1.00 DC	WTR after age 4.5
Zadnik et al. ⁴³	231 children	Tropicamide 1%/ autorefraction	6–12	8.6% ≥ 1.00 DC	30% ATR 50% WTR 20% oblique

WTR, With the rule; ATR, against the rule.

for corneal development.^{35,38} A recent, detailed study of ocular components and infant astigmatism found that infant astigmatism was the combination of predominantly with-the-rule corneal toricity and against-the-rule lenticular toricity. The reduction in the prevalence of astigmatism over time appeared to be due to the decreases in toricity of the cornea and anterior lens surface, and the reduction in the variance in toricity of the cornea and both lenticular surfaces.²

Table 2-4 shows results of studies on older children, ranging in age from 1 to 15 years.^{7,18,27,34,39} Here, the process of emmetropization and the development of juvenile-onset myopia are evident. The mean refractive error shifts from hyperopic to near emmetropic, as a result of both emmetropization and the increasing number of myopes with age.

The effect of race and ethnic background on the distribution of refractive error is discussed below. Table

2-5 shows the distribution of refractive error by age of school-age children.^{7,12,18,21,39,40} These results again document the decreasing prevalence of hyperopia and concomitant increasing prevalence of myopia with increasing school age.

The prevalence of astigmatism in school-age children is presented in Table 2-6.^{29,31,32,41-43} The prevalence of clinically significant astigmatism decreases with increasing school age by all measurement methods.

During the high school years, refractive error is thought to be stable. Little eye growth appears to occur during these years, and myopes stay myopes, hyperopes stay hyperopes, and emmetropes remain emmetropes, with little change in degree of refractive error within an individual in a given category.^{12,44} However, the phenomenon of adult-onset myopia is well documented.¹¹ The modern literature on the prevalence of myopia in general population samples and academia-based samples is summarized in Table 2-7,⁴⁵⁻⁴⁷ as reported by the Working Group on Myopia Prevalence and Progression.¹¹ These samples reflect the high prevalence seen in some college-age populations; some of these myopes reflect actual adult-onset myopia, whereas others represent adult progression of existing myopia from school-age onset.

The Working Group on Myopia Prevalence and Progression¹¹ carefully analyzed secular trends in the prevalence of myopia in college-based samples. They could document no difference between the prevalence of myopia in the early 20s as tallied in 1920 to 1930⁴⁸⁻⁵⁰ and the modern National Health and Nutrition Examination Survey (NHANES) data, despite the increased percentage of the general population enrolled in college in the 1980s. The association with education or near work found in many studies of adult myopes⁵¹⁻⁵⁴ and the implication that excessive near work somehow causes myopia are discussed later. The Beaver Dam Eye

Study⁵⁵ documented a decrease in the prevalence of myopia in older age groups, similar to that found in Eskimo samples years ago,⁵⁶ but whether this reflects a true secular trend, the effect of increasing near work, the effect of a Westernized diet (in the case of the Eskimos) or a change in diet (in Beaver Dam, Wisconsin), some selective mortality of aged myopes, or a multitude of other factors is difficult to determine.

Gender

The trends in refractive error distribution seen with gender, as opposed to age, are not as well defined and may in fact be confounded by age. A large sample of children from the United Kingdom yielded no significant differences in refractive error between boys and girls.⁵⁷ In other studies, the trend has gone both ways, and it is therefore probably inconclusive. Hirsch¹⁴ found a more myopic mean refraction in boys than in girls among 5- to 6-year-olds but more myopia among girls by age 14 years, and Alsbirk⁵⁸ reported a similar trend in adults. These results are also sensitive to the operational definition of myopia as described in Table 2-2. Myopia is more prevalent in Danish school-age girls than in Danish boys of all ages,²⁵ but both myopia and hyperopia are more prevalent in Finnish schoolchildren than in Goldschmidt's Danes.⁵⁹ Both groups of investigators questioned the influence of puberty and earlier maturation typically found in girls. Further, it is possible that all the above results favoring females are due to their greater participation in studies of this type.¹¹

Ethnicity

Contemporary data supporting the general clinical impression that the prevalence of myopia differs with race are relatively sparse (Table 2-8). Few studies have simultaneously compared different races, and it is problematic at best to compare data across races in different geographic areas, different cultures, and samples with different socioeconomic and educational bases. The best estimate comes from the NHANES data reported by Sperduto et al.,¹² but no prevalence rates for races other than white and African-American are reported. Across all age groups, the prevalence of myopia in whites (26%) was twice that in African-Americans (13%) in the NHANES survey. That difference was the most marked among 18- to 24-year-olds (differing by a factor of 3 times) and least evident among 45- to 54-year-olds (a difference of only 1.5 times).

The claim that there is a high prevalence of myopia among Asians is difficult to document. Oft-cited high prevalence rates among Singapore medical students⁶⁰ parallel those of British biomicroscopists⁶¹ and American optometry students,⁶² both predominantly white samples. Given the association between myopia and intelligence, higher educational level certainly inflates the prevalence of myopia in these samples.⁶³

TABLE 2-7 Prevalence of Myopia in Modern Young Adult Samples, Either Population- or Academia-Based

Study	Myopia	Type of Sample
Nakamura ⁴⁶	20% of Caucasians 30% of Nisei	Military recruits
Sutton & Ditmars ⁴⁷	45% at entrance 60% at graduation	West Point cadets
Gmelin ⁴⁵	51% at entrance 67% at graduation	West Point cadets

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TABLE 2-8 Literature-Based Prevalences of Myopia by Ethnic Category

Race/Ethnicity	Source	Age	Prevalence of Sample
African-American	NHANES ¹²	12–54 yr	13.0%
Caucasian (white)	NHANES ¹²	12–54 yr	26.3%
Caucasian (white)	Beaver Dam Eye Study ⁵⁵	43–84 yr	26.2%
Caucasian (white)	Fledelius ⁷⁴	16–66+ yr	30.0%
Caucasian (white)	Hawaii ¹⁸⁰	Schoolchildren 5–18 yr	7.0%
Asian	Hawaii ¹⁸⁰	Schoolchildren 5–18 yr	13.0%
Hispanic	Hawaii ¹⁸⁰	Schoolchildren 5–18 yr	5.0%
Asian	Oakland, California, public schools ¹⁸¹	Sixth-grade schoolchildren	13.7%
Hispanic	Oakland, California, public schools ¹⁸¹	Sixth-grade schoolchildren	9.1%
African-American	Oakland, California, public schools ¹⁸¹	Sixth-grade schoolchildren	5.1%
Asian	Hong Kong schools ⁶⁴	Schoolchildren 6–17 yr	55.0%
Asian	Taiwan schools ¹⁸²	Schoolchildren 6–12 yr	17.6%
Caucasian (white)	OLSM	Schoolchildren 6–14 yr	8.8%
Asian	OLSM	Schoolchildren 6–14 yr	27.3%
Caucasian (white)	CLEERE ¹⁸³	Schoolchildren 5–17 yr	4.4%
Asian	CLEERE ¹⁸³	Schoolchildren 5–17 yr	18.5%
African-American	CLEERE ¹⁸³	Schoolchildren 5–17 yr	6.6%
Hispanic	CLEERE ¹⁸³	Schoolchildren 5–17 yr	13.2%

NHANES, National Health and Nutrition Examination Survey; OLSM, Orinda Longitudinal Study of Myopia; CLEERE, Collaborative Longitudinal Evaluation of Ethnicity and Refractive Error.

The larger population-based studies and assessments of school-age children listed in Table 2-8 indicate a general pattern in which the prevalence of myopia is highest in Asian, intermediate in white, and lowest in African-American schoolchildren. Table 2-9 presents similar comparisons of college-age students. Regardless of the person's racial background, when myopia does occur it is presumably due primarily to excessive axial length. There is no evidence to support the idea that myopia in some ethnic groups is caused by steep corneas or high-powered crystalline lenses. Component studies of Asian schoolchildren demonstrate that their myopia is due to excessive axial length, as it is in white children.⁶⁴ Rigorous ocular component measurement in samples with a great deal of ethnic variation is rare; the Orinda Longitudinal Study of Myopia (OLSM) suffers from this limitation, too.⁷ However, there is intriguing evidence that children from a group with a low prevalence of myopia show different ocular component profiles than do those with a higher prevalence.^{65,66} Specifically, Melanesian children ages 6 to 19 years had a prevalence of myopia of 2.9%,⁶⁵ whereas Malaysian children appeared similar to U.S. samples,³⁹ with a prevalence of myopia of 4.3% at 7 to 8 years and 25.6% by 15 to 16 years.⁶⁶

Cross-sectional differences between 6- and 17-year-old Melanesian and Malaysian children were compared.⁶⁶ The Melanesian children on average had no

TABLE 2-9 Literature-Based Prevalences of Myopia in College-Age Samples by Ethnic Category

Ethnicity	Source	Age (yr)	Prevalence of Myopia
Chinese (in Singapore)	Au Eong et al. ¹⁸⁴	17–18	48.5%
Eurasian (in Singapore)	Au Eong et al. ¹⁸⁴	17–18	34.7%
Indian (in Singapore)	Au Eong et al. ¹⁸⁴	17–18	30.4%
Malay (in Singapore)	Au Eong et al. ¹⁸⁴	17–18	24.5%
Israeli	Rosner & Belkin ⁵⁴	17–19	15.8%
British	Sorsby et al. ¹⁸⁵	18–22	11.0%
Swedish	Goldschmidt ²⁵	18–22	14.5%

change in lens thickness but substantial decreases in lens power (3.50 D) during the time the eye increased in length by 1 mm and the prevalence of myopia increased to 8%. In contrast, Malaysian children underwent substantial lens thinning (0.33 mm) and smaller

decreases in lens power (2.40 D) during a similar 1-mm increase in axial length that resulted in a prevalence of myopia of 28%. The lack of difference in axial growth suggests that the crystalline lens of the Malaysian children was primarily responsible for the increased myopia. It appeared that the lens was less efficient in compensating for ocular growth, decreasing fewer diopters per unit change in axial length. This lens-based model parallels that obtained from the predominantly white sample in the OLSM, suggesting that a slower growth rate for the Malaysian lens or a less responsive refractive index profile may be a risk factor for the onset of myopia. Unfortunately, the Malaysian study did not collect complete ocular component data (e.g., lens curvatures from phakometry) or longitudinal data.

Geography

Geographical differences in myopia prevalence can be seen in Table 2-10, although differences among studies with regard to sampling and confounding by other factors such as diet, education, and time spent reading are difficult to sort out from true differences based on geography. Certainly, no clear geographical differences emerge beyond those identified in the preceding section on racial differences in myopia prevalence in the United States.

Diet

Table 2-11 shows some of the various dietary insufficiencies that have been associated with myopia. Gardiner⁶⁷ conducted a nonrandomized clinical trial of the effect of animal protein supplement on the progression of myopia in children. The treated group either altered diet or took protein supplements to make 10% of the intake of calories come from animal protein. The control group followed their regular, unmonitored diet. At one year, the treated group showed less myopic

progression (by -0.25 to -0.50 D per year) compared with the control group. The effect of dietary protein on myopic progression displayed some dose-response effect, in that myopic progression was less in those who achieved the highest levels of intake of animal protein. Unfortunately, this trial was not randomized. The treated children were studied at the hospital clinic by their parents, whereas the control children were seen at their school clinic. Without randomization, unknown sources of bias—factors related to myopia but unrelated to the treatment—may create differences between treated and control children and give false results. For example, differences in family income, parents' educational level, or whether one or both parents were myopic could create differences in myopic progression unrelated to protein intake. Interestingly, the treated children differed from controls in refractive error, but they were initially more myopic rather than less.

Edwards et al.⁶⁸ examined the nutrition and diet of 102 7-year-old Hong Kong children. Compared with children who did not become myopic, 34 children who were myopic by age 10 years had a lower intake of several items, including: protein; fat; vitamins B₁, B₂, and C; phosphorus; iron; and cholesterol. None of these children were malnourished. Malnourishment appears to be associated with a lower amount of hyperopia in infancy, perhaps because of some effect it might have on the crystalline lens⁶⁹ or because it slows ocular growth. The smaller eyes of premature infants are not associated with high hyperopia but rather with myopic or less hyperopic refractive errors.^{70,71}

Some investigators have proposed that proper calcium metabolism is important to maintenance of scleral rigidity and resistance to any expansive effects of intraocular pressure (IOP). Lane⁷² found that myopes had a higher concentration of calcium in hair samples than do emmetropes or hyperopes. Lane somewhat arbitrarily took this as evidence that calcium was being depleted from the body into the hair. Calcium supple-

TABLE 2-10 Literature-Based Prevalences of Myopia by Geographical Location

Location/Ethnicity	Source	Age (yr)	Prevalence of Myopia
Israel/Jewish	Hymans et al. ¹⁸⁶	>40	11.6%
Israel/Jewish	Shapiro et al. ¹⁸⁷	University students 18–25	13.0%
Nigeria/Black	Abiose et al. ¹⁸⁸	12–20	<2.0%
United States/Native American	Wick & Crane ¹⁸⁹	6–10	13.0%
Australia/Aborigines	Taylor ¹⁹⁰	20–30	4.8%
Australia/European origin	Taylor ¹⁹⁰	20–30	13.5%
Alaska/Eskimo	van Rens & Arkell ¹⁹¹	5–80+	44.7%

Hymans et al. defined myopia as >-1.00 DS; Shapiro et al. and Wick & Crane defined myopia as ≥-0.25 DS in at least one eye; Abiose et al. did not define myopia criterion. Taylor defined myopia as >-0.75 DS; van Rens & Arkell did not define myopia criterion.

TABLE 2-11 Dietary Problems Associated with Myopia Study

Study	Dietary Problem
Gardiner ⁶⁷	Less animal protein
Edwards et al. ⁶⁸	Less protein, fat, vitamins, iron, and cholesterol
Halasa & McLaren ⁶⁹	African tribe after 2 years of famine
Feldman ⁷³	Calcium
Lane ⁷²	Calcium, chromium
Tamura & Mitsui ¹⁹¹	Japanese children with exposure to organophosphorus particles

mentation appeared to have little effect on slowing myopia progression in a case series reported by Feldman.⁷³ Blood levels of total, bound, and ionic calcium were not appreciably different between myopes and hyperopes.

Until the multitude of confounding factors that influence diet and refractive error is untangled, the precise role of nutrition in the etiology of myopia will remain unclear. Demonstrating that alteration of diet can affect the onset or progression of myopia will require a formal, randomized clinical trial potentially fraught with ethical dilemma.

Time

If analyzing the foregoing factors' influences on refractive error distributions is difficult, determining whether secular trends have occurred in the distribution of refractive error across the last century is nearly impossible. Researchers working in different decades used different measurement methods and different criteria for defining the types of refractive error. Different age groups cannot be directly compared. Comparisons among groups of different ethnic origins and from different parts of the world do not lead to conclusions. Nevertheless, Table 2-12 presents an attempt to sort out these relationships.¹¹

In a study designed to determine whether the prevalence of myopia might be increasing, Fledelius⁷⁴ looked at a Danish hospital-based sample and found the same trend toward decreasing prevalence of myopia with increasing age into the elderly range that was found in the Beaver Dam Eye Study in Wisconsin⁵⁵ and the oft-cited study of Alaskan Eskimos.⁵⁶ It is still unknown whether these trends reflect true increases in the prevalence of myopia in recent decades (argued against by the juvenile-onset data across decades in Table 2-12),

TABLE 2-12 Secular Trends in Prevalence of Juvenile Onset Myopia

Age (yr)	Study	≥ -1.00 DS Myopia
6	Kempf et al. ⁴⁰	
	Blum et al. ³⁹	2.0%
7	Kempf et al. ⁴⁰	1.2%
	Blum et al. ³⁹	3.0%
8	Hirsch ¹⁴	0.9%
	Blum et al. ³⁹	3.5%
9	Hirsch ¹⁴	1.9%
	Blum et al. ³⁹	5.5%
10	Kempf et al. ⁴⁰	2.4%
	Blum et al. ³⁹	7.5%
11	Hirsch ¹⁴	4.4%
	Blum et al. ³⁹	10.5%
12	Blum et al. ³⁹	12.0%
13.0	Kempf et al. ⁴⁰	3.9%
	Blum et al. ³⁹	13.0%
14	Hirsch ¹⁴	5.4%
	Blum et al. ³⁹	14.5%

changes in the ocular components related to aging, sampling bias, or some other effect or association.

Personality

The conventional wisdom on associations between personality traits and refractive error is that myopia is associated with introversion.⁷⁵ Further, myopes have been shown to exhibit an inhibited disposition, a disinclination for motor activity and social leadership, whereas hyperopes are carefree, impulsive, hyperactive, and socially passive.⁷⁶ These associations are statistically weak, however.^{77,78}

Systemic Conditions

Numerous systemic disorders have an effect on the development of the eye and therefore affect its refractive state. A more complete discussion of the following conditions has been provided by Curtin.⁷⁹ Albinism is the inability to produce the pigment melanin, resulting in a lack of pigmentation of the hair, skin, and eyes. The prevalence of the general form of albinism is 1 in 10,000. In both the generalized and ocular forms, albinism has been associated with myopia and high astigmatism. Down syndrome, or trisomy of chromosome 21, results in myopia in about one third of affected individuals. Its prevalence is estimated to be between 2 and 34 in 10,000. Several connective tissue and skeletal disorders, such as Marfan's syndrome, Ehlers-Danlos syndrome, and Stickler's syndrome, are also associated with myopia. Maumenee⁸⁰ has characterized the refractive

error and the facial, somatic, sensory, and ocular characteristics of other, rarer connective tissue disorders. Most result in myopia, but two (Jansen's syndrome and spondyloepiphyseal dysplasia) may result in hyperopia. Laurence-Moon/Bardet-Biedl syndromes are characterized by a retinal pigment degeneration and general somatic abnormalities such as polydactyly and mental retardation. Refractive errors are rarely emmetropic, with nearly equal numbers of myopes and hyperopes.⁸¹ Homocystinuria, an error of metabolism, is characterized by excretion of homocystine in the urine and excesses of homocystine and methionine in the blood.⁸² The effect on the body is the production of fair hair and skin and mental retardation. The major ocular findings are lens dislocation and myopia in a high proportion of affected individuals (90% for each), and light irides (70%), hypotony (33%), retinal elevation (25%), and cataract (20%). Numerous severe developmental disorders were also listed by Curtin⁷⁹ as associated with myopia as the common refractive error: Pierre Robin syndrome, syringomyelia, Turner's syndrome, Noonan's syndrome, and De Lange's syndrome.

More recent reports have noted that myopia may be an associated finding in hantavirus infection. A multi-hospital study followed 62 serologically proven cases during 1992 and 1993, and acute myopia was found in 24% of patients.⁸³ Systemic lupus erythematosus has also been related to myopia in two case reports, presumably because of changes in lenticular curvature and anterior displacement associated with ocular edema.^{84,85}

Dental caries is a common systemic condition with low morbidity that has been associated with myopia in past literature but has not been found to be a significant risk factor for myopia in subsequent studies. Hirsch and Levin⁸⁶ found an association between myopia and dental caries, as well as between the amount of myopia and the number of decayed teeth in 155 college-age students. No such relationship was found in a sample of 196 high school students by Keller⁸⁷ or in a sample of 102 Hong Kong children at age 9 years by Edwards and Chan.⁸⁸

Fledelius⁷⁴ noted an increase in the prevalence of myopia among patients with diabetes in a cross-sectional study of 1416 patients referred for general eye examinations. Among diabetic patients, 10.2% had refractive errors between -1.00 and -1.75 D, compared with 6.2% of nondiabetics. Myopia of -2.00 D or less was found in 12.3% of persons with diabetes and 9.9% of persons without diabetes.

Ocular Diseases

It is clear from both animal and human data that clear visual input is necessary for normal emmetropization to occur. Rabin et al.⁸⁹ reported on 80 subjects with binocular and monocular disruptions of normal vision from

sources such as cataract, retroental fibroplasia, and ptosis. The distribution of refractive errors of these groups was shifted toward more myopia or less hyperopia compared with patients who experienced normal development. Numerous conditions that interfere with normal vision have been reported to affect refractive error through the induction of a deprivation-like myopia. These include corneal opacification,⁹⁰ eyelid closure,⁹¹ vitreous hemorrhage,⁹² and congenital cataract.⁹³ Astigmatic and myopic spherical equivalent refractive errors have been reported in connection with hemangioma.⁹⁴ Astigmatism is also reported to increase after the surgical correction of congenital ptosis.⁹⁵

In addition, Nathan et al.⁹⁶ analyzed the refractive error distribution of 433 pediatric patients with low vision. In contrast to the foregoing diagnoses, which interfere with normal vision through media opacity, this pediatric population suffered from retinal and neurological disease. All diseases disrupted emmetropization in that the variation in refractive error was quite high. The majority of diseases were associated with myopia, including aniridia, cerebral palsy, coloboma, glaucoma, nystagmus, optic atrophy, optic nerve hypoplasia, retinitis pigmentosa, retinopathies, retinopathy of prematurity, and toxoplasmosis. Myopigenic conditions appeared to involve predominantly peripheral visual impairment. Only three diagnoses were associated with hyperopia (albinism, maculopathies, and rod monochromacy). These primarily involved foveal development. The magnitude and variability of the refractive errors were more severe with earlier age of onset of the impairment.

A host of hereditary abnormalities with an effect on visual acuity, ocular development, and therefore refractive error were catalogued by Curtin.⁷⁹ Conditions associated with hyperopia include achromatopsia, nystagmus, and microphthalmia. Conditions associated with myopia include achromatopsia, nystagmus, microcornea, keratoconus, Fabry's disease (corneal and lenticular accumulation of glycosphingolipid), microphakia, ectopia lentis, coloboma, choroideremia, gyrate atrophy, fundus flavimaculatus, retinitis pigmentosa, progressive bifocal chorioretinal atrophy, extensive myelination of nerve fibers, and rarer familial diseases such as Wagner's disease (membranous vitreous, arterial sheathing, choroidal sclerosis, and cataract), familial exudative vitreoretinopathy, and familial external ophthalmoplegia.⁹⁷

Glaucoma may be connected to myopic refractive error in two ways. First, as part of the near-work theory of the etiology of myopia, prolonged reading may increase intraocular pressure (IOP), driving the expansion of the eye by mechanical force.^{98,99} Second, glaucoma and myopic refractive errors may be associated conditions. Genetic links have been proposed on the basis of the shared higher prevalence of positive steroid

response among myopes and glaucoma patients.¹⁰⁰ Elevated IOP has been associated with refractive error in nonglaucomatous eyes in several studies,^{100–103} with differences on the order of 1 to 2 mmHg. Other studies have indicated no such association, however.^{104–106} The risk of ocular hypertension appears to be higher in myopes than in emmetropes,¹⁰⁷ as is the risk of open-angle glaucoma and conversion to glaucoma from ocular hypertensive status.¹⁰⁰ Conversely, Daubs and Crick¹⁰⁸ found that, although myopia was a risk factor for glaucomatous field loss, there was no association between ocular tension and refractive error status. The small size of their sample of ocular hypertensives may have limited their statistical power, however.

The two largest studies are by David et al. and Bengtsson.^{102,104} David et al. studied 2403 subjects over the age of 40 who accepted an invitation to participate in a glaucoma screening and found a slightly higher IOP on average in myopes. Bengtsson's sample was more population based, consisting of 88.8% of the total population of 1917 people over the age of 8 years living in a particular village. Although this was arguably the better sample with respect to age range and generalizability, no association between IOP and refractive error was reported by Bengtsson.

Besides keratoconus, other corneal conditions may affect refractive error because of the alterations in corneal curvature they create. Phlyctenular keratitis has been reported to cause myopic changes.⁸¹ Pellucid marginal degeneration thins the inferior cornea and flattens the vertical corneal meridian, resulting in high amounts of against-the-rule astigmatism.¹⁰⁹ Alterations of the refractive properties of the crystalline lens may also affect refractive error. Posterior polar and nuclear cataract tend to produce myopic changes,¹¹⁰ and age-related changes in the gradient index profile of the crystalline lens tend to result in hyperopic shifts.¹¹¹

A change in the elevation of the photoreceptor plane as a result of retinal pathology may also have an effect on refractive error and visual acuity. The classic example is central serous chorioretinopathy, in which the sensory retina is moved anteriorly by the accumulation of fluid underneath it, with a concomitant increase in hyperopia or decrease in myopia.¹¹² High myopia, also termed *pathological myopia*, may have an adverse effect on the retina, because the extremely large eye size stretches and places tension on the retina. A thorough funduscopic evaluation of the central and peripheral retina is required when examining the highly myopic patient. A study of 513 eyes that were at least 24 mm in axial length showed that lattice degeneration, pavingstone degeneration, white with or without pressure, and retinal holes and tears were all significantly associated with a longer axial length.¹¹³ Examination of 308 post-mortem eyes with high myopia collected over a 67-year period showed the following prevalences for retinal

findings associated with myopia: myopic configuration of the optic nerve head, 37.7%; posterior staphyloma, 35.4%; degenerative changes of the vitreous, 35.1%; cobblestone degeneration, 14.3%; myopic degeneration of the retina, 11.4%; retinal detachment, 11.4%; retinal pits, holes, or tears, 8.1%; subretinal neovascularization, 5.2%; lattice degeneration, 4.9%; Fuchs spot, 3.2%; and lacquer cracks, 0.6%.¹¹⁴

FACTORS ASSOCIATED WITH REFRACTIVE ERROR

Heredity

Genetic factors also play a significant role in the incidence of myopia. Goldschmidt²⁵ provided an extensive review of this literature. A recent study demonstrated similarities between siblings that were not observed between parent and offspring, due to the interaction of dominant genes, the visual environment, or a combination of the two.¹¹⁵ Less has been written about the heritability of component characteristics of myopia, though Alsbirk⁵⁸ found an apparently lower heritability for refractive error than for axial length, anterior chamber depth, or corneal curvature.

Previous studies on the familial patterns of ocular component and refractive error development differ on how large a role genetics plays in myopia and whether the role of heredity differs for classical juvenile-onset myopia and high myopia.¹³ Large-scale pedigree studies attempting to identify the specific mode of inheritance have been few and far between and vary widely in the proposed mode of inheritance.^{116,117}

Studies of the heritability of refractive error and the ocular components consist of two types: those based on correlations between parents and children and those based on monozygotic and dizygotic twin comparisons. Generally, in studies of parents and offspring, higher heritabilities have been found for axial length and corneal power than for the other ocular components or for refractive error.^{58,118–120} In studies of twins, the heritabilities for corneal power, axial length, and refractive error have all been high and approximately equal,^{118,121,122} and the differences in refractive error and the ocular components have been smaller for monozygotic than for dizygotic twins.^{123–125}

Monozygotic twins resemble each other more closely than dizygotic twins. Sorsby et al.¹²⁴ measured refraction, corneal curvature, anterior chamber depth, lens power and thickness, and axial length in 78 monozygotic twin pairs, 40 pairs of dizygotic same-sex twins, and 48 unrelated pairs. The unrelated pairs were included for comparison because they shared neither genes nor the effects of a common familial environment. Zygosity was determined on the similarity of

physical appearance, fingerprints, tasting of phenylthiourea (if one twin could taste it and the other could not, the twins were considered dizygotic), and blood type.

Monozygotic twins were within ± 1.25 D of each other 90% of the time. Only 55% of dizygotic twins and 52% of unrelated pairs were within ± 1.25 D. Monozygotic twins were within ± 1.65 D of each other 95% of the time. Only 62% of dizygotic twins and 60% of unrelated pairs were within ± 1.65 D. The analysis for refraction and the other ocular components is summarized in Table 2-13.

Teikari et al.¹²⁶ reported on similarities between refractions in 6314 pairs of twins from the Finnish Twin Cohort Study. Correlations in liability for refractive error between monozygotic twins were 0.81 compared to 0.30 to 0.40 for dizygotic twins. From these data, Teikari et al. obtained an estimate of heritability, or the proportion of variability in refractive error that can be accounted for by variability in genetics. Heritabilities were 0.82 for males and 1.02 for females. A recent estimate of heritability from 506 female twin pairs in the United Kingdom also found high heritabilities for refractive error across the spectrum of myopia to hyperopia, 0.84 to 0.86 due to additive genetic effects.¹²⁷

The preliminary results from the first 3 years of the OLSM show that the addition of near work to a parental history of myopia model for predicting refractive error marginally improved the model's predictive ability. Interestingly, the addition of near work did not improve the model's predictive ability for the ocular components. On the other hand, models that begin with near work that attempt to predict refractive error and the optical ocular components are almost all improved by the addition of parental refractive error history information. These results lend credence to a nature model with a modest nurture component.¹²⁸

With this genetic etiology in mind, several studies have linked at least the pathological form of myopia to

several loci. Bornholm eye disease, an X-linked form of myopia characterized by myopia of more than -6.00 D, deutanopia, and moderate optic nerve hypoplasia, was reported to be linked to the distal part of the X chromosome at Xq28.¹²⁹ Knobloch syndrome, which includes severe myopia and encephalocele, has been mapped to 21q22.¹³⁰ Other recent molecular genetic studies of families with two or more individuals with -5.50 D or more of myopia have found significant linkage with regions on chromosomes 18p11.31,¹³¹ 12q21-23 Young et al.,¹³² 17q21-22,¹³³ and 7q36.¹³⁴ These varied loci suggest substantial heterogeneity in the transmission of pathological myopia. These loci have not been associated with lower amounts of more common myopia.^{135,136} Lower amounts of myopia have been linked recently to regions on chromosome 22q12 in Ashkenazi Jewish families¹³⁷ and 11p13 in the region of the PAX6 gene in twin pairs in the United Kingdom.¹³⁸

Near Work

Human Near-work Theories and Evidence

A body of work on the nurture theory of myopia development indictors excessive reading during childhood as the cause of abnormal eye growth.^{11,139} Examples include an increased prevalence of myopia among the first school-educated Eskimos⁵⁶; a decreased prevalence of myopia during World War II in Japan¹¹; the association between myopia, intelligence, and near work⁵¹⁻⁵⁴; and the observation of adult-onset myopia in college populations.¹¹ Experimental and epidemiological lines of evidence have indicated that schooling, study, reading, and other near work are associated with excessive axial elongation and myopia,^{51,54,56,63,140-143} but evidence that near work directly causes myopia is difficult to obtain from purely observational studies.

The characteristic of accommodation that has most consistently been associated with refractive error is tonic

TABLE 2-13 Range of Values for Refraction and the Ocular Components over Which 95% of Monozygotic Twin Pairs Agree and the Percentage of Dizygotic and Unrelated Pairs Who Fall within That Interval

Ocular Component	Monozygotic 95% Interval	Within Monozygotic 95% Interval	Dizygotic Twins	Unrelated Pairs
Refraction (DS)	± 1.65		62%	60%
Corneal power (DS)	± 1.25		72%	56%
Anterior chamber depth (mm)	± 0.45		92%	92%
Lens power (DS)	± 1.80		75%	73%
Axial length (mm)	± 0.85		58%	65%

Data are from Sorsby A, Sheridan M, Leary GA. 1962. Refraction and Its Components in Twins (*Medical Research Council Special Report Series No. 303*). London: Her Majesty's Stationery Office.

accommodation (TA), or the accommodative state in the absence of an accommodative stimulus. Table 2-14 summarizes findings on TA and other accommodative functions in various refractive error groups.

First, both the level of TA^{144,145} and the degree of accommodative hysteresis after accommodative demand¹⁴⁶ seem clearly characteristic of refractive error type. Myopes show the lowest levels of TA and the greatest hysteresis, whereas hyperopes have higher levels of TA and the least hysteresis. The level of TA also appears to be related to refractive error type in schoolchildren.¹⁴⁷ Second, it has been shown that near work, performed for both the short term^{148,149} and the long term,¹⁵⁰ can alter TA. Such studies of adults have led to a growing belief among some researchers that TA is either a causative agent or a predictive risk factor for myopia. Longitudinal evaluation of this hypothesis has shown that TA is not a predictive risk factor for myopia. Although TA was lower in myopic children in either of

two test conditions, lower values of TA were not predictive of later onset of myopia in an evaluation of over 700 children.¹⁵¹

Animal Model Evidence and Limitations

The effects of environmental manipulation on chicken, tree shrew, and primate ocular development—creating deprivation myopia—provide some of the strongest evidence cited for a significant role for the environment in human myopia.¹⁵² It should be noted, however, that the animal models of myopia are designed only to assess environmental effects; no animal model has been used in which any genetic effect could be found even if it existed. Given the vast array of information on experimental myopia available from the animal models, especially the chicken, and given that parallels are being drawn to human myopia,^{153–155} it is time to ask whether induced myopia in animals is analogous to human myopia in an etiological sense.

TABLE 2-14 Accommodation in Various Refractive Error Groups

Accommodative Function	Study	Late-Onset Myopia	REFRACTIVE ERROR GROUP		
			Early-Onset Myopia	Emmetropia	Hyperopia
Amplitude	McBrien & Millodot ¹⁹³	Highest (≈11.00 D)	Medium to high (≈10.00 D)	Medium to low (≈9.25 D)	Lowest (≈8.50 D)
Tonic accommodation	Maddock et al. ¹⁴⁴ ; McBrien & Millodot ¹⁴⁵	Lowest (0.50 D)	Medium (0.80 D)	Medium (0.80 D)	Highest (1.60 D)
Time for tonic accommodation to stabilize	McBrien & Millodot ¹⁴⁵	Fast (1–2 min)	Fast	Fast	Slow (6–7 min)
Accommodative hysteresis	Owens & Wolf-Kelly ¹⁹⁴ ; McBrien & Millodot ¹⁴⁵	High (shift in myopic direction)	None	None	Low (counteradaptive shift)
Accommodative lag*	McBrien & Millodot ¹⁵⁷	High	High	Medium	Low
Accommodative response gradient	McBrien & Millodot ¹⁹³	Low	Low	Medium	High
Parasympathetic tone	McBrien & Millodot ¹⁴⁵	Low	Normal	Normal	High
Sympathetic tone	Gilmartin & Hogan ¹⁹⁵	Low	Normal	Normal	High
Effect of cognitive demand	Bullimore & Gilmartin ¹¹⁵ ; Bullimore & Gilmartin ¹⁹⁶	Higher	Not examined	Lower	Not examined

*0.50 D difference from highest to lowest at 5 D near stimulus.

Besides the inability of animal models to identify and investigate genetic influences on myopia development, there are three main obstacles to the uniform application of animal models to the human condition. First, there is no deprivation of form vision in the environment of the school-age child as severe as that required to induce myopia in animals. Second, the sensitive period for deprivation myopia in animals appears to be too early to account for human juvenile-onset myopia. Third, studies of the chicken using spectacle lenses to create dioptric blur involve a choroidal thickness modulation that has no known human analog.

Inducing experimental myopia by creating visual deprivation in chickens and tree shrews (through the use of translucent plastic) and primates (through lid suture) depends on a profound disruption of form vision and attendant reduction in contrast, yet there is no analogous experience in the normal visual world of the developing myope during childhood. The only candidate for this analogous visual deprivation in humans has been the developing myope's lag of accommodation, which provides a small and intermittent error of focus that would have to degrade the retinal image sufficiently to drive abnormal human eye growth.¹⁵⁶ Although myopes have been shown to exhibit a higher amount of accommodative lag, both as adults¹⁵⁷ and in childhood,¹⁵⁸ it is unclear whether the accommodative lag is the cause or the result of their refractive error and whether this lag is in fact of sufficient magnitude to parallel deprivation in animals.

Application of the deprivation/blur model from animal studies to juvenile-onset myopia is questionable, because the age of onset of myopia in humans is much later developmentally than that induced in the experimental animals. The annual incidence of myopia in children is low and relatively constant until the age of 8 years, when it rises sharply and continues to rise until it stabilizes at age 14 years.³⁹ Once juvenile-onset myopia occurs, it tends to progress until the age of 15 to 17 years.¹⁰ Therefore, humans' sensitivity to deprivation would have to occur between the ages of 8 and 16 years. The evidence from animal models identifies a sensitive period in both the chicken¹⁵⁹ and the monkey¹⁶⁰ for producing myopia by deprivation. This period in primates corresponds to human ages from birth to 7 years,¹⁶¹ well before the period during which juvenile-onset myopia develops and progresses. Although it is more difficult to equate chicken or tree shrew and human developmental stages, it is clear that the chicken's sensitivity to deprivation is greatest at hatching¹⁵⁹ and that the tree shrew's sensitivity begins 15 days after eye opening and decreases thereafter.^{162,163} Neither is greatest at "school age."

The animal myopia models' sensitive periods are more similar to the sensitive period for deprivation myopia that occurs in children between birth and 6

years from sources of true visual deprivation, such as hemangioma,⁹⁴ cataract,¹⁶⁴ corneal opacity,⁹⁰ and vitreous hemorrhage.⁹² For animal models of deprivation myopia to be directly relevant to juvenile-onset myopia development, measurable myopia in older animals resulting from small, constant levels of contrast reduction must be demonstrated. Such experiments have not yet been conducted.

Experiments in the chicken in which spectacle lenses have been used to stimulate an accommodative response could shed light on the role, if any, of near work in human myopia. Contrary to claims made in the literature that such experiments support an environmental etiology for human myopia, spectacle lenses stimulating positive accommodation have not produced myopia in a dose-dependent fashion in the chicken.¹⁶⁵ A more accurate tuning response to both plus- and minus-inducing lenses has been demonstrated in other laboratories,¹⁶⁶⁻¹⁶⁸ but, surprisingly, the choroid appears to be responsible for it.¹⁶⁸ Nonetheless, humans do not negatively accommodate, and choroidal thickness is not a significant factor in human refractive error. Magnetic resonance imaging on a small sample of hyperopes, emmetropes, and myopes, differing in refractive error by an average of 10.00 D, showed that choroidal thickness differed among the three groups by an average of 0.4 mm (1.00 DS equivalent). Differences were not noted in the peripheral choroid.¹⁶⁹

Intelligence

Numerous studies have documented associations between intelligence, school achievement, and myopia. Myopes tend to have higher scores on tests of intelligence and cognitive ability^{54,170-174} and better grades^{16,175} than do other refractive error groups. Hyperopes, on the other hand, tend to show poorer reading skill and other perceptual anomalies more frequently.^{140,147,173}

Despite the finding of an association between myopia and near work for well over a century, the relationship between these factors and intelligence has not been adequately investigated. Only one investigator has attempted to analyze all three factors in the same children, obtaining uncertain results. Ashton¹⁷⁵ used self-reported grades in school and results from cognitive tests used in the Hawaii Family Study of Cognition as measures of aptitude and achievement. Numbers of books and magazines read, hours spent doing homework and watching television, and years of education served as measures of near work. Myopia remained associated with near work after correction for aptitude and achievement and was associated with aptitude and achievement after correction for near work. The progression of myopia with age, however, was not related to near work after correction for aptitude and achievement, whereas myopic progression remained

associated with aptitude and achievement even after correction for near work. Two recent studies have also attempted to unravel the interrelationships between near work, aptitude, and parental history of myopia. Each found an association between near work and the aptitude variables tested, Iowa Tests of Basic Skills¹⁷⁶ or Raven Standard Progressive Matrices (IQ).¹⁷⁷ Each study also found an association between parental history of myopia and children's myopia. When all three variables were placed in models testing the relative strength of their associations with myopia, the study conducted in the United States found that all three variables were independently related to myopia with parental history of myopia had the strongest association.¹⁷⁶ The study conducted in Singapore found that near work and parental history became insignificant when controlled for IQ, making IQ the most important factor.¹⁷⁷ Either the Iowa Tests assess something different than the Raven Matrices, or there may be ethnic variation in what is most important in myopia. Interestingly, each study found that near work was less important when another variable was controlled for, such as IQ or parental history of myopia.

Socioeconomic Status

The association between refractive error and socioeconomic status has been documented in two reports from large population-based studies: cycle III of the Health Examination Survey (1966 to 1970) and the National Health and Nutrition Examination Survey (NHANES) from 1971 to 1972. In each study, myopes tended to be overrepresented among the higher socioeconomic strata and underrepresented among lower income levels. Angle and Wissmann¹⁴⁰ categorized 15,536 subjects ages 12 to 17 years old by refractive status and 10 levels of family income. (Note that the absolute value of family income loses some meaning with time due to inflation.) Myopia was least frequent in the lowest income group (16.8% of those with incomes below \$500) and increased steadily with increasing income to 35.1% of those with incomes over \$15,000. Sperduto et al.¹² analyzed data from 5282 subjects ages 12 to 54 years. Once again, myopia was least frequent among the families with the lowest incomes (10.0% to 26.6% of those with incomes below \$5000) and most common in the highest income families (27.6% to 30.3% of those with incomes above \$10,000).

Reports of the prevalence of myopia are often a product of how stringent the criterion for myopia is. This is further complicated in large studies by the impracticality of performing a refraction on all subjects and the inability to use a cycloplegic agent. Myopic refractive error was quantified in these studies as the

spherical equivalent determined from several sources: neutralization of spectacles, trial lens power that improved distance acuity, and retinoscopy. Hyperopia was not quantified.

One possible source of the association between family income and myopia is the association between intelligence, education, and myopia. The probability of earning a higher wage certainly increases with intellectual ability and success in school. This ability and achievement are also related to myopia (see Intelligence). It is unclear whether this association represents environmental or genetic influences. On the genetic side, myopia may be related to intelligence because of some link between genes for both traits. On the environmental side, those who are more intellectually inclined and who succeed in school are probably also doing more near work than those who do not have as successful an academic experience. If the specific and independent contributions of each factor toward the prevalence of myopia are to be untangled, both factors must be assessed within the same study. To date, no researchers have conclusively performed such an analysis, and the question remains open.

SUMMARY

After reading this chapter, it might seem that the threats to emmetropia are so numerous and that so many factors are needed to produce it (e.g., normal visual experience and the harmonious and disease-free development of all ocular optical components) that emmetropia should be a rarity. What is remarkable is not that emmetropia happens at all, but that it is actually the rule, occurring so commonly that the distribution of refractive error is highly peaked near emmetropia and far from normal. Clinically, however, the emmetrope is an infrequent visitor for refractive vision care before presbyopia occurs. Ametropia is encountered as a rule in clinical practice. Information in this chapter should further the clinician's understanding of the frequency with which a patient's refractive error occurs in the population and what demographic variables may possibly influence it. Assessment of refractive error may help in the diagnosis and management of the associated systemic and ocular pathologies described in this chapter. It may also guide the examination of the eye, as in the case of examination of the fundus of the highly myopic patient. Discussing the factors associated with refractive error will provide valuable information to patients. Unfortunately, although many of the factors described in this chapter suggest actual etiologies, definitive data are not available that might provide patients with information on the relative contributions of each factor. Such information awaits further study.

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3

Development of the Ametropias

David A. Goss

In this chapter, developmental changes in human ocular refractive error and some of the prevailing theories of the etiology of refractive error are discussed. The emphasis is on clinical studies, but some laboratory studies that shed light on the causes of refractive error and the influences on refractive development are discussed. Different periods in the human lifespan have recognizable trends in changes in refractive error. Refractive changes in each of the following periods are discussed: (1) infancy and early childhood (from birth to about 5 years of age), (2) childhood and adolescence (from about 5 years of age to the middle or late teen years), (3) young adulthood (from the middle or late teen years to about 40 years of age), and (4) later adulthood (starting at about 40 years of age). It may be noted that this age division is virtually identical to that used by Grosvenor¹ in his classification of myopia by age of onset and age-related prevalence.

REFRACTIVE CHANGES FROM BIRTH TO 5 YEARS OF AGE

Mohindra and Held² reported a study of 400 full-term infants in Massachusetts, in whom refractive error was measured in a dark room by manifest retinoscopy.^{3–6} There was a wide distribution of refractive errors in the first month of life, from more than –10.00 D of myopia to more than +5.00 D of hyperopia. The distributions of refractive errors in seven age groups are given in Figure 3-1. In the birth to 4-week age group, the mean refractive error was –0.70 D. The mean shifted toward hyperopia with increasing age, being +0.59 D for the 129- to 256-week (roughly 2.5 to 5 years) age group. The standard deviation decreased from 3.20 D in the birth to 4-week age group to 0.85 D in the 129- to 256-week age group. This can be observed in the narrowing of the distribution of refractive errors with age in Figure 3-1. These changes could be explained by both myopic and hyperopic infant shifting toward emmetropia.

Ingram and Barr⁷ presented longitudinal refractive error data for 148 children in the United Kingdom. Refractive measurements were made by retinoscopy

under cyclopentolate cycloplegia.⁸ From 1 year of age to 3.5 years of age, the prevalence of myopia decreased and the prevalence of emmetropia increased, because children with myopia at 1 year of age shifted toward hyperopia. Children with hyperopia between +1.00 and +2.25 D tended to have decreases in hyperopia. Some children with +2.50 D or more hyperopia increased in hyperopia, and about the same number had decreases in hyperopia.

Gwiazda et al.⁹ presented data for 72 children seen at regular intervals from before 6 months of age for periods of 9 to 16 years. Refractions were performed by the Mohindra⁴ dark-room retinoscopy procedure (see Chapter 30) for children up to 3 years of age and by standard manifest retinoscopy procedures (see Chapter 18) for children older than 3 years. Thirty-one infants had negative spherical equivalent refractions in the first 6 months of life. The mean spherical equivalent refractive error for this group moved toward emmetropia in the first year of life and crossed to the hyperopic side by about 2.5 years of age. Twenty subjects in their study had refractive errors of +0.50 D or more plus before 6 months of age. The mean refractive error decreased from over +1.50 D before 1 year of age, reaching about +1.00 D at about age 2 years and staying around +0.75 to +1.00 D to past 5 years of age. Like Mohindra and Held,² Gwiazda et al.⁹ found that the standard deviation of refractive error decreased over the first 12–18 months of life.

There is a higher prevalence of astigmatism in infants than in older children and adults.^{10–12} Samples of primarily white infants have been reported to have a high prevalence of against-the-rule astigmatism, which decreases over the first few months and years of life.^{13–15} Dobson et al.¹³ reviewed cycloplegic refraction records of consecutive patients in a Boston hospital medical center. In 85 children under 3.5 years of age, against-the-rule astigmatism was found 2.5 times as often as with-the-rule astigmatism. In children between 5.5 and 9.5 years of age, with-the-rule astigmatism was 3 times as common as against-the-rule astigmatism. Gwiazda et al.¹⁴ determined refractive error by Mohindra's dark-room retinoscopy procedure in 1000 children ages 0 to

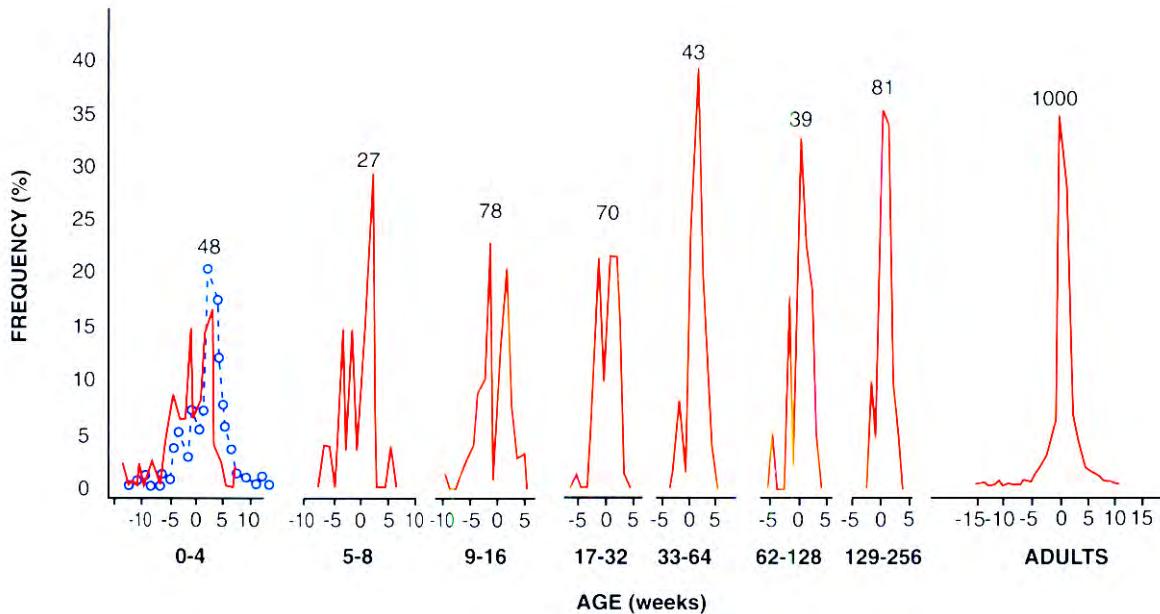


Figure 3-1

Distributions of refractive errors during the first 5 years divided into seven age groups. Data are from Mohindra I, Held R. 1981. Refraction in humans from birth to five years. *Doc Ophthalmol Proc Series* 28:19–27. For comparison, the refractive error distribution for adults is also shown, which comes from Sorsby A, Sheridan M, Leary GA, Benjamin B. 1960. Visual acuity and ocular refraction of young men. *Br Med J* 1:1394–1398.

6 years in Cambridge, Massachusetts. Against-the-rule astigmatism was more common than with-the-rule astigmatism before 4.5 years of age, but after that with-the-rule astigmatism was more common. There were 29 children who had 1.00 DC or more of astigmatism at 6 months of age. Of these children, 16 had against-the-rule, 8 had with-the-rule, and 5 had oblique astigmatism. All 29 had reductions in astigmatism by the time they were 4 to 6 years old. Some of the infants who had against-the-rule astigmatism shifted to with-the-rule astigmatism by 6 years of age. Howland and Sayles¹⁵ took photorefraction measurements of 312 infants and young children in New York State. The prevalence of astigmatism of 1.00 DC or more was approximately 7 times greater in children 1 year old or younger than in children 4 years old or older. For children up to 2 years old, the ratio of types of astigmatism was 15 against-the-rule to 9 oblique to 1 with-the-rule.

The tendency toward decreasing against-the-rule astigmatism in the first few years of life was also observed by Abrahamsson et al.,¹⁶ who followed 299 Swedish 1-year-olds for 3 years. The infants were selected on the basis of having at least 1.00 DC of astigmatism in one or both eyes at about 1 year of age. Refractions were performed annually by retinoscopy after instillation of cyclopentolate. Right eyes were used for analysis. In a few cases it was only the left eye that had at least 1.00 DC of astigmatism at the beginning of the study, and the right eye had 0.50 DC of astigmatism. By 2 years of age, one-sixth of the subjects had spheri-

cal refractions. By 4 years of age, one-third of the subjects had spherical refractions. Most of the subjects who lost their astigmatism had started with 1.00 DC of astigmatism, but some lost as much as 2.50 DC. Of the 299 subjects, 272 (91%) had against-the-rule astigmatism. The cylinder axis usually did not change significantly during the 3 years of the study. During the 3 years, there was a shift in the distribution of amount of astigmatism to lower values. There were 49 subjects who started with at least 2.00 DC of astigmatism. Most of them experienced a decrease in astigmatism, but 9 (18%) experienced an increase. Of these 9, a disproportionate number (5) had with-the-rule astigmatism.

Premature infants, especially those with very low birth weights, are often found to have a high degree of myopia.¹⁷ Usually, this myopia decreases with maturity over the first few months of life. Many of these infants are emmetropic by 1 year of age if no other ocular anomalies develop.^{18,19} In a longitudinal study conducted in Israel, Scharf et al.²⁰ found that 42% to 45% of 134 eyes of premature babies were myopic shortly after birth. Of these myopic eyes, 46% were emmetropic at 7 years of age. Of the eyes that were hyperopic at birth, 77% were emmetropic at 7 years of age.

The retinopathy of prematurity often occurs as a consequence of the high oxygen concentration necessary to keep low birth weight infants alive.²¹ The associated ocular media opacification, known as *retrolental fibroplasia*, results in a high myopia.^{22,23} Distributions of refractive error in children born with low birth weight

are similar to those of children born full-term, except for the greater number of children with high myopia in low birth weight groups.^{24,25}

REFRACTIVE CHANGES DURING THE SCHOOL-AGE YEARS

Longitudinal Studies on Populations not Selected by Visual Characteristics

Classic longitudinal studies of refractive error conducted on populations of school-age children not selected by visual characteristics were reported by Hirsch²⁶⁻³² and by Langer.³³ Hirsch performed his study in the town of Ojai, California, collecting refractive data by manifest retinoscopy during school screenings performed twice a year. The data used for analysis were the means of the spherical equivalents obtained for the right eye and left eye for each subject at each screening session. Hirsch found that in most cases the change in refractive error from 6 or 7 years of age to 11 or 12 years of age was linear. For the children whose changes in refractive error were linear, the mean slope was -0.07 D per year. There was a negative skew in the distribution of slopes of refractive error change, because of the higher negative slopes for children with myopia.

In one of the reports on his longitudinal study, Hirsch³¹ presented data on refractive error at 13 or 14 years of age as a function of what the refractive error had been at age 5 or 6 years. Included were data for 766 eyes of 383 children. When the children in the study were 13 to 14 years old, 92 of the 766 eyes had at least -0.50 D of myopia, 605 were classified as emmetropic

(refractions of -0.49 to +0.99 D), and 69 had hyperopia of at least +1.00 D. Hirsch randomly selected 100 of the 605 emmetropic eyes for data analysis. His comparison of refractive errors at the two different ages is given in Table 3-1. Presuming that the 100 eyes are representative of the sample, each of the numbers in the emmetropia column could be multiplied by about 6. On the basis of the data in Table 3-1, the following conclusions may be reached: (1) children with +1.50 D or more of hyperopia at 5 or 6 years of age will still be hyperopic at age 13 or 14 years; (2) the majority of children with refractive errors of +0.50 to +1.24 D at 5 or 6 years of age will be in the emmetropic range (defined by Hirsch as -0.49 to +0.99 D) at 13 or 14 years; (3) most children who enter school with refractions of 0 to +0.49 D will be myopic at 13 or 14 years of age; and (4) children who are myopic at 5 or 6 years will become more myopic.

Langer³³ performed his study in Leaside, Ontario, a suburb of Toronto. Manifest retinoscopy was performed when children were in kindergarten and first grade and every other year thereafter. The data used for analysis were spherical equivalents. From the age of 5 or 6 years to 15 or 16 years of age, refractive error changed linearly in 93% of the children. The mean rates of refractive error change were -0.21 D per year for girls and -0.16 D per year for boys. The reason these mean rates are more negative than the mean rate in Hirsch's study is that there were more children with myopia in Langer's population. Like Hirsch, Langer found that the distribution of rates of refractive error change had a negative skew as a result of myopes' having higher negative rates of change. He found that the refractive error at 15 or 16 years of age could be predicted within 0.50 D by linear extrapolation from the first 3 points in 81% of the cases.

TABLE 3-1 Predictability of Refractive Errors at Age 13 or 14 Years as a Function of Refractive Error at Age 5 or 6 Years

SPHERICAL EQUIVALENT REFRACTION AT AGE 13-14 YR	SPHERICAL EQUIVALENT REFRACTION AT AGE 5-6 YR		
	Myopia ≥ 0.50 D	Emmetropia -0.49 to +0.99 D	Hyperopia ≥ 1.00 D
Over -0.26 D	4	0	0
-0.25 to -0.01 D	6	0	0
-0.00 to +0.24 D	7	6	0
+0.25 to +0.49 D	37	4	0
+0.50 to +0.74 D	21	33	5
+0.75 to +0.99 D	15	41	10
+1.00 to +1.24 D	2	15	14
+1.25 to +1.49 D	0	1	7
Over +1.50 D	0	0	33
TOTAL	92	100	69

Numbers are numbers of eyes. The emmetropia group at age 13 to 14 is a random sample of 100 out of 605 eyes. Data are from Hirsch MJ. 1964. Predictability of refraction at age 14 on the basis of testing at age 6—interim report from the Ojai Longitudinal Study of Refraction. Am J Optom Arch Am Acad Optom 41:567-573.

Changes in Hyperopes Compared with Myopes

Both Hirsch and Langer noted that, among schoolchildren, the greatest changes in refractive error occurred in those with myopia. Hofstetter³⁴ had shown this earlier, using patients' records from an optometry practice in Bloomington, Indiana. For myopes between the ages of 10 and 20 years, almost all of the changes were toward increased myopia. The distribution of refractive change for the hyperopes was normal, with a mode of zero.

Hofstetter's conclusion³⁴ that refractive change is faster when a child crosses from hyperopia into myopia was supported by Mäntyjärvi.³⁵ Mäntyjärvi's analysis was based on the right eye spherical equivalents of cycloplegic refraction in children 7 to 15 years of age. Forty-six hyperopic children and 133 myopic children were followed for at least 5 and up to 8 years. The children with hyperopia had a mean rate of refractive error change of -0.12 D per year ($SD = 0.14$, range = $+0.11$ to -0.45 D per year). The children with myopia had a mean rate of -0.55 D per year ($SD = 0.27$, range = 0 to -1.63 D per year). There were also 30 children who were initially hyperopic and became myopic during the period of observation. While the children were hyperopic, their mean rate of refractive error change was -0.21 D per year ($SD = 0.21$, range = $+0.25$ to -0.75 D per year). While they were myopic, the mean rate was -0.60 D per year ($SD = 0.45$, range = -0.08 to -1.63 D per year).

Mäntyjärvi's findings were supported by three studies in Hong Kong. Lam et al.,³⁶ studying 6- to 17-year-olds, found a mean rate of refractive error change of -0.46 D/yr in myopes compared to -0.17 D/yr in nonmyopes. Edwards³⁷ reported a mean rate of -0.51 D/yr for myopes between the ages of 7 and 12 and -0.10 D/yr for nonmyopes of the same ages. Fan et al.³⁸ found mean rates of -0.63 D/yr for myopes and -0.29 D/yr for nonmyopes in the 5- to 16-year age range.

Onset of Myopia in Youth

Using data from several studies in different locations (see Chapter 1), Grosvenor¹ proposed a system for the classification of myopia based on its age-related prevalence and age of onset. The four types of myopia in this system are congenital, youth-onset, early adult-onset, and late adult-onset. Youth-onset myopia has its onset in the school-age years and is the most common type of myopia. Most studies give a prevalence of myopia of about 2% at 5 or 6 years of age and a prevalence of 20% to 25% at 15 to 16 years of age.¹

The increased prevalence of myopia during the school-age years is illustrated by cross-sectional data from vision screenings by Hirsch³⁹ and Young et al.^{40,41} and by the previously mentioned longitudinal data

from Langer.³³ Hirsch reported prevalence based on manifest retinoscopy of the right eyes of 9552 schoolchildren in the Los Angeles area. Young et al. reported prevalence based on manifest retinoscopy of the right eyes of 652 children in Pullman, Washington. The findings of these studies are summarized in Table 3-2. In the data of Young et al.,^{40,41} there was a pronounced jump in prevalence for girls from 7 and 8 years of age to 9 and 10 years of age and a similar large increase in prevalence for boys from 9 and 10 years of age to 11 and 12 years of age. Large increases in prevalence, although not quite as obvious as in the data of Young et al.,^{40,41} can also be observed in the Hirsch and Langer data sets at similar age spans. From this it may be inferred that the most common ages of myopia onset in girls precede those in boys by about 2 years. Further support for a tendency toward later incidence for boys comes from refraction data from 3000 Ohio schoolchildren,⁴² as shown in Figure 3-2. In a study of several thousand schoolchildren in Hong Kong,³⁸ the incidence of myopia increased in boys from 9% at 6 years of age or less to nearly 20% at 10 years of age. In girls, incidence increased from about 12% at 6 years of age or less to over 27% at 11 years of age.

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Figure 3-2

Percentage of children with myopia (horizontal meridian of the right eye) in a random sample of 3000 schoolchildren in Ohio. Plotted in the upper panel are the raw data and in the lower panel are the data treated by smoothing by threes. (From Hirsch MJ. 1963. *The refraction of children*. In Hirsch MJ, Wick RE [Eds], *Vision of Children*, p 153. Philadelphia: Chilton.)

TABLE 3-2 Prevalence of Myopia in Schoolchildren Based on Manifest Retinoscopy in Studies Conducted in the Los Angeles Area (Hirsch³⁹); Pullman, Washington (Young et al.^{40,41}); and Leaside, Ontario (Langer³³)

	AGE (YR)				
	5-6	7-8	9-10	11-12	13-14
Any amount of myopia (%)					
Hirsch ³⁹					
Girls	6.15	9.71	17.18	21.60	25.36
Boys	7.43	11.02	15.68	20.74	22.53
Langer ³³					
Girls	2.04	3.97	12.20	29.18	34.52
Boys	0.00	3.08	11.68	20.48	34.30
Myopia greater than 1.00 D (%)					
Hirsch ³⁹					
Girls	0.45	0.98	2.01	5.77	5.78
Boys	0.67	0.90	1.82	3.08	5.08
Young et al. ^{40,41}					
Girls	4.17	2.60	19.44	20.00	25.71
Boys	0.00	5.62	9.68	27.27	28.57
Langer ³³					
Girls	0.00	0.00	6.71	10.26	19.58
Boys	0.00	1.54	5.11	5.71	15.01

Progression of Childhood Myopia

Once myopia appears in childhood, it increases until the middle to late teens.⁴³ Typical patterns of childhood myopia progression are shown in Figures 3-3 and 3-4. Additional examples of patterns of childhood myopia progression, shown in Figure 3-5, include some points before the onset of myopia and show that the refractive change accelerates at the onset of myopia. Perusal of the examples in these figures suggests that the change in refractive error is largely linear from the beginning to the end of childhood myopia progression. Using an F test for linearity and visual inspection, Langer³³ determined the refractive changes to be linear in all of the children with myopia greater than -0.50 D in his study population. Goss⁴⁴ studied the linearity of the change in refractive error with age from ages 6 to 15 years in the optometric practice records of 198 children with myopia. Of the cases, 90% to 94% were found to fit a linear model by an F test for linearity and the statistical significance of the correlation coefficient. When cases such as those in Figure 3-5 were excluded, 96% were found to be linear. In 100% (57 of 57) of the patients who had seven or more examinations from age 6 to 15 years, changes were linear.

The rate of myopia progression varies considerably from one child to another. Goss and Cox⁴⁵ calculated rates of childhood myopia progression using linear

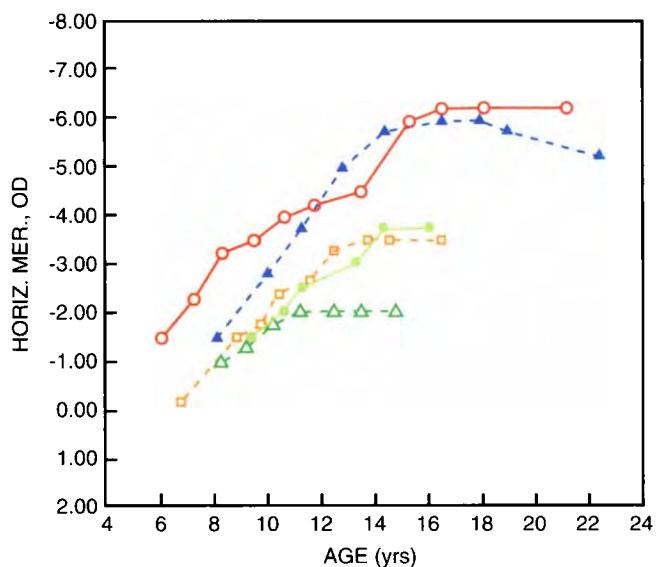


Figure 3-3

Typical patterns of childhood myopia progression. Data are from five male subjects from Goss DA, Winkler RL. 1983. Progression of myopia in youth: Age of cessation. *Am J Optom Physiol Opt* 60:651-658. Refractive error in the horizontal meridian of the right eye (HORIZ. MER., OD) is plotted on the y-axis. Each set of common symbols represents the refractive findings for one person.

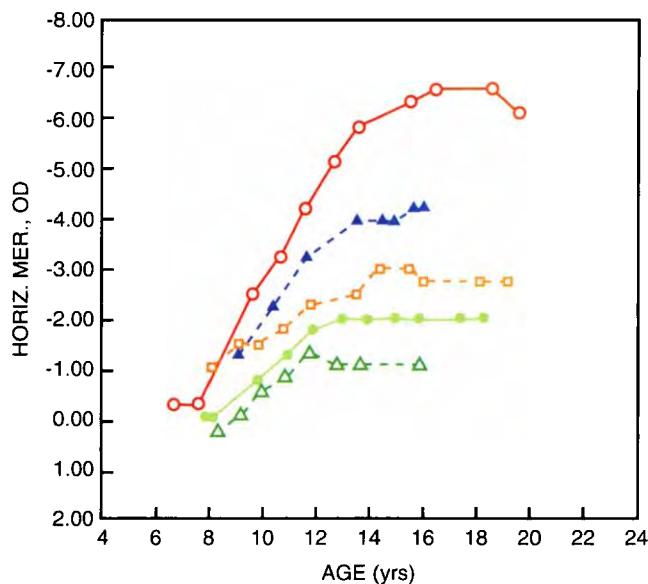


Figure 3-4

Typical patterns of childhood myopia progression. Data are from five female subjects from Goss DA, Winkler RL. 1983. Progression of myopia in youth: Age of cessation. *Am J Optom Physiol Opt* 60:651–658. Refractive error in the horizontal meridian of the right eye (HORIZ. MER., OD) is plotted on the y-axis. Each set of common symbols represents the refractive findings for one person.

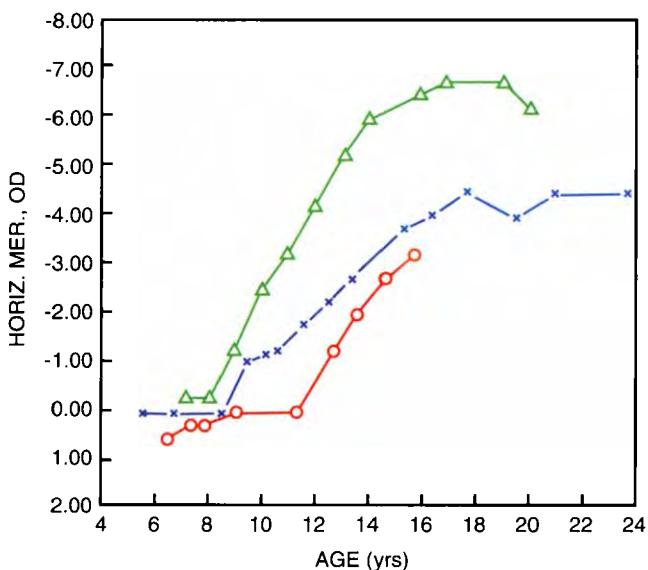


Figure 3-5

Patterns of childhood myopia progression illustrating acceleration of refractive change at the onset of myopia. Refractive error in the principal meridian nearest horizontal in the right eye (HORIZ. MER., OD) is plotted on the y-axis. (From Goss DA. 1987. Linearity of refractive change with age in childhood myopia progression. *Am J Optom Physiol Opt* 64:779.)

TABLE 3-3 Distribution of Childhood Myopia Progression Rates in Diopters per Year

Rate	No. of Males	No. of Females
+0.20 to 0.00	0	4
-0.01 to -0.20	37	20
-0.21 to -0.40	51	48
-0.41 to -0.60	41	44
-0.61 to -0.80	15	23
-0.81 to -1.00	12	7
-1.01 to -1.20	2	1
-1.21 to -1.40	0	0
-1.41 to -1.60	0	1

From Goss DA, Cox VD. 1985. Trends in the change of clinical refractive error in myopes. *J Am Optom Assoc* 56:611.

regression analysis on data collected from five optometry practices in the Midwest. Refractive data were the refractive errors in the principal meridian nearest horizontal in the right eye from manifest subjective refractions. Rates were determined for patients who had four or more refractions before the age of 15 years. The mean rate for males was -0.40 D per year ($SD = 0.24$, range = -0.01 to 1.09 D per year), and the mean rate for females was -0.45 D per year ($SD = 0.25$, range = $+0.12$ to -1.52 D per year). The distribution of rates is given in Table 3-3.

Mäntyläjärvi³⁵ studied rates of progression among myopic children examined at a community health center in Finland. Refractive data were the spherical equivalents of the right eye from retinoscopy after instillation of cyclopentolate. Children were followed from age 5 to 8 years up to the age of 15 years. For 133 children (75 girls and 58 boys), the mean annual rate of change was -0.55 D per year ($SD = 0.27$, range = 0 to -1.63 D per year). The standard deviation and the range were similar to those found by Goss and Cox,⁴⁵ but the mean rate was more negative.

Usually childhood myopia progression slows or stops in the middle to late teens. Goss and Winkler⁴⁶ used four methods to derive an index of the age at which childhood myopia progression stops or slows. Manifest subjective refraction data were collected from three optometry practices in the upper Midwest United States. One method for derivation of childhood myopia progression cessation ages was to determine the best fitting straight line through points from 6 to 15 years of age by linear regression analysis. Myopia cessation age was the age at which the regression line intersected the zero slope line through the mean amount of myopia found

at examinations after 17 years of age. The mean cessation age for 66 males was 16.66 years ($SD = 2.10$), and the mean cessation age for 57 females was 15.21 years ($SD = 1.74$). The difference in cessation age between males and females was statistically significant ($p < .0001$). The standard deviations of about 2 years reflect quite a bit of variability in cessation age.

On the basis of the data presented thus far, we can construct a typical trace of amount of myopia as a function of age. This is illustrated in Figure 3-6. Before the onset of myopia, there is a slow reduction in the amount of hyperopia (shown in Figure 3-6 as the line segment before the onset of myopia). At about the time the child's vision crosses into myopia (labeled "onset of myopia in youth" in Figure 3-6), there is an acceleration of refractive change. Onset age can be at any time in childhood, after which increases in myopia (childhood myopia progression) are generally linear, continuing to a cessation age sometime in the middle to late teens. The rate of childhood myopia progression is quite variable from one individual to another. After the cessation age, the graph may have zero slope or further, generally smaller, increases in myopia (young adulthood myopia progression, which is discussed later in this chapter).

Ocular Optical Component Changes in Progression of Childhood Myopia

The ocular optical component change responsible for childhood myopia progression is axial elongation of the vitreous chamber of the eye. Some of the first evidence for this came from longitudinal studies conducted in England by Sorsby. Sorsby et al.⁴⁷ measured refractive error by cycloplegic retinoscopy, corneal power by keratometry, and crystalline lens power by phakometry and calculated axial length from the refractive error, corneal power, and crystalline lens power. They observed that during childhood, axial length increased and the refractive power of the eye decreased, the latter change resulting from decreases in crystalline lens power and, to a lesser extent, decreases in corneal power. Occurring by themselves, axial length increases resulting from increases in vitreous depth would cause refractive error changes toward myopia, and decreases in ocular refractive power would cause refractive error changes toward hyperopia. Sorsby and Leary⁴⁹ published longitudinal data for 129 children, 25 of whom were myopic. They noted that myopia developed when the refractive effects of axial elongation exceeded the effect of decreased

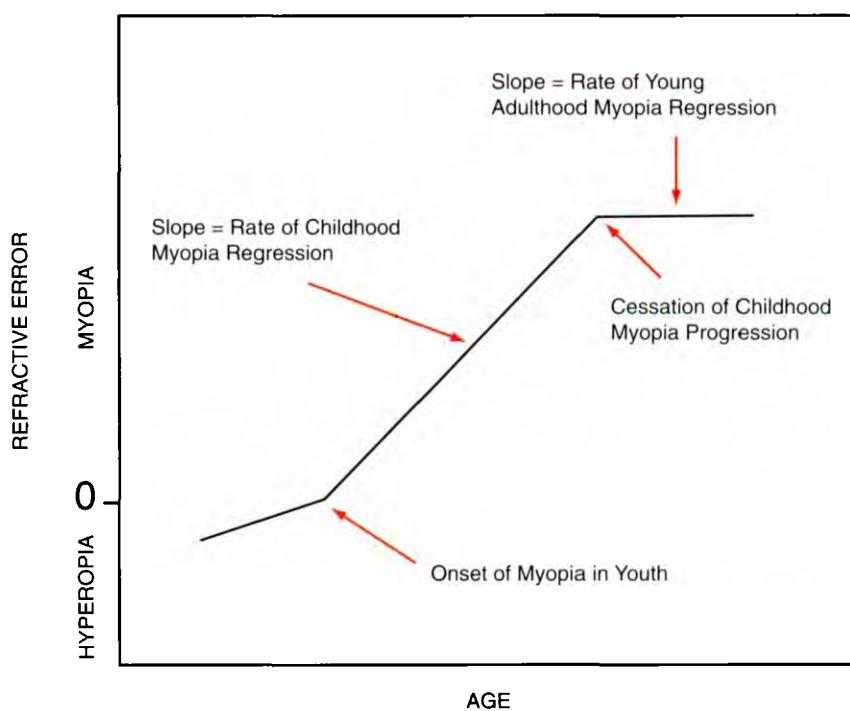
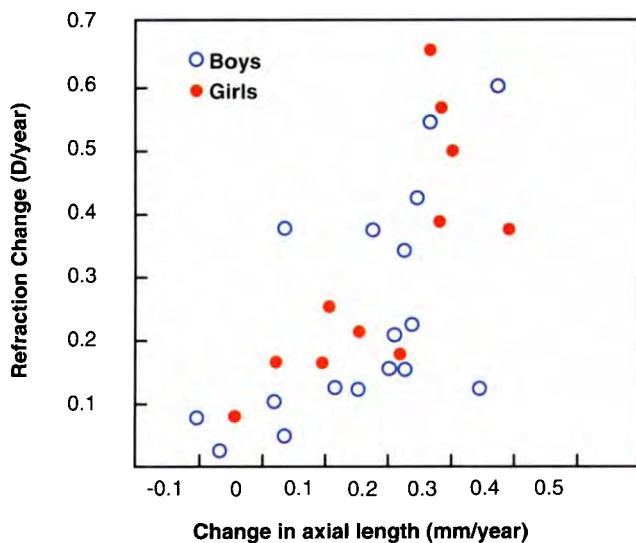


Figure 3-6

Generalized pattern of childhood myopia progression starting from about 5 or 6 years of age and extending into young adulthood. The slopes of childhood and young adulthood progression vary from one person to another, as do onset and cessation ages.

**Figure 3-7**

Relationship between change in axial length and myopic refractive error change for 25 myopic children in the study by Sorsby and Leary (1970). (From Grosvenor TP. 1989b. Primary Care Optometry, 2nd ed, p 38. New York: Professional Press.)

ocular refractive power. Grosvenor⁵⁰ plotted the myopic change in refractive error as a function of increase in axial length in the 25 myopes from Sorsby and Leary's study⁴⁹ and found a high correlation (Figure 3-7).

In Japan, Tokoro and Kabe⁵¹ also observed axial elongation in association with myopia progression (Table 3-4). For the two younger age categories, axial length increased an average of 0.32 mm per year. Individual increases in axial length were shown graphically by Tokoro and Suzuki⁵²; their graphs are reproduced in Figure 3-8. The upper set of plots shows the sequence of myopia increases up to about 15 or 16 years of age that is typical of childhood myopia progression. In the lower group of plots, it may be noted that axial length similarly increases until the middle to late teens. Cross-sectional data suggest that axial length increases that are associated with normal growth of the eye in emmetropic and hyperopic children stop by the early teens but that axial elongation in myopic children continues to the middle to late teens, similar to the progression of childhood myopia.^{47,53,54} Emmetropic children between the ages of 6 and 14 years show increase in axial length, decrease in crystalline lens thickness, and decrease in crystalline lens power.⁵⁵

Fledelius⁵⁶⁻⁶¹ provided additional data showing axial elongation to be the component mechanism of childhood myopia progression. Fledelius reported on changes in refractive error and the ocular components in Danish children between 10 and 18 years of age. The subjects were 70 children who had been born with low birth weight (<2000 g) and 67 children who had been

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Figure 3-8

Changes in refractive error (upper panel) and axial length (lower panel) with age. (From Tokoro T, Suzuki K. 1969. Changes in ocular refractive components and development of myopia during seven years. Jpn J Ophthalmol 13:31.)

born full-term in a Copenhagen hospital. As a result of selection criteria, there were more myopic children in this sample than in a random sample of the population. As the summary of the data in Table 3-5 shows, the mean changes in anterior corneal radius, anterior chamber depth, and crystalline lens thickness were not significantly different from zero. Most of the increases in axial length could be attributed to increases in vitreous depth. Fledelius⁵⁷ reported statistically significant correlations between change in vitreous depth and change in refractive error between the ages of 10 and 18 years ($r = -0.62$ for the low birth weight subjects and -0.76 for the full-term subjects). Although the cornea was steeper in the low birth weight subjects, the ocular optical component change associated with childhood myopia progression in both the low birth weight and full-term subjects was increased vitreous depth.

Lam et al.³⁶ also found a high correlation of increase in vitreous depth with change in refractive error among 6- to 17-year-old Hong Kong schoolchildren. The coefficients of correlation (r) were -0.76 for males and -0.69 for females.

A comparison of myopic and emmetropic young adults found greater vitreous depth, corneal power, and

TABLE 3-4 Mean Changes in Refractive Error and Ocular Optical Components in 1 Year in Myopes

Variable	7- TO 10-YR-OLDS (18 EYES)		11- TO 15-YR-OLDS (15 EYES)		16- TO 22-YR-OLDS (9 EYES)	
	M	SD	M	SD	M	SD
Refractive error (D)	-0.60	0.27	-0.70	0.38	-0.13	0.15
Corneal power (D)	-0.06	0.08	-0.03	0.08	-0.02	0.16
Crystalline lens power (D)	-0.36	0.31	-0.23	0.15	-0.05	0.19
Axial length (mm)	+0.32	0.11	+0.32	0.16	+0.03	0.04

Data are from Tokoro T, Kabe S. 1964. Relation between changes in the ocular refraction and refractive components and development of the myopia. Acta Soc Ophthalmol Jpn 68:1240-1253.

TABLE 3-5 Mean Refractive Error and Ocular Optical Component Values at Age 18 Years and Mean Changes Between Ages 10 and 18 Years in Danish Children with Low Birth Weight and Full-Term Birth

	MALES		FEMALES	
	LBW (n = 36)	FT (n = 36)	LBW (n = 34)	FT (n = 31)
Means at age 18				
Refractive error (D)	-2.2	-0.2	-0.9	-0.6
Anterior corneal radius (mm)	7.67	7.93	7.56	7.82
Axial length (mm)	24.23	24.19	23.38	23.73
Mean changes between ages 10 and 18				
Refractive error (D)	-1.75	-1.26	-1.29	-1.07
Anterior corneal radius (mm)	-0.004	+0.020	-0.005	+0.003
Anterior chamber depth (mm)	+0.08	+0.17	+0.08	+0.09
Lens thickness (mm)	+0.03	-0.02	-0.01	-0.02
Vitreous depth (mm)	+0.77	+0.58	+0.58	+0.41
Axial length (mm)	+0.90	+0.73	+0.64	+0.48

Data are from Fledelius.^{56-58,60,61}

LBW, Low birth weight; FT, full-term birth.

greater posterior crystalline lens radius in myopes.⁶² The differences in anterior chamber depth, crystalline lens thickness, anterior crystalline lens radius, and crystalline lens equivalent power between myopes and emmetropes were not statistically significant. There are also gender differences in the ocular optical components, with females tending to have shorter eyes, steeper corneas, and more powerful crystalline lenses in both school children⁶³ and young adults.⁶²

Factors That Affect the Rate of Childhood Myopia Progression

The earlier in life the onset of myopia occurs, the greater the amount of myopia developed by the late teens to young adulthood.^{45,64-67} Septon's⁶⁷ plot of average

myopia in young adulthood as a function of age of onset is given in Figure 3-9. There does not seem to be any correlation between the times childhood myopia progression starts and stops.⁴⁵ A higher rate of childhood myopia progression is associated with earlier onset of myopia.⁶⁸⁻⁷² For example, Rosenberg and Goldschmidt⁷² found a mean annual increase in myopia of -0.47 D (SD = 0.28) for 30 girls with onset of myopia at 9 to 10 years. Thirty-six girls with onset at 11 to 12 years had a mean annual increase in myopia of -0.37 D (SD = 0.42). Related to the relationship of higher progression rates with earlier onset is the fact that higher progression rates occur in children who already have a higher amount of myopia at a given age.^{70,71}

As a group, people with myopia spend more time reading and doing other forms of near work, more often

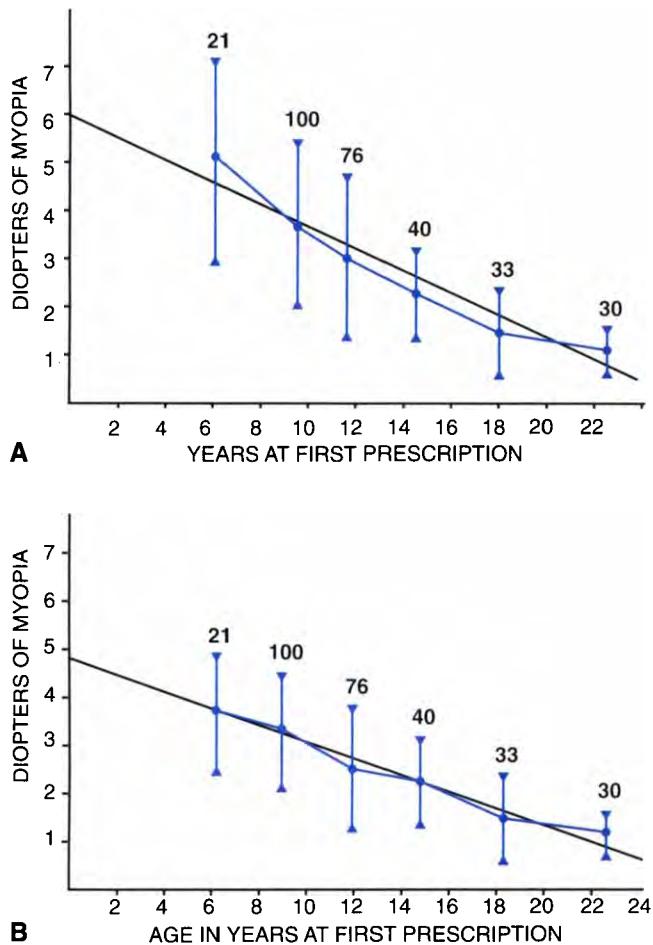


Figure 3-9

Average amount of myopia in optometry students as a function of age of onset. Plotted on the x-axis is the average age at first prescription for myopia for 2.5-year interval groupings. A, Data for 300 students with myopia of 6.12 D or less. B, Data for 332 students with myopia of 11.00 D or less. The bars indicate 1 standard deviation. The number of persons in each grouping is shown above each point. (From Septon RD. 1984. Myopia among optometry students. *Am J Optom Physiol Opt* 61:748.)

have occupations that require near work, have better reading ability, and have more years of education than do nonmyopes.⁷³⁻⁸⁷ A study conducted in Finland^{88,89} revealed a relationship between greater childhood myopia progression over a 3-year period and greater amount of time spent on near work. Children were first seen at 9 to 11 years of age. Refractions were performed after instillation of cyclopentolate. The child and accompanying parent completed a questionnaire that included questions about use of the eyes. There was a low but statistically significant correlation between change in refractive error and amount of time spent reading and doing near work daily ($r = -0.25, p < .001$). Shorter reading distance was also associated with greater

myopia progression ($r = +0.22, p < .001$).⁸⁸ Multiple regression analysis of the data from this study revealed an association between greater time spent on close work and greater myopia progression in both boys and girls and an association between closer reading distance and greater myopia progression in girls but not boys.⁸⁹ Jensen⁹¹ did not find that amount of time spent reading affected myopia progression rate. A cohort of Danish schoolchildren followed for 2 years was divided into those who averaged 2 hours of reading or less per day and those who averaged more than 2 hours per day. The amount of myopia progression in 2 years was not significantly different in the two groups.

Jensen⁹¹ reported a difference in myopia progression rates as a function of intraocular pressure in Danish children. The refractive data used for analysis were right eye autorefraction findings after cyclopentolate cycloplegia. Intraocular pressure was measured with a Goldmann applanation tonometer. Children were first seen at 9 to 12 years of age. Twenty-seven children with intraocular pressure over 16 mmHg had myopia progression that averaged -1.32 D ($SD = 0.70$) over the next 2 years. The mean increase in myopia in 2 years for 20 children with intraocular pressure of 16 mmHg or less was -0.86 D ($SD = 0.55$). The difference in the amounts of myopia progression was statistically significant at the 0.05 level. Jensen also reported that children with pigment crescents at the optic nerve head (observed by ophthalmoscopy) had greater myopia progression than did children with no such fundus changes. Twenty-eight children with crescents had a mean myopia progression of -1.39 D over the next 2 years. Twenty-one children without fundus changes at baseline had a mean myopia progression of -0.81 D in 2 years. The children who had crescents at baseline also had more myopia at baseline. At the beginning of the 2-year follow-up period, the mean amounts of myopia were -3.26 D in those with crescents and -2.20 D in those without fundus changes. Given that the presence of a crescent is related to the presence and amount of myopia,⁹² the higher rate of childhood myopia progression may be secondary to the fact that higher rates of progression occur in children with higher amounts of myopia at a given age.

Hirsch³¹ found that proportionately more children with against-the-rule astigmatism at 5 or 6 years of age developed myopia by 13 or 14 years of age than did those with no astigmatism or with-the-rule astigmatism. However, once they are myopic, against-the-rule astigmats do not appear to have greater rates of childhood myopia progression. Goss and Shewey⁹³ did not find a difference in myopia progression rates in different types of astigmatism. Rates of progression were calculated by linear regression for both the principal meridian nearest horizontal and the principal meridian nearest vertical from right eye subjective refraction data collected from the patient files of five optometry practices. Included

were myopic patients who had four or more examinations between the ages of 6 and 15 years. Patients were classified according to the type of astigmatism at their initial examination. Considering the horizontal meridian, with-the-rule astigmats had a mean rate of myopia progression of -0.40 D per year ($n = 37$, $SD = 0.27$), children with zero astigmatism had a mean rate of -0.40 D per year ($n = 165$, $SD = 0.22$), and children with against-the-rule astigmatism had a mean horizontal meridian myopia progression rate of -0.44 D per year ($n = 73$, $SD = 0.28$). These rates were not significantly different by analysis of variance. With respect to the vertical meridian, with-the-rule astigmats had a mean myopic progression of -0.46 D per year ($SD = 0.31$), children with zero astigmatism had a mean vertical meridian myopia progression rate of -0.40 D per year ($SD = 0.23$), and children with against-the-rule astigmatism had a mean rate of -0.41 D per year ($SD = 0.28$). The rates for the vertical meridian were not significantly different by analysis of variance.

Pärssinen⁹⁴ also did not find a relation between astigmatism and amount of childhood myopia progression. Children in the third to fifth grades of school in Finland were followed for 3 years. The correlation between amount of astigmatism at the beginning of the 3-year period and amount of myopia progression over the 3 years was not statistically significant ($r = +0.07$, $n = 238$). There was no significant difference between the mean amounts of myopia progression in children who had with-the-rule astigmatism (-1.50 D) and those who had against-the-rule astigmatism (-1.60 D) at the start of the study.

Higher childhood myopia progression rates are associated with near-point esophoria. Roberts and Banford^{95,96} calculated rates of myopia progression for children seen in their optometry practices in New York State. Refractive data used for analysis were the means of the spherical equivalent of the manifest subjective refraction in the two eyes. Near phorias were measured by the von Graefe prism dissociation method (see Chapter 21) with a test target at 40 cm. The mean rate of progression for 76 children with more than 4^{Δ} of exophoria was -0.43 D per year. The mean rate of progression for 105 children with phorias in the range of ortho to 4^{Δ} exophoria was -0.39 D per year. For 167 children with esophoria at near, the mean rate was -0.48 D per year. As the amount of esophoria increased, the rate of progression increased (Figure 3-10). Using right eye manifest subjective refraction data from four optometry practices and two longitudinal university studies, Goss⁷⁰ performed linear regression to determine myopia progression rates in children who had four or more refractions between the ages of 6 and 15 years. The mean rate of progression as a function of the near-point phoria through the habitual near-point prescription is presented in Figure 3-11. Phorias were taken through the subjective refraction

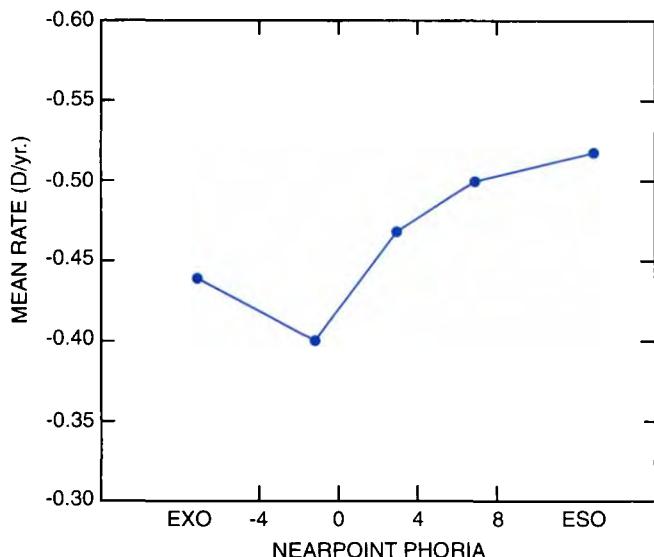


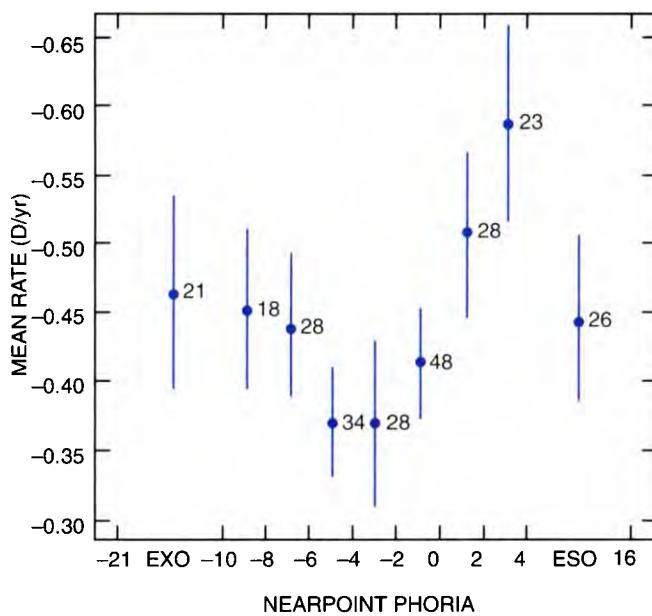
Figure 3-10

Roberts and Banford's data^{95,96} for mean rate of childhood myopia progression as a function of 40-cm dissociated phoria. (From Goss DA. 1994. Effect of spectacle correction on the progression of myopia in children—a literature review. J Am Optom Assoc 65:126.)

using the von Graefe prism dissociation method with a target at 40 cm. For patients who wore bifocal lenses, the habitual phoria was calculated on the basis of the add power and the accommodative convergence/accommodation ratio. Sixty-seven children with exophoria greater than 6^{Δ} had a mean rate of progression of -0.45 D per year ($SD = 0.27$). The mean rate for 110 children with habitual near phorias in Morgan's⁹⁷ normal range from ortho to 6^{Δ} exophoria was -0.39 D per year ($SD = 0.25$). The mean rate for 77 children with esophoria was -0.50 D per year ($SD = 0.32$). The three groups' rates were significantly different by analysis of variance ($p < .025$). The similarity of the results in Figures 3-10 and 3-11 suggests that childhood myopia progression rates are lowest when the near-point phoria is in the normal range, a little higher when the phoria is a high exo, and highest in near-point esophoria.

A summary of the factors associated with higher rates of childhood myopia progression is given in Table 3-6. It also appears that rates of childhood myopia progression may be higher in Asian populations than in predominantly white populations. Mean rates for children wearing single-vision spectacle lenses in various studies in the United States and Western Europe have varied from about -0.30 to -0.60 D per year.* In studies conducted in Asian countries, mean rates have been in the neighborhood of -0.40 to -0.80 D per year.^{36-38,51,103-106}

*References 35, 45, 70, 71, 90, 95, 96, 98-102.

**Figure 3-11**

Mean rate of childhood myopia progression as a function of near-point dissociated phoria through the habitual near-point prescription. Data are from Goss.^{70,100} The error bars indicate 1 standard error, and the numbers are the number of patients for each point. (From Goss DA. 1994. Effect of spectacle correction on the progression of myopia in children—a literature review. J Am Optom Assoc 65:126.)

Myopia Control

The attempt to slow the progression of myopia is often referred to as *myopia control*, and numerous methods have been used to try to achieve it. Most have involved different types of lens treatment regimens or the application of pharmaceutical agents.^{100,107–111} After a brief discussion of pharmaceutical techniques, the two most common methods of myopia control, rigid contact lenses and bifocal spectacle lenses, are discussed in detail.

Pharmaceutical techniques for myopia control have included daily application of atropine, which has been successful in slowing myopia progression.^{112–117} However, this treatment is unpopular with patients, and myopia progression may accelerate upon cessation of the drug treatment.^{118,119} The use of atropine has several disadvantages and side effects, including complete cycloplegia, photophobia from pupillary mydriasis, and the possibility of allergic or idiosyncratic drug reactions or the potential for systemic toxicity if the atropine is not applied properly.^{120,121} It has been suggested that the effect of atropine in slowing myopia progression may come from effects on the retina, rather than from the cycloplegia.¹²² Chronic atropinization may be detrimental to the development of retinal ganglion cells.¹²³ Because of the various problems associated with daily application of atropine, it appears wise to look for other alternatives for myopia control. One pharmaceutical agent that has received some attention is pirenzepine; studies are underway to assess its effectiveness for myopia control.

Even though childhood myopia progression rates have been reported for higher intraocular pressures,⁹¹ progression rates did not decrease when intraocular pressure was lowered by the regular application of timolol.⁹⁰ Children in the second to fifth grades who had at least -1.25 D of myopia participated in a 2-year study. The 49 children in the control group had a mean increase in myopia of -1.14 D (SD = 0.71). The myopia of the 45 children in the timolol group increased an average of -1.18 D (SD = 0.59).

Myopia Control with Rigid Contact Lenses

Rigid contact lenses can slow the rate of childhood myopia progression by flattening the cornea even though axial elongation of the eye continues. Nolan¹⁰¹ reported that 44 patients with myopia who started wearing contact lenses before the age of 14 years had a mean change in refractive error of +0.03 D in 1 year. In comparison, 64 myopic patients who wore spectacle lenses had a mean change of -0.42 D in 1 year.

In England, Stone^{124,125} and Stone and Powell-Cullingford¹²⁶ found mean refractive changes in 5 years of +0.09 D in myopic contact lens wearers and -1.84 D in myopic spectacle lens wearers. As measured by

TABLE 3-6 Factors Associated with Higher Rates of Childhood Myopia Progression

Factor	Study
Earlier onset age and/or higher initial amount of myopia	Bücklers, ⁶⁸ Fletcher, ⁶⁹ Rosenberg and Goldschmidt, ⁷² Grosvenor et al., ⁷¹ Goss ⁷⁰
Near-point esophoria	Roberts and Banford, ^{95,96} Goss ⁷⁰ Jensen ⁹⁰
Temporal crescents and other myopic fundus changes	Jensen ⁹¹
Higher intraocular pressure	Pärssinen et al., ⁸⁸ Pärssinen and Lyyra ⁸⁹
Greater amount of time spent reading and doing near work	Pärssinen et al., ⁸⁸ Pärssinen and Lyyra ⁸⁹
Less time spent on outdoor activities	Pärssinen et al., ⁸⁸ Pärssinen and Lyyra ⁸⁹

keratometry, the cornea flattened an average of 1.01 D in the contact lens wearers and 0.12 D in the spectacle lens wearers in 5 years.

Baldwin et al.⁹⁸ found that fitting contact lenses to minimize corneal flattening achieved no myopia control. Subjects were between the ages of 7 and 13 years. Forty-two contact lens wearers were followed for an average of 11 months. They had average changes in refractive error of -0.54 D (SD = 0.67) in the horizontal meridian and -0.44 D (SD = 0.80) in the vertical meridian. A group of 23 spectacle lens wearers, followed for an average of 10 months, experienced an average myopia progression of -0.34 D (SD = 0.66) in the horizontal meridian and -0.41 D (SD = 0.61) in the vertical meridian. Average changes in corneal power as measured by keratometry were not significantly different from zero in either group.

The foregoing three studies used polymethylmethacrylate contact lenses. A study was conducted in Houston using rigid gas-permeable contact lenses.¹²⁷⁻¹³⁰ One hundred myopic children, between the ages of 8 and 13 years, were fitted with silicone acrylate gas-permeable contact lenses. Fifty-six of the subjects were still in the study at the end of 3 years. These children's myopia increased an average of -0.48 D (SD = 0.70) over the 3-year period, for a myopia progression rate of -0.16 D per year. A control group wearing spectacle lenses had a mean rate of progression of -0.51 D per year. The contact lens wearers had an average corneal flattening of 0.37 D (SD = 0.32) as assessed by keratometry and an average axial length increase of 0.48 mm (SD = 0.48). Twenty-three of them stopped wearing the contact lenses for an average of 2.5 months after the end of the study. During this short period of time, the mean increase in myopia was 0.27 D, and corneal

steepening averaged 0.25 D. The authors concluded that rigid gas-permeable contact lenses are effective in myopia control by flattening the cornea, but the myopia control effect was present only while contact lens wear continued.

In a study conducted in Singapore, Katz et al.¹³⁰ did not find a reduction in myopia progression with rigid gas permeable contact lenses. In this study, 97 children 6 to 12 years old wore contact lenses, and 188 wore spectacles for 2 years. The contact lens wearers had a mean -1.33 D increase in myopia compared to a mean -1.28 D increase in spectacle lens wearers. Children in the contact lens group wore the lenses a median of 7 hours per day. Twenty-one children who wore contact lenses 12 or more hours per day increased in myopia an average of -1.09 D, not significantly different from the -1.30 D average for the 176 children who wore spectacles for 12 or more hours per day. Keratometry powers decreased in both study groups, an average of -0.13 D in the contact lens wearers and -0.07 D in the spectacle lens wearers.

Myopia Control with Bifocal Spectacle Lenses

Bifocal lenses allow the patient to have one lens power for distance viewing and another lens power for near viewing. This is advantageous in certain accommodation and vergence disorders, such as accommodative insufficiency and convergence excess^{132,133} (see Chapter 21). It also appears to provide some degree of myopia control in some cases. The findings of studies on the effect of bifocals on childhood myopia progression are summarized in Tables 3-7 to 3-9.

Miles¹³⁴ presented data for myopic children seen in his ophthalmological practice. Forty-eight patients wore single-vision spectacle lenses and then switched to

TABLE 3-7 Mean Rates of Childhood Myopia Progression in Diopters per Year for Single-Vision and Bifocal Lens or Progressive Addition Lens (PAL) Correction

Study	SINGLE VISION		BIFOCAL OR PAL	
	n	Rate	n	Rate
Miles ¹³⁴	48	-0.75	48	-0.40
Roberts and Banford ^{95,96}	396	-0.41	85	-0.31
Oakley and Young ¹⁰²	298	-0.49	269	-0.03
Neetens and Evens ¹³⁵	733	-0.45	543	-0.30
Goss ⁹⁹	52	-0.44	60	-0.37
Grosvenor et al. ⁷¹	39	-0.34	85	-0.35
Pärssinen et al. ⁸⁸	79	-0.49	79	-0.54
Jensen ⁹⁰	49	-0.57	51	-0.48
Leung and Brown (PAL) ¹⁴³	32	-0.62	36	-0.36
Edwards et al. (PAL) ¹⁴⁴	133	-0.63	121	-0.56
Gwiadza et al. (PAL) ¹⁴⁵	234	-0.49	235	-0.43

TABLE 3-8 Methodologies and Results of Studies on the Effect of Bifocals on Childhood Myopia Progression

Study	Subjects	Type of Multifocal Lens	Summary and Interpretation of Results
Miles ¹³⁴	SV: 103, 6–14 yr old SV then BF: 48, 8–16 yr old St. Louis	28-mm wide flat-top segment bifocals, decentered for slight BI effect	Rates were less after switch to BF. Age is a potential confounding factor, but inspection of graphs suggests lower rates with BF over common age spans.
Roberts and Banford ^{95,96}	SV: 396 BF: 85 New York State; examined at least twice before age 17	Bifocals; most additions +0.75 to +1.50 D in power	Rates were significantly less in BF wearers than in SV group, the difference being greatest for patients with near-point esophoria and high accommodative convergence accommodation ratios.
Oakley and Young ¹⁰²	SV: 298 BF: 269 Oregon	Flat-top segments with top at pupil center, +1.50 to +2.00 D add	Reduction in rate with BF was larger than in any other study, which authors attributed to high placement of add or possible inadvertent investigator bias.
Neetens and Evens ¹³⁵	SV: 733 BF: 543 Patients who had myopia of <1.00 D at 8 or 9 yr of age Holland	Bifocals; total near-point power equal to zero for myopia up to 3.00 D; +2.50 D add for myopia ≥3.00 D	Mean amount of myopia at 18 yr of age was less for BF wearers (-3.55 D) than for SV wearers (-5.07 D).
Goss ⁹⁹	SV: 52 BF: 60 Children 6–15 yr of age Illinois, Iowa, and Oklahoma	Bifocals; various add powers, mostly +0.75 D and +1.00 D	Rate of progression was less with BF in patients with near-point esophoria. There was no difference for patients with orthophoria and exophoria.
Grosvenor et al. ⁷¹	SV: 39 +1.00 D BF: 41 +2.00 D BF: 44 6–15 yr of age at start of study Texas	Executive bifocals; top of reading segment 2 mm below pupil center	There was no significant difference in mean rates between SV, +1.00 D add BF, and +2.00 D add BF groups.
Pärssinen et al. ⁸⁸	240 children in three groups: full-time wear of SV lenses, SV lenses for distance use only, BF lenses Mean beginning age 10.9 yr in each group Finland	28-mm wide flat-top bifocals, top of reading segment 2 to 3 mm below pupil center; +1.75 D add	Mean amount of refractive change in BF group was not significantly different from that in either SV group.

continued

TABLE 3-8 Methodologies and Results of Studies on the Effect of Bifocals on Childhood Myopia Progression—cont'd

Study	Subjects	Type of Multifocal Lens	Summary and Interpretation of Results
Goss and Grosvenor ¹³⁸	SV: 32 BF: 65 Children 6–15 yr of age Reanalysis of Grosvenor et al.'s data	Executive bifocals; +1.00 D and +2.00 D adds	Difference between rates for BF- and SV-wearing esophores was similar to findings of Roberts and Banford and of Goss, but was not statistically significant because of small sample size.
Jensen ⁹⁰	SV: 49 BF: 51 Children in 2nd through 5th grades at start of study Denmark	35-mm wide segment bifocals; top of segment at lower pupil margin; +2.00 D add	Amount of refractive change was somewhat less with BF than with SV but was not statistically significant; for children with intraocular pressure ≥ 17 mmHg, change was less with BF than with SV.
Fulk and Cyert ¹⁴⁰	SV: 14 BF: 14 Boys 6–13.9 yrs, girls 6–12.9 yrs at start of study, all had esophoria at near Oklahoma	28-mm wide flat-top bifocals +1.25 D adds top of segment 1 mm above lower limbus	Reduction in rate with bifocals similar to that of esophoric subjects in other studies, but difference not statistically significant due to small sample size
Fulk et al. ¹⁴¹	SV: 39 BF: 36 Boys 6–12.9 yrs, girls 6–11.9 yrs at start of study, all had esophoria at near Oklahoma	28-mm wide flat-top bifocals +1.50 D adds top of segment 1 mm above lower limbus	Amount of progression in 30 months was significantly less in the bifocal group than in the single vision group when progression adjusted for age
Leung and Brown, ¹⁴³ Brown et al. ¹⁴²	SV: 32 PAL: 36 9–12 yrs at start of study Hong Kong	Progressive addition lenses: 22 subjects wore +1.50 D adds, 14 subjects wore +2.00 D adds	Progressives yielded statistically significant reductions in rate of progression; reduction in rate greater in cases of nearpoint esophoria
Edwards et al. ¹⁴⁴	SV: 133 PAL: 121 7–10.5 yrs at start of study Hong Kong	Progressive addition lenses with +1.50 D add	Reduction in rate with progressives not statistically significant; reduction in rate in cases of esophoria similar to that in other studies but not statistically significant because minority of subjects had esophoria
Gwiazda et al. ^{145,146}	SV: 234 PAL: 235 6–11 yrs at start of study 4 centers in United States	Progressive addition lenses with +2.00 D adds	Reduction in progression with progressives statistically significant; greatest reduction in rate in subjects with nearpoint esophoria and higher lags of accommodation

Adapted from Goss DA. 1994. Effect of spectacle correction on the progression of myopia in children—a literature review. J Am Optom Assoc 65:124–125.

SV, Single-vision lens wearers; BF, bifocal lens wearers; PAL, progressive addition lens wearers; BI, base in.

TABLE 3-9 Mean Rates of Childhood Myopia Progression in Diopters per Year for Single-Vision and Bifocal Lens or Progressive Addition Lens (PAL) Correction for Children with Esophoria at Near

Study	n	SINGLE VISION		BIFOCAL OR PAL	
		Rate	n	Rate	n
Roberts and Banford ⁹⁶	167	-0.48	65	-0.28	
Goss and Grosvenor ¹³⁸	7	-0.51	18	-0.31	
Goss and Uyesugi ¹³⁷	52	-0.59	66	-0.33	
Fulk and Cyert ¹⁴⁰	14	-0.57	14	-0.39	
Fulk et al. ¹⁴¹	39	-0.50	36	-0.40	
Edwards et al. (PAL) ¹⁴⁴	21	-0.63	21	-0.45	
Brown et al. (PAL) ¹⁴²	14	-0.65	16	-0.29	
Gwiazda et al., higher lag (PAL) ¹⁴⁶	34	-0.57	42	-0.36	
Gwiazda et al., lower lag (PAL) ¹⁴⁶	55	-0.38	55	-0.41	

bifocal spectacle lenses. Miles reported that myopia increased at a rate of -0.75 D while the patients wore single-vision lenses over age spans of 6 to 14 years. While the patients wore bifocals over age spans of 8 to 16 years, myopia progressed an average of -0.40 D per year. The difference in rates may have been due in part to the older ages when the bifocals were worn. However, inspection of Miles's composite graphs of myopia progression suggests that over common age spans, myopia progression was less with bifocals than with single-vision lenses.

Roberts and Banford^{95,96} reported on the results of bifocal control of myopia in their optometry practices in New York State. Included were myopic patients refracted in their practices at least twice before 17 years of age. Three hundred ninety-six patients (231 girls and 165 boys) wore single-vision lenses over the entire observation period, and 85 patients (47 girls and 38 boys) wore bifocals over the entire observation period. Most bifocal add powers were +0.75 to +1.50 D. The mean refractive error for the two eyes from the manifest subjective refraction spherical equivalents was used for analysis. To remove age as a variable in the rates of progression, Roberts and Banford adjusted rates using a formula derived from correlation analysis for the relationship of rate and age. The mean rate of progression for the bifocal wearers was -0.31 D per year, and that for the single-vision lens wearers was -0.41 D per year, a statistically significant ($p < .02$) difference in rates.

Oakley and Young¹⁰² presented findings from Oakley's practice in central Oregon. The bifocal lenses usually consisted of a 0.50 D undercorrection for distance vision and a flat-top reading segment with +1.50 to +2.00 add. The lenses were fit so that the top of the bifocal segment was at the center of the pupil when the eyes were in the primary position of gaze. The mean rate of myopia progression in the right eyes of 226 white

patients who wore bifocals was -0.02 D per year, whereas the mean rate for 215 white single-vision lens wearers was -0.53 D per year. For American Indian patients, the mean rates were -0.10 D per year for 43 wearing bifocals and -0.38 D per year for 83 wearing single-vision lenses. Oakley and Young attributed the success of bifocal control of myopia in their study to the high placement of the bifocal segment, although they noted that inadvertent examiner bias may have also affected the results. They stated that "virtually all" of the bifocal subjects and most of the single-vision lens subjects had near-point esophoria. Although near-point esophoria is common among myopic children, such a high prevalence seems unusual.

Neetens and Evens¹³⁵ presented the results they obtained with a large group of myopic patients they treated between 1959 and 1982. Patients were first seen at the age of 8 or 9 years with complaints of blurred distance vision. Bifocal power was such that the total near-point power was zero for myopia up to -3.00 D. The bifocal add power was +2.50 D for myopia of more than -3.00 D. At 18 years of age, the mean refractive errors were -5.07 D for 733 single-vision lens wearers and -3.55 D for 543 bifocal lens wearers. Assuming a beginning amount of myopia of -0.50 DS, the yearly rates of myopia progression were -0.30 D for the bifocal group and -0.45 D for the single-vision lens group.

Goss⁹⁶ collected data from three optometry practices in Illinois, Iowa, and Oklahoma in which bifocal lenses were used almost as often as single-vision lenses for myopic children. Selection criteria were as follows: (1) four or more refractions performed between the ages of 6 and 15 years, (2) myopia of at least -0.50 D, (3) astigmatism never manifested in excess of 2.50 DC, (4) no strabismus or amblyopia, (5) no contact lens wear before the last refractive data recorded for use in analysis, (6) no ocular disease, and (7) no systemic disease

that might affect ocular findings. In almost all of the bifocal wearers, the reading add power was +0.75 to +1.25 D. Rates of progression were calculated by linear regression analysis using the principal meridian nearest horizontal from the manifest subjective refraction for points between 6 and 15 years of age. For 52 patients who wore single-vision lenses, the mean rate of progression was -0.44 D per year ($SD = 0.26$). The mean rate of progression in 60 bifocal lens wearers was -0.37 D per year ($SD = 0.24$). The difference in rates was not statistically significant.

A prospective study was conducted with single-vision lens, +1.00 D add bifocal, and +2.00 D add bifocal groups at the University of Houston.^{7,136} Subjects were 6 to 15 years of age at the beginning of the study and had to have a spherical equivalent refractive error of -0.25 D or more, normal visual acuity, normal binocular vision, normal ocular health, and no contact lens wear. The bifocals were Executive bifocals with the top of the reading segment placed 2 mm below the center of the pupil. Of the 207 subjects who started the study, 124 (58 males and 66 females) completed the full 3 years. Rates of myopia were calculated by dividing the difference in the right-eye spherical equivalents of the manifest subjective refractions at the first and last study examinations by 3 years. The mean yearly rates of myopia progression were -0.34 D for 39 single-vision lens wearers, -0.36 D for 41 subjects who wore +1.00 D add bifocals, and -0.34 D for 44 subjects in the +2.00 D add bifocal group. The difference in rates between groups was not statistically significant.

Pärssinen et al.⁸⁸ conducted a study in Finland in which they placed 121 girls and 119 boys in one of three treatment groups: (1) full-time wear of single-vision lenses with a full correction of myopia, (2) single-vision lenses with full correction of myopia worn only for distance vision, and (3) bifocal lenses with a +1.75 D reading addition in a 28 mm wide flat-top segment with the top of the segment 2 to 3 mm below the center of the pupil. Subjects had no history of spectacle lens or contact lens wear, no ocular disease, no serious systemic disease, and spherical equivalent refractive errors of -0.25 to -3.00 D. There were also limitations on the amount of anisometropia, astigmatism, lateral phoria, and vertical phoria subjects could have. The mean age of each of the treatment groups at the beginning of the study was 10.9 years. Refractive data used for analysis were subjective refractions after cyclopentolate cycloplegia. The length of time subjects remained in the study varied but was 3.0 to 3.1 years for 95% of the subjects. There were 79 subjects in each of the three treatment groups at the end of the study. Compliance with instructions on spectacle wear was more than 75% in each of the three treatment groups, according to subjects' self-reports. The mean right-eye refractive error changes from the beginning to the end of the study were

-1.76 D ($SD = 1.0$) in subjects who wore single-vision lenses for distance vision only, -1.48 D ($SD = 0.9$) in those who wore single-vision lenses continuously, and -1.67 D ($SD = 0.9$) in the bifocal group. For the left eye, the mean changes in refractive error were -1.88 D ($SD = 1.0$) in subjects who wore single-vision lenses for distance vision only, -1.46 D ($SD = 0.9$) in subjects who wore single-vision lenses continuously, and -1.58 D ($SD = 0.9$) in the bifocal group. The differences in the amounts of change between the bifocal group and the group who wore single-vision lenses continuously were not statistically significant. Taking the average of the amount of change for the two eyes and dividing by 3 years yields myopia progression rates of -0.49 D per year for the continuous-use, single-vision lens group and -0.54 D per year for the bifocal group.

In Jensen's study,⁹⁹ children who wore +2.00 D add bifocals with a 35-mm wide segment having its top at the lower edge of the pupil were compared with a control group who wore single-vision lenses. The mean change in 2 years in the right-eye spherical equivalent cycloplegic autorefraction in 51 children wearing bifocals was -0.95 D ($SD = 0.56$). In 49 control subjects, the mean progression in 2 years was -1.14 D ($SD = 0.71$). The difference in means was not statistically significant. An interesting finding was that subjects with intraocular pressure greater than 16 mmHg at the start of the study had significantly less myopia progression in 2 years with bifocals than with single-vision lenses. The 24 subjects in the bifocal group with intraocular pressure greater than 16 mmHg had a mean myopia progression of -0.97 D ($SD = 0.64$) in 2 years. For 27 members of the control group, the mean progression was -1.32 D ($SD = 0.70$).

Comparison of the myopia progression rates with bifocals with the rates with single-vision lenses in Table 3-7 shows that most studies have found lower rates with bifocals but that study results have varied widely. Differences in outcomes might be explained by differences in study populations, inclusion criteria, bifocal design, proper use of the bifocals, or unknown factors. A summary of the design and interpretation of the various studies is given in Table 3-8. Another aspect of the efficacy of bifocal control to consider is whether it is more likely in some types of cases than in others. Bifocals appear to be effective in lowering myopia progression rates in children with esophoria at near.

The studies conducted by Roberts and Banford^{95,96} was based on their New York State private practice records. Phorias were measured through the subjective refraction using the von Graefe method (see Chapter 21) with a test distance of 40 cm. One hundred eighty-one patients in the single-vision lens group who had orthophoria or exophoria at near had a mean myopia progression rate of -0.41 D per year. Seventeen patients who wore bifocals and had orthophoria or exophoria at

near had a mean progression rate of -0.38 D per year. Among patients with esophoria at near, the mean yearly rates were -0.48 D for 167 single-vision lens wearers and -0.28 D for 65 patients wearing bifocals.

In his retrospective study based on private practice records, Goss⁹⁹ also studied near phorias at 40 cm with the von Graefe technique. Patients were divided into those with near phorias within Morgan's⁹⁷ normal range of orthophoria to 6^{Δ} exophoria and those with phorias on either side of the normal range. Among those with phorias greater than 6^{Δ} exophoria, the mean rates were -0.47 D per year ($n = 9$, SD = 0.31) for those wearing single-vision lenses and -0.48 D per year ($n = 3$, SD = 0.22) for those wearing bifocal lenses. The mean yearly rate for those who had normal phoria status and wore single-vision lenses was -0.43 D ($n = 27$, SD = 0.21). Bifocal wearers with normal phorias had a mean rate of -0.45 D per year ($n = 18$, SD = 0.27). Among patients with esophoria at near, the mean yearly rates were -0.54 D ($n = 10$, SD = 0.30) for the single-vision lens group and -0.32 D per year ($n = 35$, SD = 0.20) for the bifocal group. The difference in rates for esophoric patients was statistically significant at the 0.05 level. In this study and in the studies of Roberts and Banford,^{95,96} the myopia of esophores who wore bifocals progressed 0.20 D per year less than that of esophores who wore single-vision lenses. Goss also reported that the rate of progression was less with bifocals when the near-point binocular cross cylinder test was higher in plus. The lower rate with bifocals in children with higher plus on the binocular cross cylinder may be secondary to the association of higher binocular cross cylinder plus with esophoria.¹³⁷

In their myopia control study in Houston, Grosvenor et al.⁷¹ found that phoria did not affect whether bifocals reduced myopia progression. However, this may be accounted for by methodological differences between their study and Goss's study.⁹⁹ For example, Grosvenor et al. included subjects who were ages 6 to 15 years at the beginning of the study; at the end of the study, some of the subjects were almost 18 years old, when myopia progression has usually slowed. Goss's calculation of rates included only refractions between 6 and 15 years of age. Grosvenor et al. included subjects if their spherical equivalent refractive errors were -0.25 D or more, without considering whether subjects might have mixed astigmatism. Grosvenor et al. had some mixed astigmats among their subjects, whereas the study by Goss did not. Mixed astigmats have refractive changes that are generally less than those of myopes.⁹³

Goss and Grosvenor¹³⁸ reanalyzed the data of Grosvenor et al.⁷¹ using the findings from examinations between 6 and 15 years of age after omitting the results from the mixed astigmats. Rates of myopia progression were calculated by linear regression. Among subjects who had more than 6^{Δ} exophoria on the von Graefe

phoria with a 40-cm test distance, rates of progression averaged -0.50 D per year (SD = 0.26) for five subjects who wore single-vision lenses and -0.43 D per year (SD = 0.23) for six subjects who wore bifocals. When a normal phoria was observed, mean yearly rates were -0.43 D (SD = 0.32) for 20 subjects wearing single-vision lenses and -0.42 D (SD = 0.27) for 41 subjects wearing bifocals. When esophoria at near was observed, the mean yearly rates were -0.51 D (SD = 0.22) for 7 persons wearing single-vision lenses and -0.31 D per year (SD = 0.31) for 18 bifocal wearers. These results are similar to the results from Roberts and Banford^{95,96} and Goss,⁹⁹ as can be seen in Table 3-9. In all three studies, esophores had about 0.20 D per year less myopia progression with bifocals than with single-vision lenses.

Subjects in Jensen's study¹¹⁰ had phoria measurements taken by cover test prism neutralization with a target at 30 cm. Most subjects had exophoria. For 31 exophoric children who wore single-vision lenses, the mean myopia progression in 2 years was -1.11 D (SD = 0.79), compared with -0.88 D (SD = 0.64) for 28 exophoric children who wore bifocals. Among subjects with orthophoria, the mean changes in 2 years were -1.05 D (SD = 0.64) for 10 children in the single-vision lens group and -0.90 D (SD = 0.44) for 13 children in the bifocal group. Among subjects observed to have esophoria on the cover test, the mean 2-year refractive changes were -1.38 D (SD = 0.45) for eight single-vision lens wearers and -1.23 D (SD = 0.40) for 10 bifocal lens wearers. Myopia progression was somewhat less with bifocals than with single-vision lenses in each of the phoria groups, but the differences were not statistically significant. There was also a little more progression among esophoric subjects than among subjects with orthophoria or exophoria, but the differences were not statistically significant.

Jensen¹¹⁰ did not find as much reduction in myopia progression with bifocals in esophoric subjects as did Roberts and Banford,^{95,96} Goss,⁹⁹ and Goss and Grosvenor.¹³⁸ In the latter studies, phorias were measured using the von Graefe prism dissociation method with a target at 40 cm. Jensen measured phorias using cover test prism neutralization with a 30-cm test distance. Besides the difference in test distance, there are some differences that may be found between results from the von Graefe test and the cover test. Jensen did not indicate what type of fixation target she used for the cover test. If a letter target is not used on the cover test, as it is on the von Graefe test, accommodation, and therefore accommodative convergence, will be less, causing the phoria to be more in the exo direction. Some small phorias may not be observed during the cover test; for example, von Noorden¹³⁹ suggested that the minimum discernible movement on the cover test is 2^{Δ} to 4^{Δ} . In addition, the presence of the Phoropter on the von Graefe test may induce a small amount of

proximal convergence. Therefore, it seems likely that some of the subjects whom Jensen placed in the orthophoria category would have been esophoric on the von Graefe phoria test. This presumption is supported by the fact that Jensen's sample had the lowest percentage of esophoria of the four study samples (Roberts and Banford: 232 out of 430 subjects, or 54%; Goss: 45 out of 102 subjects, or 44%; Goss and Grosvenor: 25 out of 97 subjects, or 26%; Jensen: 25 out of 145 subjects, or 17%).

Two prospective studies conducted in Oklahoma^{140,141} support reduction in childhood myopia progression when near-point esophoria is present. In the first study, 14 children in single vision lenses progressed at a rate of -0.57 D per year compared to a rate of -0.39 D per year in 14 children wearing bifocals. In the second study, 39 children wore single-vision spectacles and 36 children wore bifocals with +1.50 D adds for the full 30-month follow-up period. Mean rates of progression were -0.50 D per year for the single-vision lens wearers and -0.40 D per year for the bifocal lens wearers. Amounts of myopia progression were significantly different in the two groups when adjusted for age ($p = .0046$).

In summary, the results of the bifocal myopia control studies have varied from sizable reduction in progression rate to no effect. Some studies have indicated that myopia control with bifocals is more likely when certain findings are present, including esophoria at near^{95,96,99,140,141} and higher intraocular pressure.¹¹⁰ None of the studies have suggested that myopia progression can be stopped with bifocals. However, if the myopia progression rate can be lowered 0.20 D per year, the amount of myopia reached at the cessation of childhood myopia progression will be about 1.00 D less if myopia progression occurred over a 5-year period, or as much as 2.00 D if the onset of myopia was 10 years before cessation of progression. The powers of the bifocal plus additions varied from one study to another, but the fact that myopia progression rates were lowest when the near phoria was ortho to low exo^{95,96,138} suggests that the power of the plus add should be such that the near phoria is shifted to the ortho to low exo range.

Myopia Control with Progressive-addition Spectacle Lenses

The finding of reduction of rates of myopia progression with plus adds by bifocals in children with near point esophoria is a trend observed in studies with progressive-addition lenses (PALs). In one study conducted in Hong Kong,^{142,143} 32 children wore single-vision lenses, 22 wore PALS with +1.50 D reading adds, and 14 wore PAL with +2.00 D adds. Subjects were between 9 and 12 years old at the beginning of the study. The mean amounts of myopia progression in 2 years were -1.23 D in the single-vision lens group, 0.76 D in those

who wore +1.50 D adds, and -0.66 D in the subjects who wore +2.00 D adds.¹⁴³ Differences in the amount of myopia progression were statistically significant. Brown et al.¹⁴² reported on the results of that study with subjects divided by 40-cm dissociated phoria findings from the Maddox wing test. The amounts of progression expressed as rates for subjects with esophoria were 0.65 D per year ($n = 14$) for subjects who wore single-vision lenses and 0.29 D per year ($n = 16$) for those who wore PAL. In subjects with orthophoria or exophoria at near, the mean rates were -0.59 D per year ($n = 18$) in single vision lens wearers and -0.42 D per year ($n = 20$) in PAL wearers. The sample size was too small to yield a statistically significant interaction between lens type and phoria grouping.

Another study conducted in Hong Kong¹⁴⁴ compared single-vision lens wearers to subjects wearing PAL with +1.50 D reading adds. Subjects were 7 to 10.5 years old at the start of the study and were followed for 2 years. The mean amount of progression in 121 subjects who were retained in the single-vision lens group for 2 years was -1.26 D ($SD = 0.74$). Progression over 2 years in 133 subjects wearing PAL averaged -1.12 D ($SD = 0.67$). There were 21 subjects in each group who were found to have esophoria at 33 cm with the Howell phoria card. Those with esophoria who wore single-vision lenses had a mean progression of -1.26 D ($SD = 0.90$) in 2 years. Subjects with esophoria who wore PAL averaged -0.89 D ($SD = 0.67$) change in myopia in 2 years.

A multicenter study of PAL for myopia control was conducted by Gwiazda et al.^{145,146} Subjects were 6 to 11 years of age at the beginning of the study and were followed for 3 years. Subjects in the PAL group had +2.00 D adds. The mean amounts of change in myopia in 3 years were -1.28 D ($n = 235$; $SE = 0.06$) in the PAL group and -1.48 D ($n = 234$; $SE = 0.06$) in the single-vision lens group. The difference in the means was statistically significant ($p = .004$). The greatest reduction in rate was found for subjects with esophoria at 33 cm by cover test and a higher lag of accommodation at 33 cm as determined by Canon R-1 Autorefractor. For such subjects, the mean amounts of myopia progression in 3 years were -1.08 D ($n = 42$) in the PAL group and -1.72 D ($n = 34$) in the single-vision lens group.¹⁴⁶

Table 3-9 summarizes rates of myopia progression with multifocals and single-vision lenses in children with esophoria at near. These results can be compared to the results irrespective of phoria in Table 3-7. It may be noted that progression rates were usually about 0.2 D less per year with multifocals than with single vision lenses in esophoria.

Changes in Astigmatism

Hirsch's study²⁹ in Ojai, California, may be the only longitudinal study on changes in astigmatism in an unse-

lected sample of schoolchildren. Refractive error was determined by manifest retinoscopy performed twice a year for 8 years on 167 children. For each child, the 16 tests were divided into four groups of four tests each, and the child's average astigmatism in each grouping was determined. The average ages of the four groups were 6.5, 8.5, 10.5, and 12.5 years. At 6.5 years of age, 81% of the children had less than 0.25 DC of astigmatism. This had decreased to 72% by 12.5 years of age. The percentage of children who had 0.25 DC or more with-the-rule astigmatism remained fairly constant in the range of 10% to 14% in each of the age groups, with 4% to 6% having 0.75 DC of with-the-rule astigmatism. The percentage of children with 0.25 DC or more against-the-rule astigmatism increased from 3% at 6.5 years of age to 11% at 12.5 years of age. None of the 167 children had against-the-rule astigmatism of 1.25 DC or greater. In terms of individual rates of change in astigmatism, 58% of the children had changes in astigmatism of less than 0.02 DC per year. Eleven percent of the children changed in the with-the-rule direction between 0.03 and 0.07 DC per year, whereas 23% changed in the against-the-rule direction at those rates. Two percent had astigmatism change rates of 0.08 to 0.22 DC per year in the with-the-rule direction, and 5% had against-the-rule changes at those rates.

Goss and Shewey⁹³ studied the changes in astigmatism in myopic children using data collected from private optometry practices. Measures of astigmatism were the recordings for the manifest subjective refraction. Examinations between the ages of 6 and 15 years were used for analysis. Rates of astigmatism change were calculated by linear regression; with-the-rule astigmatism assigned a positive value and against-the-rule astigmatism a negative number. Against-the-rule astigmatism was more common in this group of myopes than in Hirsch's unselected sample.⁹³ The mean rate of astigmatism change for 165 children who had zero astigmatism at their initial examination was not significantly different from zero. Children who had with-the-rule astigmatism at their first examination had a mean astigmatism change rate of 0.06 DC per year in the with-the-rule direction ($n = 37$, SD = 0.11). Those who had against-the-rule astigmatism had a mean astigmatism change rate of 0.03 DC per year in the against-the-rule direction ($n = 73$, SD = 0.12).

Pärssinen⁹⁴ studied changes in astigmatism over a 3-year period in 238 children who had low myopia, had not previously worn spectacle lenses, and had astigmatism of 2.00 DC or less. The subjects were in the third to fifth grades and were a mean age of 10.9 years at the beginning of the study. Whereas at the beginning of the study 45% of the right eyes had no astigmatism and only 3% had 1.00 DC or more of astigmatism, at the end of 3 years 24% had no astigmatism and 14% had 1.00 DC or more of astigmatism. There were more

against-the-rule than with-the-rule astigmats in this group of myopic children, as there were in the study by Goss and Shewey⁹³; this is in contrast to Hirsch's unselected sample,²⁹ in which with-the-rule astigmatism was more common. Pärssinen found with-the-rule astigmatism in 10% of the right eyes at the beginning of the study and 18% of the right eyes at the end of the 3 years. Against-the-rule astigmatism was present in 33% of the subjects at the beginning of the study and 44% at the end of the study. Oblique astigmatism (principal meridians between 31 and 59 degrees and between 121 and 149 degrees) was uncommon, being present in less than 1% of the children at the beginning of the study and 2.5% at the end of the 3 years. The mean amount of astigmatism, irrespective of whether it was with or against the rule, increased from 0.26 DC to 0.45 DC over the 3 years, for a mean rate of increase of 0.06 DC per year.

The findings of these three studies suggest that, during the school-age years, astigmatism tends to increase a small amount. There are changes in both the with-the-rule and against-the-rule direction, although changes in the against-the-rule direction are a little more common. The greater prevalence of against-the-rule astigmatism in the two myopic samples than in Hirsch's unselected sample²⁹ is consistent with Hirsch's observation³¹ that 5- to 6-year-old children who have against-the-rule astigmatism are more likely to become myopic than are children who have with-the-rule astigmatism.

REFRACTIVE CHANGES IN YOUNG ADULTHOOD

General Trends

In this section, refraction changes from the late teens or early 20s to about 40 years of age are discussed. For most persons, the young adulthood years are a time of relatively stable refraction. However, the onset or progression of myopia is not uncommon. Some people also experience a small shift in the hyperopic direction.

Brown^{147,148} calculated the mean annual changes in refraction for clinic and private practice patients in Chicago. Refractions were performed by retinoscopy under atropine cycloplegia. The mean annual change in the 20- to 34-year-old age span was -0.05 D per year, based on 2971 computations.¹⁴⁸ The mean annual change in the 35- to 43-year-old age period was +0.03 D per year, based on 597 computations. Slataper¹⁴⁹ reported similar data based on cycloplegic refractions performed in his private practice in Houston. Calculating the mean spherical equivalent refractive error for each age, he determined that the mean refractive error was plus (hyperopic) each year from 20 to 40 years of age, with the amount of plus decreasing from 20 to 31

and increasing from 31 to 40 years. From 20 to 40 years, the magnitudes of all but one of the yearly changes in the mean refractive error were less than 0.10 D (the exception being a change of -0.12 D from 20 to 21 years of age), and most of the differences from 1 year to another were less than 0.04 D.

Hofstetter³⁴ collected manifest subjective refraction data from the files of an optometrist in Bloomington, Indiana. Analysis of refractive change in individual patients was based on the mean spherical equivalent refractive error of both eyes. Data were presented as scatterplots of refractive change in diopters per month versus the refractive error at the first of the two examinations used for calculation of rate. The vast majority of patients in the 21- to 34-year-old age range had refractive change rates of zero or very close to zero. When myopes had refractive changes, they were usually in the myopic direction. The most negative change was not quite -0.03 D per month, perhaps about -0.35 D per year. The refractive change rates for the myopes in the 21- to 34-year-old age range were lower in magnitude than the rates for the myopes in the 10- to 20-year-old age range. When hyperopes showed refractive changes, they were most often in the hyperopic direction. The largest change in the hyperopic direction was between +0.02 and +0.03 D per month, the annual change rate being about +0.30 D per year. It is possible that some of the hyperopic shifts were due to more manifestation of formerly latent hyperopia.

Because the samples in the preceding studies were based on clinical populations, it may be presumed that they contained more persons with vision problems and refractive changes than would a nonselected sample. Morgan¹⁵⁰ studied refractive error changes between 13 and 33 years of age in 51 females and 44 males who were not visually selected and who were initially examined as part of a study on the growth and development of children. The mean spherical equivalent refractive error for the females was +0.09 D at age 13 years and -0.13 D at age 33. The mean refractive error for males was +0.39 D at age 13 and +0.35 D at age 33. Forty-four (46%) of the 95 subjects had changes of less than \pm 0.75 D over the 20-year period, 28 (29%) changed -0.75 D or more in the myopic direction, and 23 (24%) changed +0.75 D or more in the hyperopic direction. Among the females, the mean horizontal meridian refractive error was 0.12 D more plus than the vertical at age 13 and 0.24 D less minus than the vertical at age 33, combining for a with-the-rule astigmatism shift of 0.12 DC in 20 years. In the males, the vertical meridian was 0.05 D more plus than the horizontal at age 13 and 0.08 D more plus than the horizontal at 33 years, for an against-the-rule shift of 0.03 DC in 20 years.

Grosvenor¹⁵¹ published a questionnaire in *Optometric Weekly* requesting optometrists to report their own spectacle corrections at 5-year intervals, and the results were

published in subsequent issues of the journal.¹⁵¹⁻¹⁵⁴ A total of 111 questionnaires that contained complete data for ages 20 to 40 years were returned from respondents. One respondent was female, 109 were male, and one person did not indicate gender on the survey form. Using the most plus or least minus meridian of the right eye, Grosvenor determined that the mean refractive error shifted 0.10 DS in the myopic direction over the 20-year period, going from -0.08 DS at age 20 to -0.18 DS at age 40. The standard deviation increased from 1.47 D at 20 years to 1.92 DS at 40 years. Some of the survey respondents became myopic between 20 and 40 years of age. There was an increase in the number of persons who were myopic (correction of -0.50 D or more) and a decrease in the number of persons near emmetropia (-0.25 to +1.25 D). Sixty-five (59%) of the respondents had \pm 0.50 D or less change in refractive correction from 20 to 40 years of age. Individuals with myopia at age 20 often became more myopic, but the myopia increase was usually less than 1.00 D. The largest myopia increase was -2.00 D. Individuals with +1.00 D or more hyperopia at age 20 tended to become hyperopic. The largest increase in plus prescription was +1.50 D.

Of the 111 respondents in Grosvenor's study,¹⁵⁵ 65 (59%) had no astigmatism at 20 years of age, 29 (26%) had a with-the-rule correction, and 17 (15%) had an against-the-rule correction. At 40 years of age, 42 (38%) had no astigmatism, 44 (40%) had with-the-rule cylinder, and 20 (18%) had against-the-rule cylinder. At both 20 and 40 years of age, there were 5 respondents with oblique astigmatism. The change in astigmatism was zero in 61 (55%) persons, in the with-the-rule direction in 31 (28%) persons, in the against-the-rule direction in 18 (16%) persons, and toward oblique astigmatism in 1 person. The mean change in astigmatism over the 20-year period from 20 to 40 years of age was 0.10 DC in the direction of with-the-rule astigmatism.

Anstice¹⁵⁶ studied changes in astigmatism using the records of 621 patients from the files of an optometrist and a university optometry clinic in New Zealand. Mean amounts of astigmatism were calculated as a function of age, with with-the-rule astigmatism given a plus sign and against-the-rule astigmatism a minus sign. At 15 to 19 years of age, the mean astigmatism was zero ($n = 54$); at 20 to 24 years, it was +0.05 DC ($n = 33$); at 25 to 29 years, it was +0.27 DC ($n = 33$); at 30 to 34 years, it was +0.15 DC ($n = 26$); and at 35 to 39 years, it was +0.32 DC ($n = 34$). Thus there was a slight shift in the with-the-rule direction. The number of persons who had with-the-rule astigmatism increased over this age span.

Myopia Onset and Progression

The onset of myopia in the 20- to 40-year-old age range is called early adult-onset myopia in Grosvenor's¹

classification of myopia. By synthesizing the results of several studies, Grosvenor estimated that the prevalence of myopia of 0.50 DS or more increased from about 20% at 20 years to about 30% at 40 years of age. Grosvenor's use of 20 years as the start of the age range for early adult-onset myopia is somewhat arbitrary; age of physical maturity is more likely a better distinction between the youth-onset and early adult-onset myopias. Both youth-onset myopia and early adult-onset myopia can progress in young adulthood. The rate of early adult-onset myopia progression is usually less than the rate of youth-onset progression.

Comparing childhood myopia progression and young adulthood myopia progression, Goss et al.¹⁵⁷ identified three basic patterns of the change in refractive error with age. They called these patterns *adult stabilization*, *adult continuation*, and *adult acceleration*. In adult stabilization (Figure 3-12), childhood myopia progression is followed by stabilization of refractive error in young adulthood. Adult continuation (Figure 3-13) is characterized by childhood myopia progression followed by a generally slower progression of myopia in young adulthood. In adult acceleration (Figure 3-14), refractive change in the myopic direction accelerates in

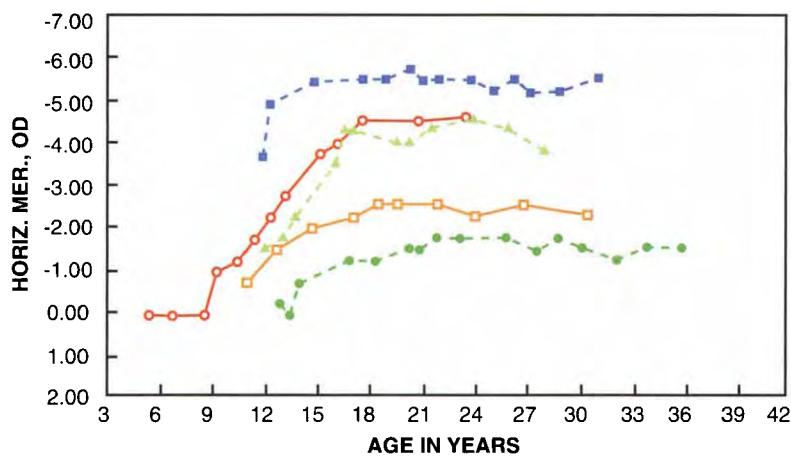


Figure 3-12

Examples of adult stabilization of myopia progression. Note that there is little or no increase in myopia in young adulthood after childhood myopia progression. HORIZ. MER., OD, refractive error in the horizontal meridian of the right eye. (From Goss DA, Erickson P, Cox VD. 1985. Prevalence and pattern of adult myopia progression in a general optometric practice population. Am J Optom Physiol Opt 62:472.)

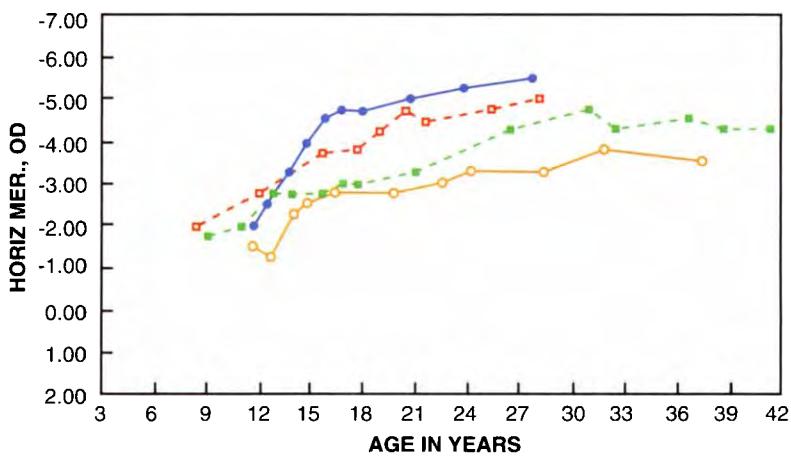
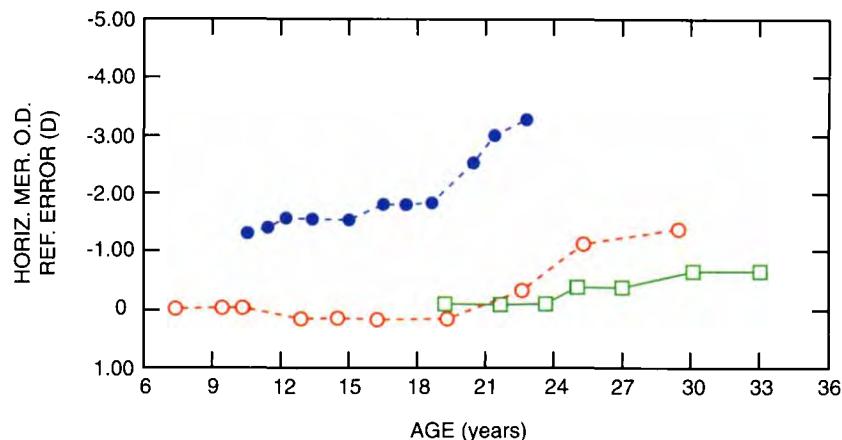


Figure 3-13

Examples of adult continuation, in which the rate of young adulthood myopia progression is less than the rate of childhood myopia progression. HORIZ. MER., OD, horizontal meridian of the right eye. (From Goss DA, Erickson P, Cox VD. 1985. Prevalence and pattern of adult myopia progression in a general optometric practice population. Am J Optom Physiol Opt 62:472.)

**Figure 3-14**

Examples of adult acceleration, in which either the rate of young adulthood myopia progression is greater than the rate of childhood myopia progression or myopia has its onset in early adulthood. HORIZ. MER. O.D., horizontal meridian of the right eye; REF., Refractive.

young adulthood. If emmetropia or hyperopia is present when the accelerated refractive change occurs and myopia develops, the patient would fit into Grosvenor's¹ early adult-onset myopia classification.

Using data from five optometry practices in the Midwest, Goss et al.¹⁵⁷ subjectively classified patients with a sufficient number of data points into one of three categories. The majority of cases—68% of the males and 87% of the females—fit into the adult stabilization category. Adult continuation was found in 25% of the males and 13% of the females. The least common pattern was adult acceleration, which was found in 6% of the males and none of the females. Among males who had three or more refractions after the age of 18 years, the mean rate of refractive change based on linear regression was -0.07 D per year. For females, the mean rate was -0.03 D per year. When only patients in the adult continuation and adult acceleration categories were considered, most young adulthood myopia progression rates were between -0.05 and -0.20 D per year. These rates are less in magnitude than the rates of childhood myopia progression.

The rates of young adulthood myopia progression reported by Goss et al.¹⁵⁷ are similar to those found by Kent,¹⁵⁸ who presented case reports of myopia onset after 18 years of age. Five of the patients were followed for periods of time varying from 9 to 26 years. The rates of progression for these five patients varied from -0.04 to -0.16 D per year. The amount of myopia ultimately developed in early adult-onset myopia is usually fairly low. The highest amount of myopia developed (spherical equivalent) in Kent's five cases was -2.50 D.

Riffenburgh¹⁵⁹ presented nine case studies of myopia onset after the age of 20, taken from a private practice. All nine persons, who included graduate students, telephone operators, accountants, and bookkeepers, were

involved in near-work activities in some way. Several studies with particular academic groups suggest that onset and progression of myopia in young adulthood may be more likely in persons who do a lot of near work. The onset and progression of myopia have been reported in students in military academies,¹⁶⁰⁻¹⁶³ undergraduate students,¹⁶⁴ and graduate students and law students.^{165,166} For example, Dunphy et al.¹⁶⁵ presented data on the changes in refractive error for 200 eyes of 110 graduate students in the Harvard Business and Law Schools. The students were 20- to 30-year-old males. The changes in spherical equivalent cycloplegic refraction in 1 year were $+0.50$ to $+0.25$ D in 29 eyes, $+0.12$ to -0.12 D in 89 eyes, -0.25 to -0.50 D in 73 eyes, and -0.62 to -0.87 D in 9 eyes. Excluding those with changes of $+0.12$ to -0.12 D, the hyperopes at the first examination were about equally split between those who had plus changes and those who had minus changes. Most of the myopes had increases in myopia, and all of those who had changes of -0.62 to -0.87 D were initially myopes.

Synthesizing the results from the above studies and unpublished studies in military academies and other academic settings, the National Academy of Sciences Working Group on Myopia Prevalence and Progression¹⁶⁷ and Baldwin et al.¹⁶⁸ reached the following conclusions:

1. In populations not containing a large number of persons in college, less than 10% of emmetropes and low hyperopes will develop myopia before 40 years of age. In contrast, as many as 20% to 40% of low hyperopes and emmetropes who enter colleges and military academies are likely to become myopic.
2. In young adults, the shift toward myopia experienced by low hyperopes and emmetropes is

generally less common and lower in amount than the increase in myopia experienced by persons already myopic.

3. It is unlikely that a person who enters college with a noncycloplegic refraction of +1.00 D or greater in either principal meridian will become myopic by the end of 4 years of study.
4. Persons with low hyperopia at 17 or 18 years of age appear to be more likely to become myopic in heavy near-work situations than are older persons with low hyperopia.

On the basis of a review of cross-sectional and longitudinal studies, Grosvenor¹⁶⁹ concluded that onset and progression of myopia in the early adulthood years are due to increases in vitreous depth, with an additional contribution of corneal steepening. Three studies compared ocular optical components of one-time measurements of small groups of early adult-onset myopes and emmetropes. McBrien and Millodot¹⁷⁰ found greater vitreous depth, greater anterior chamber depth, and less crystalline lens thickness in early adult-onset myopes than in emmetropes. Grosvenor and Scott¹⁷¹ found greater corneal power in early adult-onset myopes. Bullimore et al.¹⁷² reported greater vitreous depth in early adult-onset myopes than in emmetropes.

Longitudinal data have shown both vitreous depth increases and corneal power increases in young adults with onset and progression of myopia. Based on plots of changes in keratometer power and amount of myopia with age, Baldwin¹⁷³ suggested that adults with increases in myopia tend to have increases in corneal power. Keratometer data were available for one of five cases of early adult-onset myopia presented by Kent.¹⁵⁸ Increases in spherical equivalent keratometer power were 0.58 D in the right eye and 0.65 D in the left eye, corresponding to spherical equivalent refractive error changes of -0.94 D in the right eye and -0.75 D in the left eye.

In longitudinal records from optometry practice files, Goss et al.¹⁵⁷ found that all of 11 patients with adult continuation or adult acceleration of myopia progression had corneal steepening. Of 20 patients with adult stabilization of myopia progression, 11 had corneal flattening, eight had corneal steepening, and one experienced no change. Goss and Erickson¹⁷⁴ used the same set of data to calculate correlation coefficients of rate of refractive error change with rates of keratometer power between the ages of 18 and 40 years. Increases in myopia were associated with increases in corneal power. For the principal meridian nearest horizontal, the correlation coefficients were +0.29 ($p < .20$) for males and +0.58 ($p < .05$) for 15 females. The correlation coefficients for the principal meridian nearest vertical were +0.66 ($p < .001$) for the males and +0.69 ($p < .005$) for the females.

Adams¹⁷⁵ presented a case report of a higher than usual amount of early adult-onset myopia. Spherical

equivalent refractive errors went from -0.25 D OU at 19 years of age to -1.25 D OU at 24 years of age to -4.75 D OU at 42 years. From 24 to 42 years of age, keratometer powers increased 0.75 D (horizontal, OD), 0.25 D (vertical, OD), 1.00 D (horizontal, OS), and 0.37 D (vertical, OS). Because the amount of corneal power increase would not account entirely for the myopia progression and ocular axial lengths were 25.8 mm at 42 years of age, Adams suggested that this myopia progression was due primarily to axial length increase. Adams and McBrien¹⁷⁶ presented 2-year results of a longitudinal study in a group of clinical microscopists. Sixteen of those who were initially emmetropic developed myopia during the study. The mean amount of myopia developed was -0.64 D. Vitreous depth increased an average of 0.19 mm, and corneal steepening occurred, with a mean decrease in corneal radius of 0.05 mm; both of these changes were significantly different from zero.

Grosvenor and Scott¹⁷⁷ published data on ocular optical component changes in 16 persons with early adult-onset myopia. The change in 3 years of spherical equivalent refractive error correlated significantly with increase in vitreous depth ($r = -0.77$). The direction of the correlation between change in spherical equivalent refractive error and change in spherical equivalent autokeratometer power was in the direction of increased corneal power in association with increased myopia; however, it was not statistically significant ($r = -0.13$). Calculated crystalline lens power change did not correlate significantly with refractive error change.

To summarize the results of the longitudinal studies, the two studies with data on vitreous depth both found increases^{176,177} corneal steepening was observed by Baldwin,¹⁷³ Kent,¹⁵⁸ Goss et al.,¹⁵⁷ Goss and Erickson,¹⁷⁴ and Adams and McBrien,¹⁷⁶ but not by Grosvenor and Scott.¹⁷⁷ Although crystalline lens power was not determined directly by phakometry in any of the longitudinal studies, when Grosvenor and Scott calculated lens power based on the other components, they did not find a significant correlation between change in lens power and change in refractive error.

REFRACTIVE CHANGES FROM AGE 40 ON

Trend Toward Hyperopia

In cycloplegic refraction findings from his private practice in Houston, Slataper¹⁴⁸ observed that the mean spherical equivalent refractive error increased in plus from 40 years of age to the mid-60s, after which it decreased. The mean refractive error was +0.73 D at 40 years of age and +1.97 D at 64 years of age, a change of +1.24 D over the 24 years and a yearly rate of change of

+0.05 D. The mean subsequently changed from +1.72 D at 65 years of age to +1.21 D at age 74.

Hofstetter³⁴ investigated changes in manifest subjective refractions in an optometry practice in Bloomington, Indiana. Patients in the 36- to 68-year-old range had a mode refractive error change rate of zero. Plus changes in refractive error were much more common than minus changes, which were uncommon. For patients who had more than +2.00 D of hyperopia, the mode rate of change was about +0.06 D per year.

Hirsch¹⁷⁸ analyzed data from his practice in California from 460 women and 360 men aged 45 years or older. Refractive errors in the analysis were the means of the spherical equivalents of the two eyes from the manifest subjective refraction. The median refractive error for patients aged 45 to 49 years was +0.18 D. The median refractive error increased in hyperopia to +1.02 D at age 75 or older. Along with the shift in the direction toward hyperopia, there was an increase in variability of refractive error. The interquartile range increased from 1.00 D at ages 45 to 49 years to 2.27 D at age 75 years or older.

The prevalences of different levels of refractive error found by Hirsch¹⁷⁸ are given in Table 3-10. There was a decrease in the prevalence of refractive error within ± 1.12 D of emmetropia and an increase in the prevalence of hyperopia after the age of 45 years. Beginning in the 60s, there was also an increase in the prevalence of myopia. Hirsch pointed out that because almost everyone over 45 years of age needs optical correction for distance vision, near vision, or both, these prevalences may be fairly representative of a nonvisually selected population.

Grosvenor and Skeates¹⁷⁹ examined records of patients over the age of 45 years in an optometry practice in New Zealand. Out of 100 hyperopes, 36 changed less than ± 0.50 D per decade, 62 increased in hyperopia, and two had a myopic shift. Among 100 emmetropes, there were 43 who changed less than ± 0.50 D, 54 with a hyperopic shift, and three with a myopic shift. Out of

100 myopes, there were 66 who changed less than ± 0.50 D, 19 who moved toward hyperopia, and 15 who increased in myopia.

In summary, the overall trend in refractive changes after 45 years of age is a shift in the hyperopic direction, with some myopic persons showing increases in myopia. If age-related nuclear cataracts develop (usually after 60 years of age), there is often a shift toward myopia.

Trend Toward Against-the-Rule Astigmatism

Hirsch¹⁸⁰ studied the changes in subjectively determined astigmatism in his practice patients over the age of 40. Minus-cylinder axes within 20 degrees of 180 degrees were considered with-the-rule astigmatism, and those within 20 degrees of 90 degrees were considered against-the-rule astigmatism. Cases of oblique astigmatism and astigmatism in excess of 4.00 DC were omitted. With-the-rule astigmatism was given a positive sign and against-the-rule astigmatism a negative sign. Astigmatism gradually shifted in the against-the-rule direction, the mean going from +0.27 DC in the 40- to 44-year-old age group to -0.81 DC in the over-80 age group. In the same time span, the median astigmatism went from +0.09 DC to -0.91 DC. The change in both the mean and median in this 40-year period was about 1.00 DC, or about 0.25 DC per decade. The prevalence of with-the-rule astigmatism of more than 0.25 DC decreased from 29.0% in the 40- to 44-year-old age group to 6.8% in the over-80 group. Against-the-rule astigmatism of more than 0.25 DC was observed in 9.5% of patients 40 to 44 years old and in 65.1% of those over 80 years old. Against-the-rule astigmatism had become more common than with-the-rule astigmatism by 45 to 49 years of age (23% vs. 16% of patients).

Anstice¹⁵⁶ studied the changes in astigmatism in patients seen by an optometrist in New Zealand. With-

TABLE 3-10 Percentage of Patients Age 45 or Older with Various Refractive Error Levels

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Please refer to the printed publication.

the-rule astigmatism, defined as astigmatism with the minus cylinder axis within 30 degrees of horizontal, was given a plus sign. Against-the-rule astigmatism, defined as astigmatism with the minus cylinder axis within 30 degrees of vertical, was given a minus sign. The mean subjectively determined astigmatism was +0.08 DC for 40- to 44-year-olds, but this gradually shifted in the against-the-rule direction until it was -0.49 DC for 70- to 74-year-olds. With-the-rule astigmatism was more common than against-the-rule from the youngest age category (5 to 9 years) up to 55 to 59 years of age. After 60 years, against-the-rule astigmatism was more common.

Using corneal astigmatism data from keratometric measurements, Anstice¹⁵⁶ determined that corneal astigmatism also shifted in the against-the-rule direction. The mean corneal astigmatism went from +0.55 DC at 40 to 44 years of age to +0.09 DC at 70 to 74 years of age. Lyle¹⁸¹ likewise found a shift in corneal astigmatism toward against-the-rule in his optometry practice. Patients over 40 had experienced changes in corneal astigmatism in the preceding decade that ranged from 0.62 DC in the with-the-rule direction to 1.75 DC in the against-the-rule direction. There were more against-the-rule than with-the-rule changes, with the majority of points being in the range of about 0 to 0.62 DC against-the-rule. The prevalence of with-the-rule corneal astigmatism of 0.50 DC or more was 84% for 41- to 50-year-olds and 41% among those 61 years or older. The prevalence of against-the-rule corneal astigmatism of 0.50 DC or more was 2% at 41 to 50 years of age and 18% at 61 years or older.

Further evidence for the cornea as the source of the shift toward against-the-rule astigmatism comes from Baldwin and Mills,¹⁸² who collected data from an optometry practice that had been in existence for more than 60 years. Data were selected for patients who had had keratometric measurements taken periodically over at least 40 years, the first time at or before 30 years of age and the last time at 70 years or older. Eyes with refractive and corneal principal meridians within 10 degrees of horizontal and vertical were selected for study; 34 eyes fulfilled these criteria and were used for analysis. Discerning no trend of change in either corneal or refractive astigmatism before 40 years of age, Baldwin and Mills used the first examination after 40 years as the initial datum point for each patient. The mean vertical meridian refractive error increased in hyperopia by +0.82 D, and the horizontal meridian refractive error increased in hyperopia by +0.30 D, resulting in a 0.52 DC shift in the against-the-rule direction. The mean vertical meridian keratometer power increased 0.07 D, and the horizontal meridian keratometer power increased 0.38 D, representing a change in corneal astigmatism of 0.31 DC toward against-the-rule astigmatism.

ETIOLOGICAL CONSIDERATIONS

Emmetropization

Distributions of refractive errors in the general population are leptokurtic and skewed toward myopia^{183,184} (see Chapter 2). The peak of refractive error distributions occurs at emmetropia and low hyperopia. The term *emmetropization* is often used to describe the process theorized to explain why there are more people with emmetropia and near emmetropia than if variability in refractive error was due to random variation. It appears that emmetropization can be accounted for in part by coordinated growth of the eye and in part by some form of vision-dependent feedback system for ocular refractive development.

The growth of the eye is coordinated such that if the changes in the ocular optical components occurred by themselves they could result in large changes in refractive error,⁴⁸ but because they occur together there is much less change in refractive error. During childhood, vitreous depth increases, crystalline lens power and thickness decrease, and anterior chamber depth increases.* By itself, an increase in vitreous depth would cause a change toward myopia. By itself, a decrease in crystalline lens power would cause a change toward hyperopia. Because they occur simultaneously, the net change in refractive error is thus reduced. One study found a significant correlation of vitreous depth with posterior crystalline lens radius but not with anterior crystalline lens radius, suggesting that the posterior surface of the lens provides the emmetropization effect from the lens.⁶²

Evidence that there is a vision-dependent feedback system for refractive development comes from animal studies in which axial myopia can be induced by altering visual input.¹⁹²⁻²⁰⁴ Figure 3-15 illustrates the axial elongation observed by Raviola and Wiesel.²⁰⁵ In addition, when humans do not have normal ocular imagery, large refractive errors usually develop. Conditions such as lid hemangiomas, ptosis, neonatal eyelid closure, retroental fibroplasia associated with retinopathy of prematurity, and vitreous hemorrhage in infants and children lead to high myopia.^{22,23,205-207} Axial length was found to be greater than normal in patients with neonatal eyelid closure, juvenile corneal opacification, and congenital cataracts.^{206,209,210}

Distributions of corneal power, crystalline lens power, and anterior chamber depth are normal, but distributions of axial length have been found to be leptokurtic.²¹¹⁻²¹³ On this basis, it can be suggested that the visual feedback that directs ocular refractive development directs axial elongation of the eye.

*References 49, 54, 55, 60, 61, 185-191.

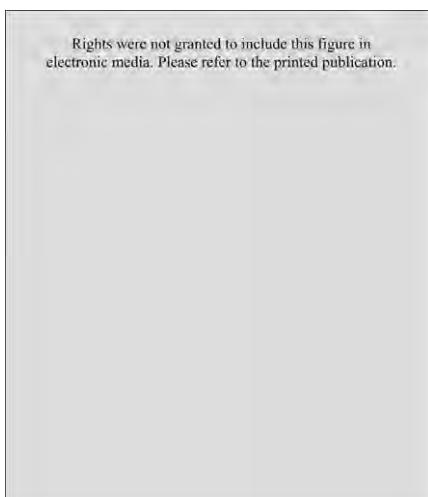


Figure 3-15

Laboratory studies with animals are showing that degraded or defocused retinal imagery induces myopia. This diagram illustrates axial myopia as a result of lid suture (to the right of the vertical line) as compared with the normal control eye (to the left of the vertical line). (From Raviola E, Wiesel TN. 1978. Effect of dark-rearing on experimental myopia in monkeys. *Invest Ophthalmol Vis Sci* 17:67.)

Because emmetropization appears to be a vision-dependent phenomenon, and because it tends to reduce either myopic or hyperopic refractive error, visual input must direct the visual system in sensing the presence and direction of refractive error. Because spectacles provide a compensation for refractive error, it could be asked whether they could disrupt or affect emmetropization in some way. The fact that bifocals and PALS slow myopia progression in some cases suggests that there is an effect. The influence of spectacle correction on emmetropization is significant in clinical management decisions in hyperopia and anisometropia in infants and small children. Because uncorrected hyperopia can be associated with esotropia and anisometropia is associated with amblyopia,²¹⁴ correction of hyperopia and anisometropia in infants and small children is generally advocated. Studies with monkeys have shown that anisometropia induces amblyopia, but conversely that experimentally induced amblyopia will also lead to anisometropia,²¹⁵ agreeing with human studies showing that refractive error changes are different in nonamblyopic eyes than in the amblyopic eyes of the same individuals.²¹⁶⁻²¹⁸ Understanding the nature of the relationship of amblyopia and refractive development will aid in understanding the emmetropization process.

Two studies have shown that spectacle undercorrection of hyperopia in infants has little or no effect on

emmetropization. Atkinson et al.²¹⁹ studied infants with hyperopia of at least +3.5 D in one meridian but with no meridian greater than +6.0 D. The children were followed from 9 months to 36 months of age. Thirty-seven infants did not receive spectacles. Forty-four infants received spectacles in which the sphere power was 1.0 D less than the refractive error in the least hyperopic meridian. The cylinder in the spectacles was half of any astigmatism over 2.5 D up to 2 years of age, and after that it was half of any amount of astigmatism. Infants with strabismus or anisometropia more than 1.5 D in parallel meridians were excluded from the study. The mean refractive error in the most hyperopic meridian decreased from +4.6 to 3.4 D in the spectacle wearers and from +4.3 to 3.1 D in those who did not wear spectacles. Thus, the reduction in hyperopia was the same in both groups. The mean amount of astigmatism decreased from 1.9 to 1.0 D in the spectacle wearers and from 1.7 to 0.7 D in those who did not wear spectacles. An additional analysis showed no difference in the results for subjects who were compliant with spectacle lens wear and those that were not.

Ingram et al.²²⁰ followed 6-month-old infants for variable times ranging from about 2 to 4 years. Included in the study were infants with more than +5.25 D hyperopia in one meridian by retinoscopy after instillation of cyclopentolate. Data were analyzed for three groups by treatment: (1) no spectacles, (2) spectacles with 2.0 D undercorrection of hyperopia in all meridians and later judged to have worn spectacles consistently, and (3) spectacles with 2.0 D undercorrection in all meridians, but later judged to not have worn spectacles consistently. The 89 nonstrabismic infants who did not wear spectacles had a mean decrease in hyperopia of 1.34 D (SE = 0.13). The 45 nonstrabismic infants who wore spectacles consistently had a mean decrease in hyperopia of 0.89 D (SE = 0.18). The 55 without strabismus who wore spectacles inconsistently had a mean decrease in hyperopia of 1.35 D (SE = 0.15). Thus, it appeared that inconsistent wear of undercorrected spectacles for hyperopia did not affect emmetropization in either study and that consistent wear did not affect emmetropization in one study but may have in the other.

Prevailing Theories of Myopia Development

Hypotheses of myopia etiology have been extensively reviewed and discussed.^{79,221-237} However, none of the numerous hypotheses proposed has been universally accepted. Hypotheses range from entirely genetic inheritance causation to emphasis on environmental and lifestyle factors. In reality, the etiology probably involves both genetic inheritance and vision activity, and perhaps other environmental factors.

Pedigree studies have identified modes of inheritance for some uncommon forms of high myopia.^{238,239} There is a resemblance of refractive errors in parents and offspring.²⁴⁰⁻²⁴³ Furthermore, a tendency toward myopia may be present in the children of myopic parents before they actually become myopic, as evidenced by the fact that they have greater vitreous depths.²⁴⁴ However, common lifestyle and environmental factors as well as genetic inheritance can contribute to family resemblance in refractive error. Statistical studies of the relative contribution of genetics to variability in refractive error have shown that refractive error cannot be attributed entirely to genetic inheritance causes and that, in some populations, apparently little of the variability in refractive error can be attributed to genetic inheritance.^{77,241,245-248}

Several theories of myopia etiology have attempted to identify mechanisms by which near work causes myopia development. One hypothesis is that sustained accommodation causes an increase in intraocular pressure, which in turn leads to a stretching of the posterior segment of the eye and axial elongation.^{87,249-251} This hypothesis appears to be at odds with the fact that intraocular pressure as measured at the cornea decreases with accommodation.²⁵²⁻²⁵⁵ Young²⁵⁶ proposed that an intraocular pressure gradient between the anterior and vitreous chambers occurs during accommodation.

On the basis of his engineering studies of the mechanical forces on the sclera generated by the extraocular muscles and accommodation, Greene^{257,258} concluded that although accommodation is unlikely to cause significant mechanical force on the sclera, the extraocular muscles might. He pointed out that the insertion of the oblique muscles posterior to the equator of the eye could produce localized tensile stress on the posterior sclera. Another possible stress on the sclera is intraocular pressure. Cocontracture of the extraocular muscles has been reported to increase intraocular pressure in animals.²⁵⁹⁻²⁶¹ Lateral gaze in humans has been observed to be associated with small increases in intraocular pressure.^{262,263} Greene²⁶⁴ concluded that it is plausible that stress on the posterior sclera from the combined effects of the extraocular muscles and intraocular pressure could contribute to axial elongation of the eye.

A recent hypothesis is that myopia results from a defocus of the ocular imagery so that the point of clearest focus would be significantly behind the retina, as would occur when a person who does a lot of near work has deficient accommodative function. One way to view the defocus hypothesis from a functional standpoint is that accommodation is a short-term mechanism for clear near-point vision and myopia is a long-term mechanism for clear near-point vision.²⁶⁵ As mentioned earlier, altered visual input can result in myopia: in

chickens, defocus of the retinal image with plus or minus lenses slows (plus) or accelerates (minus) the rate of posterior segment growth and thus induces either hyperopia or myopia, depending on the direction of the defocus.^{196,199,266,267} This effect is due not to the process of accommodation but to the defocus itself because the effect occurs regardless of whether the animals are capable of accommodation.

In another series of investigations demonstrating the chick's sensitivity to defocus, Troilo²⁶⁸ and Troilo and Wallman²⁶⁹ studied recovery from induced myopia and hyperopia. Hyperopia was induced in some animals by dark rearing, and myopia was induced in others by form-vision deprivation. The animals were returned to a normal vision environment by 2 to 4 weeks of age, at which point the hyperopic eyes manifested decreases in hyperopia because the rate of vitreous depth increase accelerated. The myopic eyes decreased in myopia because the vitreous depth stopped increasing, while the continued normal growth of the cornea and crystalline lens resulted in a decrease in refractive power.

The importance of ocular image focus to refractive development is also illustrated by the findings of Hodos and Erichsen²⁷⁰ that pigeons, quail, chickens, and cranes had myopia in the portion of the eye corresponding to the lower visual field, but not in the portion corresponding to the upper visual field. Furthermore, the amount of myopia was related to the distance from the eye to the ground. They interpreted these findings as indicating that the chick's eye develops in such a way as to allow the ground to be in focus for finding food, while having the horizon and sky also in focus so that predators can be seen.

In an experiment by Ni and Smith,²⁷¹ cats responded to defocus in either direction by developing myopia. Both plus and minus lenses resulted in myopia. The development of large refractive errors from the wearing of high (6.00 to 10.00 D) plus and minus lenses has been observed in monkeys, but the results have not shown a consistent direction of the effect, perhaps because of procedural difficulties or interruption of lens wear. Smith et al.^{272,273} and Smith²⁷⁴ found myopia in some animals as a result of minus lenses. Crewther et al.²⁷⁴ found high hyperopia in some animals regardless of whether they were treated with plus or minus lenses. In a study by Hung et al.,²⁷⁵ monkeys fitted with unilateral plus lenses tended to have the treated eye shift toward hyperopia, and monkeys wearing unilateral minus spectacle lenses tended to have myopia or less hyperopia in the treated eye.

Overall, the animal studies indicate that retinal imagery defocus affects refractive error development by adjusting the rate of vitreous depth increase. In chickens, a normally functional accommodative mechanism is not necessary for this feedback process to occur,

suggesting that it is the defocus itself, rather than a mechanical effect of accommodation, that affects refractive development. Pointing out the considerable diversity in ocular optics and morphology among vertebrates, Sivak²⁷⁶ recommended caution in generalizing the results of nonhuman animals to human ocular development. Nevertheless, he noted that the underlying molecular events leading to myopia development may be present in all species.

Various investigations are finding that in experimental animal models of myopia, retinal biochemistry is affected. For example, monkeys fitted with opaque contact lenses develop myopia and have decreased concentrations of dopamine in the retina.²⁷⁷ Image defocus affects the activity of retinal bipolar and amacrine cells in monkeys.²⁷⁸ Several experiments have demonstrated that some biochemical agents that affect the function of retinal synapses block form deprivation myopia, whereas others induce myopia.^{122,279-288} Other studies have shown that vitreous chamber enlargement in form deprivation myopia in chicks results from increased scleral growth or remodeling.²⁸⁹⁻²⁹⁵ It is unclear what exact cues may be used to recognize direction of defocus.²³⁵ Rates of axial elongation in chicks and monkeys appear to be modulated by direction of defocus.^{296,297} It has been reported that, for chicks, both direction of defocus and relative distance cues guide ocular development.²⁹⁸ Taken together, the animal studies show that the eye responds to the defocus of retinal imagery by a cascade of events that begins with changes in retinal synaptic function and results in increased vitreous depth by increased scleral growth or remodeling.

The application of these findings to human myopia may suggest a mechanism whereby an individual who does a lot of near work and whose accommodation lags more than a normal amount (with the result that the near-point object being viewed is out of focus and conjugate with a point behind the retina) experiences an increase in vitreous depth through ocular growth and develops myopia.²⁹⁹ Several studies have shown differences in accommodation and convergence between myopes and emmetropes.^{223,229,231,300-302} The dark-focus level of accommodation is less in myopes than in emmetropes.³⁰³⁻³⁰⁶ Accommodative response has been reported to be less in myopes than in emmetropes.³⁰⁷⁻³¹¹ A more convergent vergence posture and higher AC/A ratios are associated with myopia.³¹²⁻³¹⁵ The magnitude of the positive relative accommodation test has been found to be lower in emmetropic children who became myopic than in emmetropic children who remained emmetropic in three different clinical samples.³¹⁶⁻³¹⁹ There are also differences in dynamic function and adaptation processes in accommodation between myopes and nonmyopes.^{229,302,319-321} Mathematical models are being developed to describe possible rela-

tionships of accommodation and vergence function with refractive development.³²²⁻³²⁴

Etiology of Astigmatism

The etiology of most cases of astigmatism is unknown.³²⁵⁻³²⁷ One common hypothesis is that eyelid tension steepens the vertical corneal meridian and causes with-the-rule astigmatism. Most corneas have decreases in corneal with-the-rule astigmatism when the eyelids are lifted away from the eye.³²⁸ In addition, most eyes show an increase in refractive with-the-rule astigmatism as measured by autorefractor when the palpebral aperture is narrowed.³²⁹ Case reports indicate that chalazia and lid masses can induce changes in corneal astigmatism.³³¹⁻³³² However, in two studies a relationship was not found between lid tension and corneal astigmatism.^{333,334} It has been suggested that the shift toward against-the-rule astigmatism experienced by persons over 40 years of age is due to decreased lid tension. Vihlen and Wilson³³³ did find a decrease in lid tension with age. Another force that might affect corneal astigmatism is contraction of the extraocular muscles; small changes in corneal curvature have been observed to occur with convergence.^{335,336} American Indians have a high prevalence of with-the-rule astigmatism,³³⁷⁻³⁴² but the cause of this increased prevalence is unknown. The presence of with-the-rule astigmatism appears to be related to the degree of American Indian ancestry.^{334,343,344} Wilson et al.³³⁴ found lower intraocular pressure in Navajos and Cherokees with higher amounts of with-the-rule corneal astigmatism.

SUMMARY

Newborn babies have a wide range of refractive errors. Infants with myopia shift toward emmetropia. Hyperopic infants shift toward emmetropia as well, so that by 5 or 6 years of age the vast majority of children are emmetropic. White newborns have a high prevalence of against-the-rule astigmatism, which decreases over the first few years of life.

The school-age years are the most common time of myopia onset. Once myopia appears in childhood, it increases in amount until the middle to late teens. The major ocular optical component change associated with childhood myopia progression is increased vitreous depth. Several factors have been related to higher rates of childhood myopia progression. These include earlier myopia onset age, near-point esophoria, temporal crescents and other myopic fundus changes, higher intraocular pressure, greater amount of time spent reading and doing near work, and less time spent on outdoor activities. The most common methods for trying to control childhood myopia progression are rigid contact lenses

and bifocal spectacle lenses. Rigid contact lenses slow myopia progression by flattening the cornea. Myopia control with bifocals is more likely in children with near-point esophoria than in children with orthophoria and exophoria at near.

From the late teens or early 20s to about 40 years of age, refraction is usually fairly stable. Many persons with myopia experience increases in their myopia. Some emmetropic individuals become myopic at this time. The rates of young adulthood myopia progression are generally less than those of childhood myopia progression. Persons with hyperopia sometimes experience increases in their hyperopia.

After 40 years, there is generally a slow shift toward hyperopia, but some persons with myopia experience further increases in myopia. Persons who develop nuclear cataracts may have a change in refractive error toward myopia. Astigmatism changes in the direction of against-the-rule astigmatism, which becomes more common than with-the-rule astigmatism for the first time since infancy. Changes in refractive astigmatism can be attributed to changes in corneal astigmatism.

The genetic inheritance and vision activity factors that influence refractive development are not known for certain. Coordinated growth of the eye (increases in vitreous depth associated with decreases in crystalline lens power) may serve as a coarse control of refractive error development, with increases of vitreous depth in response to feedback from ocular image clarity serving as a fine control. The etiology of astigmatism is unknown; most theories have centered on mechanical causes.

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4

Accommodation, the Pupil, and Presbyopia

Kenneth J. Ciuffreda

THE ACCOMMODATIVE PROCESS

Accommodation refers to the process whereby changes in the dioptric power of the crystalline lens occur so that an in-focus retinal image of an object of regard is obtained and maintained at the high-resolution fovea.¹ Although lenticular-based focusing was first proposed by Descartes,² it was Thomas Young³ who initially demonstrated that changes in the crystalline lens itself were responsible for such focusing changes, and Hermann von Helmholtz, considered the father of physiological optics,⁴ who advanced the first basic but reasonably accurate explanation of the accommodative process.

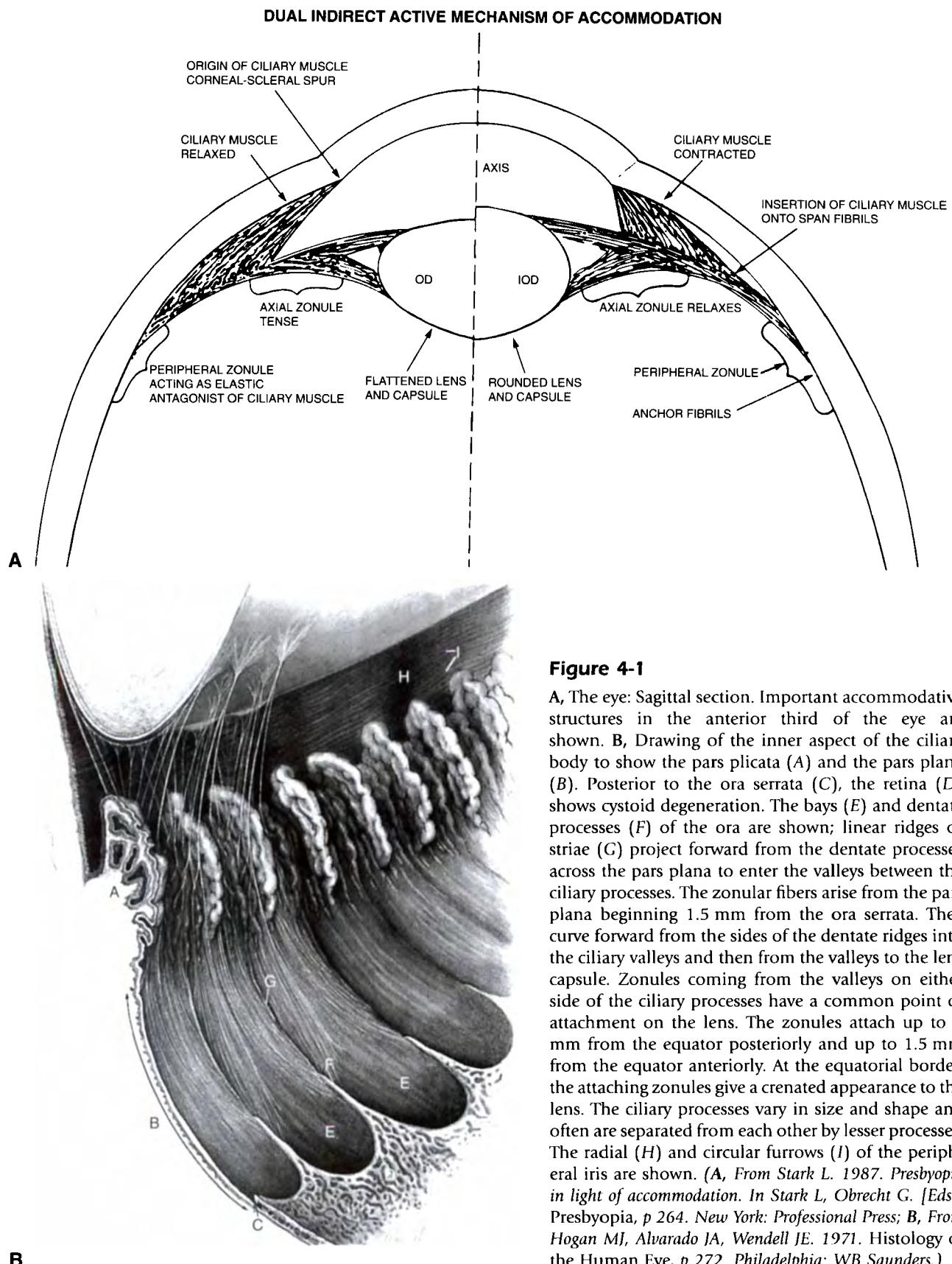
A variety of ideas have been proposed regarding how we see clearly at different distances. Some of them are as follows.

1. **There is no need for an active form of focusing.** This radical notion was accounted for by judicious use of the interval of Sturm in uncorrected astigmatism and appropriate placement of the depth of focus (approximately 1.00 D total) in all other situations. Clearly, this is insufficient to explain the clarity of vision and large range of accommodation found in children and young adults.
2. **Pupil size changes with the effort to see clearly at near.** However, the depth of focus (approximately 1.00 D total) for the smallest normal physiological pupil diameter (approximately 2.0 mm) in pre-presbyopes again can account for only a small portion of their accommodative amplitude.
3. **Corneal curvature changes with a change in focal point.** As Thomas Young³ demonstrated over 200 years ago, however, when he immersed his cornea into a beaker of water and thus neutralized its power, accommodation was still possible. Therefore, the cornea is not a factor in the accommodative process.
4. **The anteroposterior position of the lens changes with variation in focal point.** This theory has been

discounted by a variety of techniques, including biomicroscopy and ultrasonography. Moreover, given the small range over which the lens could theoretically shift in the human eye, the changes in power would be rather small and, again, could not begin to equal the 15.00 D or so amplitude found in young children.

5. **Changes in the axial length of the eyeball itself account for shifts in the position of the retinal image for objects at various distances.** Again, Young,³ with his large, protruding eyeballs and considerable commitment, provided the disconfirming evidence for this theory. Placing a clamp near the anteroposterior axis of his own eye, Young demonstrated that the size and intensity of the mechanical-pressure-generated phosphene did not change with accommodation. Thus, the eye did not change in axial length. This theory has also been discounted for the most part with clinical ultrasound. However, minute changes in axial length (<0.04 D equivalent) with accommodation have been found using laboratory-based partial coherence interferometry.⁵
6. **Changes in the shape, and therefore power, of the crystalline lens allow objects at various distances to be focused on the retina.** By both default and available evidence and logic, this is clearly the correct mechanism of the human accommodative process. Some of the processes listed above do occur in other species, however.⁶

The basic sequential biomechanical and anatomical changes that occur during accommodation are shown in Figures 4-1, 4-2, and Box 4-1.^{1,7-14} The only active element is the ciliary muscle. All other elements act in a passive manner. For example, when the ciliary muscle contracts, it pulls the ciliary ring forward and inward and stretches the choroid and posterior zonules. When the ciliary muscle subsequently relaxes, the passive restoring forces of the spring-like choroid and posterior zonules return each element to its former position. Similar passive changes occur during this process with



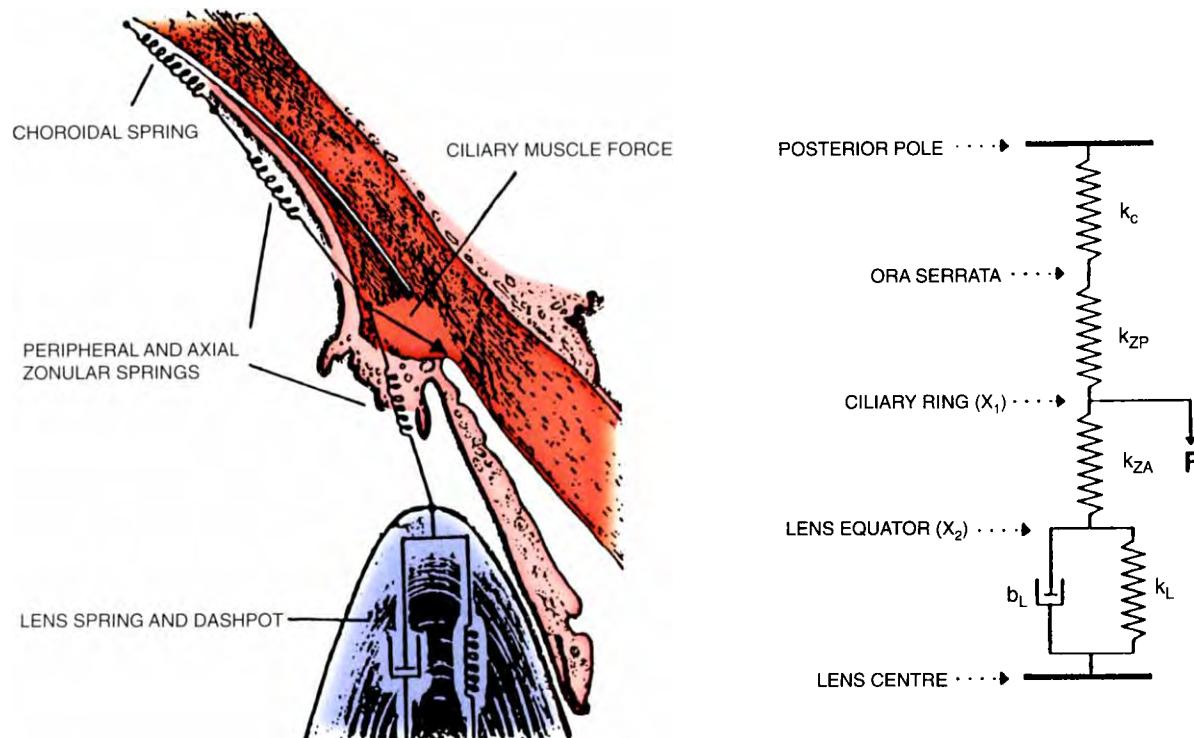


Figure 4-2

Dynamic biomechanical model. On the left, the model is shown superimposed on the anatomy of the component elements of the mechanism of accommodation. On the right, the model is given schematically. The model consists of three springs and a spring with parallel dashpot that are placed in series between two fixed points: the lens center and the posterior pole of the eye. k_c , Choroid spring constant; k_{ZP} , peripheral zonule spring constant; k_{ZA} , axial zonule spring constant; k_L , lens capsule spring constant; b_L , lens fiber cytoplasm damping coefficient; F , ciliary muscle force; x_1 , ciliary ring position; x_2 , lens equator position. (Reprinted from Beers APA, van der Heide GL. 1994. *In vivo determination of the biomechanical properties of the component elements of the accommodation mechanism*. Vision Res 34:2897. With permission from Elsevier Science Ltd.)

Box 4-1 Steps in the Biomechanics of the Near Accommodative Process (1 to 9 D Change) in a Young Adult

1. A step input increase occurs in the firing frequency of neural innervation to the ciliary muscle.
2. The contraction force of the ciliary muscle increases.
3. The ciliary muscle moves inward and anteriorly.
4. The ciliary ring advances approximately 0.5 mm along with the ciliary muscle.
5. The choroid and posterior zonules stretch approximately 0.5 mm.
6. The anterior zonular tension decreases, and the zonules relax.
7. The elastic forces of the lens capsule and the viscoelastic properties of the lens cause the lens to become more spherical. Thus the overall power of the lens increases:
 - a. The equatorial diameter decreases by 0.4 mm (from 10 to 9.6 mm).
 - b. The anterior lens pole moves back 0.3 mm.
 - c. The central anterior radius of curvature changes from 11 to 5.5 mm.
 - d. The posterior lens pole may move back 0.15 mm.
 - e. The central posterior radius of curvature decreases from 5.18 to 5.05 mm.
 - f. The central thickness increases by 0.36 to 0.58 mm.
 - g. The lens sinks 0.3 mm as a result of gravity.

respect to the anterior zonules, lens capsule, and crystalline lens. With increased ciliary muscle contraction, the anterior zonules reduce their tension and "relax," allowing the inherent forces of the lens capsule and lens itself to interact appropriately. With subsequent reduced contraction of the ciliary muscle, the anterior zonules exhibit increased tension, thus pulling on the lens capsule and lens. Again, all of these changes (except for

the ciliary muscle itself) represent passive biomechanical alterations.

The general neurological (sensory and motor) sequence of events leading to accommodation and the gross neuroanatomical pathways are presented in Figure 4-3 and Box 4-2.^{1,15} However, recent evidence suggests two important roles for the cerebellum.¹⁶ It may facilitate predictive tracking and also act as a general

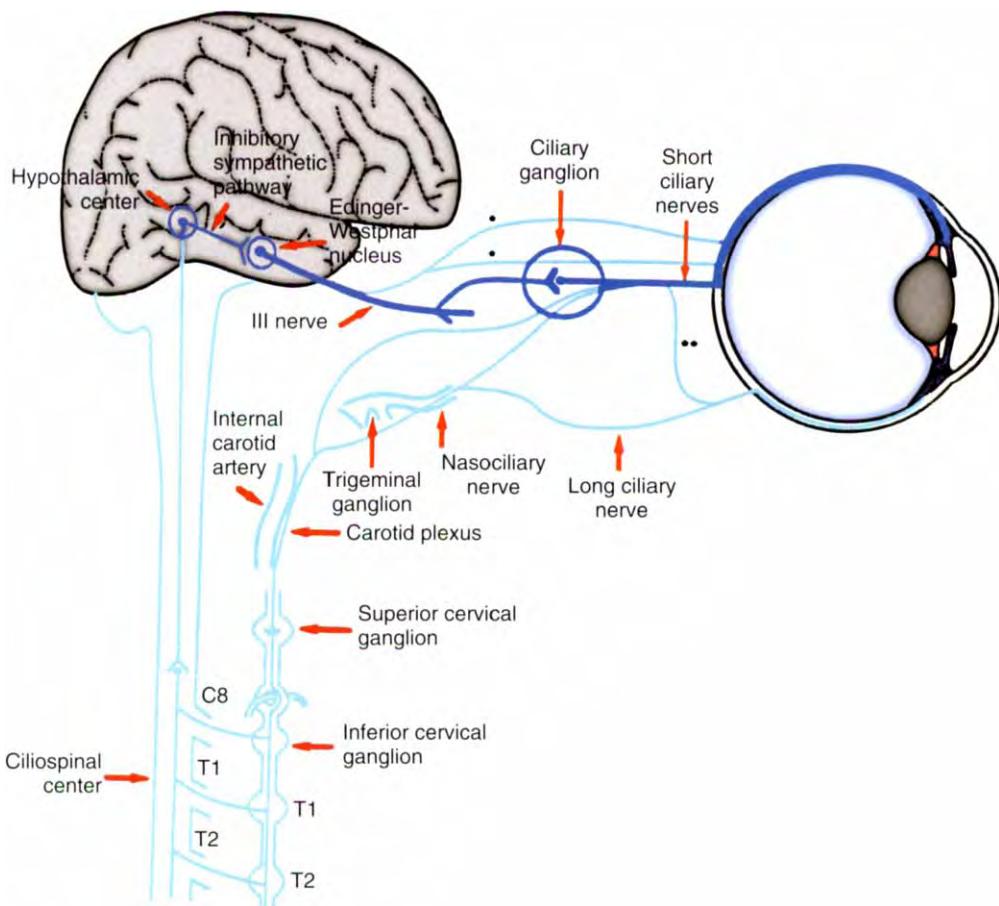
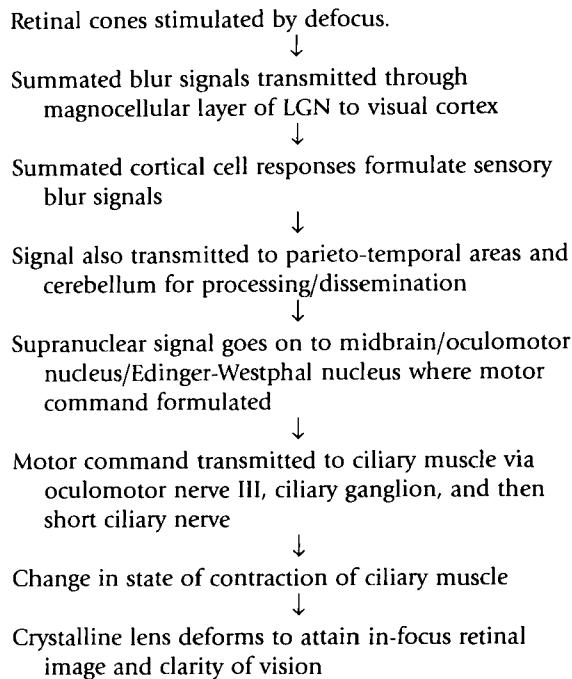


Figure 4-3

Parasympathetic and sympathetic pathways to the ciliary muscle. The major innervation to the ciliary muscle is parasympathetic and follows the pathway shown by the thick *solid lines*. The parasympathetic pathway originates in the Edinger-Westphal nucleus and courses with the third nerve, where the fibers travel to and synapse in the ciliary ganglion. The majority of the postganglionic parasympathetic fibers travel to the ciliary muscle via the short ciliary nerves, but some of them (*double asterisk*) also travel with the long ciliary nerves. There is also evidence for a direct pathway of uncertain functional significance (*single asterisk*) to the internal eye structures from the Edinger-Westphal nucleus. The sympathetic supply to the ciliary muscle (*thin solid lines*) originates in the diencephalon and travels down the spinal cord to the lower cervical and upper thoracic segments, to synapse in the spinociliary center of Budge in the intermediolateral tract of the cord. From there, second-order nerves leave the cord by the last cervical and first two thoracic ventral roots; these preganglionic fibers run up the cervical sympathetic chain to synapse in the superior cervical ganglion. The third-order fibers continue up the sympathetic carotid plexus and enter the orbit, either with the first division of the trigeminal nerve (following the nasociliary division) or independently, where they join the long and short ciliary nerves, in the latter instance passing through the ciliary ganglion without synapsing. CB, cervical vertebra 8; T1, thoracic vertebra 1, T2, thoracic vertebra 2. (From Kaufman PL. 1992. Accommodation and presbyopia. Neuromuscular and biophysical aspects. In Hart WM [Ed], Adler's Physiology of the Eye, 9th ed, p 397. St. Louis, MO: Mosby.)

Box 4-2 **Sensory and Motor Pathway for Monocular Blur-Driven Accommodation**


LGN, Lateral geniculate nucleus.

Box 4-3 **Components of Accommodation**

- Reflex accommodation
- Vergence accommodation
- Proximal accommodation
- Tonic accommodation

gain "calibrator" to ensure consistency in response accuracy.

Components of Accommodation

Analogous to the four-part component classification developed by Maddox¹⁷ over 100 years ago for vergence, Heath¹⁸ developed a classification for accommodation. He divided accommodation into functional or operational units that together form a conceptual framework for the relation among the accommodative stimuli, their separate and interactive motor effects, and the final overall steady-state system response (Box 4-3; also see Steady-State (Static) Model of the Accommodative

System). Heath's four components of accommodation include reflex, vergence, proximal, and tonic accommodation.

Reflex Accommodation

Reflex accommodation is the automatic adjustment of refractive state to obtain and maintain a sharply defined and focused retinal image in response to a blur input, that is, a reduction in overall contrast and contrast gradient of the retinal image. This occurs for relatively small amounts of blur, perhaps up to 2.00 D or so¹⁹; beyond that, voluntary accommodative effort is required.^{19–21} Small scanning eye movements, or microsaccades, assist in the process,⁹ possibly by producing multiple retinal-image luminance gradients about the fovea from which the blur information can be more easily extracted.²² Reflex accommodation is probably the largest and most important component of accommodation under both monocular and binocular viewing conditions.²³

Vergence Accommodation

Vergence accommodation is the accommodation induced by the innate neurological linking and action of disparity (fusional) vergence.²⁴ This gives rise to the convergence accommodation/convergence (CA/C) ratio, which is approximately 0.40 D per meter angle (MA) in young adults.²⁵ The CA/C ratio is determined by measuring accommodation during open-loop viewing (i.e., with blur feedback rendered ineffective) using either binocular pinholes or a blur-free difference of Gaussian target with low center spatial frequency.²⁶ These methods prevent the intrusion or "damping" action of blur-driven reflex accommodation on its response, as indeed does occur during the clinical measurement of relative vergence ranges.^{27–29} Vergence accommodation is probably the second major component of accommodation.

Proximal Accommodation

Proximal accommodation is the accommodation due to the influence or knowledge of apparent (or perceived) nearness of an object.³⁰ It is stimulated by targets located within 3 m of the individual,³¹ hence its name. With both the accommodative and disparity vergence systems open loop, so that no visual feedback is available with respect to blur and disparity, respectively, proximal accommodation is fully manifested. Its open-loop contribution can become quite large with near viewing, providing up to 80% of the total near response, that is, the combined proximal and tonic outputs.²³ However, under normal, binocular, closed-loop viewing conditions, the accommodative and disparity vergence systems receiving visual feedback dominate the response, and thus the proximal contribution becomes

quite small (around 4%, with a maximum of 10%).²³ Proximal accommodation is stimulated by perceptual cues, and therefore it does not have a separate retinal-based visual feedback loop. It represents a tertiary component of accommodation.

Tonic Accommodation

Tonic accommodation is revealed in the absence of blur, disparity, and proximal inputs,^{32,33} as well as any voluntary or unusual learned aspects. There is no stimulus per se for tonic accommodation, as there is for the other three components. Rather, it presumably reflects baseline neural innervation from the midbrain and thus represents a relatively stable input. Tonic accommodation can be measured in many ways, all of which involve removal of the other three inputs. Perhaps the best way to measure tonic accommodation is to place the individual in the center of a totally darkened room whose walls are at least 3 m away from the person, with the accommodative measuring device also away from and not visible to the person so that proximity and propinquity effects, which will inflate the true tonic value,³⁴ are prevented. Under such conditions, the mean tonic accommodative level in young adults is approximately 1.00 D, with a range from nearly 0 to 2.00 D.³⁵ Earlier mean estimates (approximately 2.00 D, with a range from 0 to 4.00 D) were inflated because of the presence of proximal and/or cognitive influences during the subjective measurement task.^{32,33,35} When the retinal image becomes markedly degraded under monocular viewing conditions (assuming little or no proximal input), accommodation shifts to the tonic accommodative default level. Tonic accommodation reduces with age because of the biomechanical limits of the crystalline lens.^{32,36-38}

Development of Accommodation

Considerable insight has been gained in the past 30 years or so into the accommodative ability of young infants. In the classic study by Haynes et al.,³⁹ dynamic retinoscopy (see Chapter 18) was used to assess steady-state accommodation at various near distances. Accommodative stimulus-response profiles (discussed in the next section) were determined in infants whose ages ranged from 6 days to 4 months. Accommodation during the first month appeared to be relatively fixed at approximately 5.00 D, whereas in the subsequent 3 months it progressively became more accurate and approached adult-like behavior. In a later study using a more compelling stimulus array, Banks⁴⁰ found more mature accommodative ability in young infants, especially during the first month of life. This has been suggested and/or confirmed by others,⁴¹⁻⁴³ although some variation has been found.⁴⁴ The calculated depth of focus showed a similar developmental trend,⁴⁵ being

large in the first month and decreasing considerably over the next 2 months. It appeared that infant accommodation was dictated by the level of neurosensory development and sensitivity at the time of testing. However, reasonable blur sensitivity can be demonstrated even in very young infants.^{46,47} However, in a study with a very large sample, Hainline et al.⁴⁸ found accommodation in infants younger than 2 months of age to be like that of either Haynes et al. or Banks et al. for near targets. Thus, limitations in sample size have obscured the results of the earlier investigators. Accurate accommodation to far targets was observed after 2 months of age, confirming Braddick et al.'s⁴² finding. Only tonic accommodation did not appear to exhibit an early developmental trend⁴⁹; it was the same (approximately 1.40 D) in infants and young adults. Accommodative amplitude in preterm infants as assessed by dynamic retinoscopy can be considerable, even greater than 8.00 D.⁵⁰ Finally, accommodative dynamics in response to steps of blur input appear to have adultlike velocities by the age of 3 months.⁵¹ With regard to vergence accommodation, Bobier et al.⁵² have demonstrated this function to be present in infants 3 to 6 months of age. It develops concurrently with both blur-driven accommodation and fusional vergence, thus allowing the normal array of binocular vision interactions to develop, or problem areas to become manifest (e.g., strabismus due to an abnormally high accommodative convergence/accommodation [AC/A ratio]).

There have been no carefully controlled studies of accommodation in young children between the ages of 1 and 4.5 years. Children at these ages are difficult to assess properly because it is not easy to ensure that one has their full attention, that they understand the test procedures and criteria, and that they exert maximal effort for measurement of accommodative amplitude and facility. However, it should be possible to obtain reasonable estimates of accommodative accuracy and sustaining ability by using dynamic retinoscopy with targets of high attentional value, for example in a game-like environment. Such knowledge will become especially important as clinicians begin to see more children in this age range as primary care practitioners, especially as a result of increasing governmentally mandated legislation in the United States. In one study in children ages 2 to 14 years,⁵³ the amplitude of accommodation decreased with age, thus being consistent with overall age-related trends for older individuals. Chen and O'Leary²⁴ found that the slope of the accommodative stimulus/response function remained relatively constant (0.92) and normal with age in young emmetropic children (3 to 14 years old) using objective methods. Hence, young children exhibit appropriate levels of accommodation to targets in free space.

Within the age range from 5 to 10 years, a number of studies have been conducted, several of which assessed

TABLE 4-1 Composite Representing the Number of Children, Mean Working Distance, and Mean Accommodative Lag M and SD (OD and OS) for Grades K to 6, Determined with MEM

Grade	N	Working Distance (Inches)	MEM OD		MEM OS	
			M	SD	M	SD
K	99	7.8	+0.28	0.44	+0.31	0.44
1	74	8.4	+0.21	0.39	+0.23	0.42
2	108	9.7	+0.30	0.30	+0.31	0.29
3	103	10.2	+0.34	0.32	+0.35	0.32
4	102	10.6	+0.32	0.32	+0.35	0.32
5	109	11.3	+0.35	0.34	+0.39	0.31
6	126	11.3	+0.45	0.30	+0.46	0.29

From Rouse MW, Hutter RF, Shiflett R. 1984. A normative study of the accommodative lag in elementary school children. Optom Vis Sci 61:693. MEM, Monocular estimate method.

the amplitude of accommodation.⁵⁵⁻⁵⁷ Essentially, the amplitude values in this younger age range added to and extended the classic Duane⁵⁸ population curve, which covers the ages 8 to 72 years (see Age, Accommodation, and Presbyopia). When the effects of individual experimenter test bias are taken into account, there is a gradual reduction in magnitude with age, as is found for older children and adults. Over this same time period, the lag of accommodation exhibits a slow but progressive increase to adult levels⁵⁹ (Table 4-1), whereas dynamic accommodative facility gradually improves⁶⁰ (Table 4-2). After 12 years of age, children respond more or less the same as normal young adults.^{61,62} However, this probably does not reflect actual physiological changes in accommodative dynamics per se as much as it reflects increased motivation, attention, and understanding of the task and its blur criterion.

Steady-State (Static) Accommodative Stimulus–Response Function

One of the most important relations to understand in this area is the accommodative stimulus–response function, or the profile of accommodative response.^{1,63-65} It provides an accurate, quantitative description of the accommodative response over a full range of accommodative stimuli, allowing practitioners to understand several fundamental principles regarding neurological control of accommodation. This profile can be divided into the following six zones or regions (one linear and five nonlinear), each with specific response characteristics (Figure 4-4).

Linear Manifest Zone

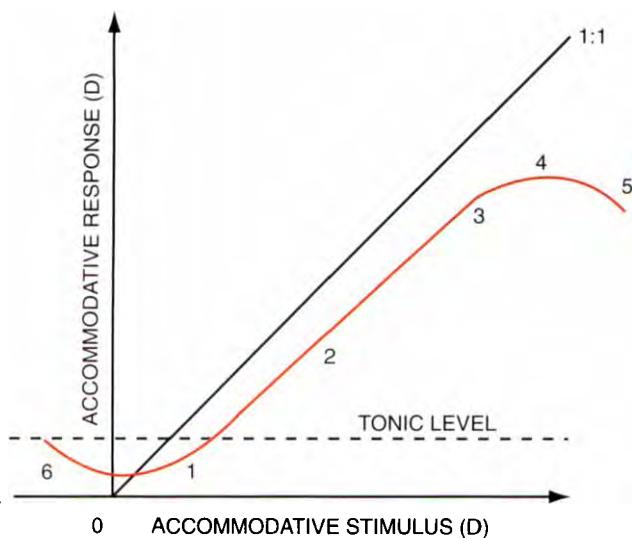
The linear manifest zone is the response midregion over which a change in the accommodative stimulus pro-

TABLE 4-2 Expected Findings for Accommodative Facility Testing of Children

Age (yr)	FACILITY NORMS (cpm)	
	Monocular	Binocular
6	5.5 ± 2.5	3.0 ± 2.5
7	6.5 ± 2.0	3.5 ± 2.5
8–12	7.0 ± 2.5	5.0 ± 2.5
More than 12	11.0 ± 5.0	8.0 ± 5.0

Adapted from Scheiman M, Wick B. 1994. Clinical Management of Binocular Vision, p 21. Philadelphia: JB Lippincott.

duces a relatively large and proportional change in the accommodative response. This results from its presumed proportional neural controller⁶⁶; that is, the system error is proportional to the system input. Thus, as the near accommodative stimulus increases, the steady-state accommodative error increases by a fixed proportion.⁶⁷ The slope of the linear response region ranges from 0.7 to nearly 1.0.⁶⁸ Recently, it was proposed that the slope, the intercept, and the amount of data scatter be combined to provide an overall index of accommodative accuracy.⁷⁰ Some degree of underaccommodation (within the depth of focus) is the rule. Clinically, this gives rise to "lag of accommodation."⁷¹ Conceptually, the accommodative system changes focus by the minimum amount to place the object just within the eye's depth of field/focus and thereby obtains a subjectively clear and high-contrast retinal image. Additional accommodation would serve no useful purpose.

**Figure 4-4**

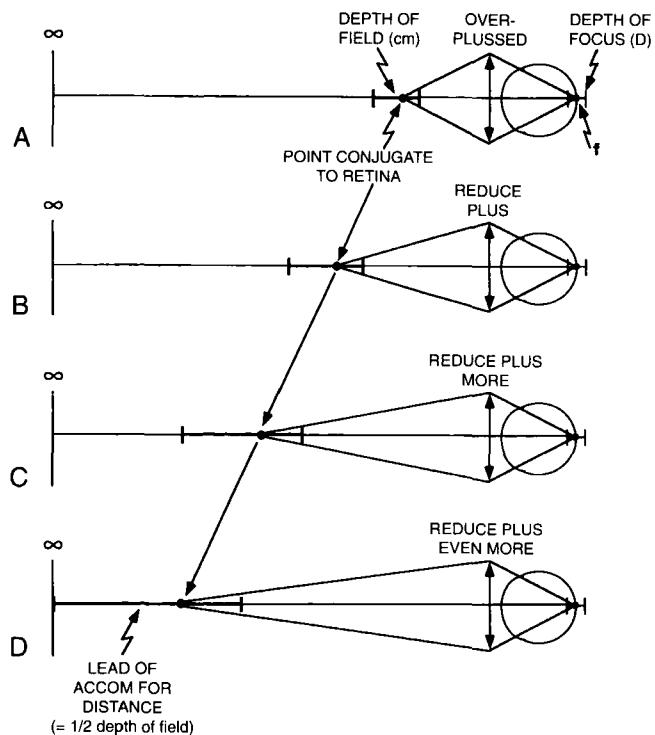
Static accommodative stimulus-response function profile (curved line). Equivalence of accommodative stimulus and response is represented by the diagonal line. Numbers represent zones of the profile. See text for details.

This lag can be measured by a variety of techniques, including dynamic retinoscopy (see Chapter 18.)

Initial Nonlinear Zone

The initial nonlinear region extends from 0 to 1.50 D or so. It might be slightly more with a physically close, closed-field optical stimulation and measurement system in which there is much proximal drive, and it might be somewhat less in a more distant, open-field system in which the proximal input is minimal or even absent (>3 m).³¹ At the very low end of this region, the steady-state accommodative response is primarily influenced by the small tonic input and the depth of focus⁷² (see Steady-State [Static] Model of the Accommodative System). The accommodative response to an infinity or zero diopter accommodative stimulus is not zero, but rather 0.25 to 0.33 D.⁷³ Therefore, there is a "lead of accommodation" when viewing at distance. This lead is a result of the same neural control property responsible for the lag of accommodation. The system again changes by the minimum amount necessary to see the distant target clearly, such that the target lies just within the distal edge of the depth of field (see Age, Accommodation, and Presbyopia). The retina need not be perfectly conjugate to a distant object for clear perception.

This leads to the important clinical concepts of hyperfocal refraction and hyperfocal distance, as well as the clinical maxim, "maximum plus for maximum visual acuity."⁷⁴ Toward the end of refractive testing, plus lenses are added and then gradually reduced, until no further increase in distance visual acuity is obtained. Thus the

**Figure 4-5**

Hyperfocal refraction and hyperfocal distance. *f*, Fovea; ACCOM, accommodation.

refractive end point or criterion is appropriately one of "maximum plus for maximum visual acuity," intended primarily to ensure that no latent hyperopia is present. As shown in Figure 4-5, at this point the eye is *not* conjugate to optical infinity or even the Snellen test chart at 20 feet (1/6 [0.17] D accommodative stimulus), but rather is myopic by about 0.25 D with respect to infinity.⁷³ The retina is optically conjugate to a point approximately 4 m away from the individual. Therefore, as the additional plus lenses are reduced, the far point and its surrounding depth of field are optically shifted farther away in space, until the distal edge of the depth of field is conjugate either to optical infinity or to a distant target. From this, the *hyperfocal distance* can be defined as the closest distance for which the eye may be conjugate and still have a satisfactory and clear retinal image of an object at infinity. The process itself and the lens combination that accomplishes it are referred to as the *hyperfocal refraction*. Again, as long as the target remains within this blur-free region, it will be seen clearly. Thus, the lead of accommodation at far here is in fact dictated by the clinical test procedure and related visual acuity criterion (see Chapter 20).

Nonlinear Transitional Zone

The region in which further increases in the accommodative stimulus level (just beyond the upper linear

manifest zone) produce progressively smaller changes in accommodative response (i.e., "soft saturation") is the nonlinear transitional zone. In this zone, progressively greater increases in accommodative error are evident. This is due to initial crystalline lens biomechanical limitations in responsivity that occur near the upper limit of the amplitude, regardless of age (see Age, Accommodation, and Presbyopia).

Nonlinear Latent Zone

The region in which yet further increases in the accommodative stimulus level fail to produce any additional change in accommodative response (i.e., "hard saturation") is the nonlinear latent zone. This region extends approximately 2.00 D beyond the nonlinear transitional zone, with its initial portion defining the amplitude of accommodation. This total lack of responsivity is due to further biomechanical limits in lens responsivity. This zone has also been referred to as the age-independent "functional presbyopic region" (see Theories of Presbyopia). The maintenance of such a high level of accommodative response over this 2.00 D zone of noncompensable retinal defocus reveals the robustness of the accommodative system to such image degradation effects.

Myopic Nonlinear Defocus Zone

The myopic nonlinear defocus zone is the region in which changes greater than 2.00 D above the accommodative amplitude produce yet further amounts of noncompensable retinal defocus, reducing the retinal-image contrast gradient sufficiently to reduce its stimulus effectiveness and thus producing a gradual decrease in the accommodative response toward the tonic accommodative level of 1.00 D or so. When very little retinal-image contrast is finally present (i.e., a contrastless "ganzfeld" condition is approximated), accommodation approaches this default tonic level, assuming both disparity and proximal accommodation are absent (see Components of Accommodation).

Hyperopic Nonlinear Defocus Region

The region in which dioptric stimulation extending just *beyond* optical infinity (e.g., as can be produced in a Badal optical system) produces noncompensable hyperopic retinal defocus is the hyperopic nonlinear defocus region. As in the myopic zone, the accommodative response gradually approaches the tonic level. However, in this case, the accommodative level increases (relative to the infinity response level of 0.25 D or so) to this default level of 1.00 D.

In both the myopic and hyperopic nonlinear defocus regions, the accommodative response shifts toward the tonic accommodative bias level. However, with any form of image degradation (e.g., contrast, luminance, or spatial frequency composition), irrespec-

tive of the response region, the accommodative stimulus effectiveness and drive are potentially reduced, with the result that the accommodative error is progressively increased. With considerable image degradation, the slope of the accommodative stimulus-response function essentially decreases to zero, with its mean response level reflecting the tonic accommodative bias level, perhaps in conjunction with proximal accommodation if the degraded stimulus environment is perceived to be within 3 m.^{23,71}

Factors that Affect Accommodation

A multitude of factors affect the accommodative response to varying degrees. As Box 4-4 shows, these factors may be categorized as the stimulus to, the cue for, and the influence on accommodation.¹

There is considerable evidence and consensus that blur is the stimulus to accommodation.^{1,15,19,75} Our eyes see a blur pattern and respond accordingly to produce an in-focus retinal image. There are simple and inexpensive autofocus cameras that operate on this principle. Thus, there is little justification to propose anything more complicated for the basic accommodative blur detection and reduction process.

In contrast, cues for accommodation provide the requisite directional information regarding the blur pattern. This can be provided by such diverse entities as optically based spherical aberration⁷⁶ (see Optical Aberrations) and perceptually based apparent distance.^{30,77} If all such directional information is removed, leaving only a change in blur pattern, the accommodative system responds directionally in a chance manner.⁷⁸ That is, on 50% of the trials the initial direction of the accommodative response will be incorrect, requiring a second response that is now in the appropriate direction to obtain clear vision. This demonstrates an important bioengineering operating principle, called the accommodative system's *even-error control*. It can detect the magnitude, but not direction, of blur.

Finally, there are factors that can influence the accommodative response. These include such diverse entities as voluntary effort,²¹ mood,⁷⁹ and target luminance.⁸⁰ As an example, if a high-contrast target, initially placed at some intermediate distance from an observer, is suddenly and rapidly displaced either farther or closer along the midline, a retinal blur pattern would be produced that would serve as the stimulus to accommodation. Any retinal blur pattern asymmetries dependent on the direction of defocus would provide the directional information, and the level of target contrast would influence the magnitude of the final steady-state accommodative response.

Box 4-4 is not an exhaustive list of the factors that affect accommodation, but most of the important ones

Box	Cues to and Influences on Accommodation	
Cues		Influences
Optical		Nonretinal Image
Chromatic aberration		Vestibular stimulation
Spherical aberration		Training/therapy
Astigmatism		Mood
Microfluctuations		Prediction
Blur asymmetry due to fixational eye movements		Voluntary effort
		Cognitive demand
		Visual imagery
		Instruction set
Nonoptical		Retinal Image
Size		Spatial frequency
Proximity		Contrast
Apparent distance		Retinal eccentricity
Disparate retinal images		Retinal-image motion
Monocular depth cues		Luminance
		Size
		Depth of focus
		Disparity-driven vergence
		accommodation

are presented. For more detailed discussion of these factors and their affect on accommodation, see Ciuffreda.¹ Figure 4-6 shows how some of these factors affect the accommodative response and the extent to which they do so. In general, the accommodative system is robust (i.e., relatively insensitive) to such factors as target contrast,^{81,82} spatial frequency,^{83,84} luminance,⁸⁰ and pupil diameter.⁸⁵ These parameters can vary considerably before the steady-state response exhibits significant additional error. In contrast, the system is much more sensitive to the effects of target retinal eccentricity^{1,86} and retinal-image motion.^{87,88} Even with small increases in these parameters, the accommodative error begins to increase. In all cases, with sufficient degradation of the retinal image, the accommodative response approaches the tonic level.

In the research laboratory, investigators carefully vary a single target parameter, such as contrast, while keeping constant all other parameters, such as luminance, color, and spatial frequency. In this way, they determine the effect of an isolated parameter on accommodative responsivity. In real-life situations, however, this is certainly not the case. For example, as the eye looks at various kinds of printed material at near, such as pictures, newsprint, books, and maps, the stimulus parameters (e.g., color, contrast, size, and spatial frequency composition) vary considerably. Furthermore,

the subjective impressions of target "quality" among people and the presumed accommodative stimulus effectiveness also vary markedly. For example, most people would rate textbook print as having good quality/effectiveness and a low-resolution photograph in the local newspaper as having poor quality/effectiveness. However, at least for stationary targets in the central field, despite wide variations in objective stimulus characteristics and subjective impressions, the accommodative response is remarkably accurate and stable (Figure 4-7).⁸⁹ It seems that nature has allowed for accommodative responsiveness and its neurological control to be quite robust for a wide range of combined stimulus features, as is indeed essential in real-world situations for optimal detection and discrimination of fine details.

A Steady-State (Static) Model of the Accommodative System and Its Interactions

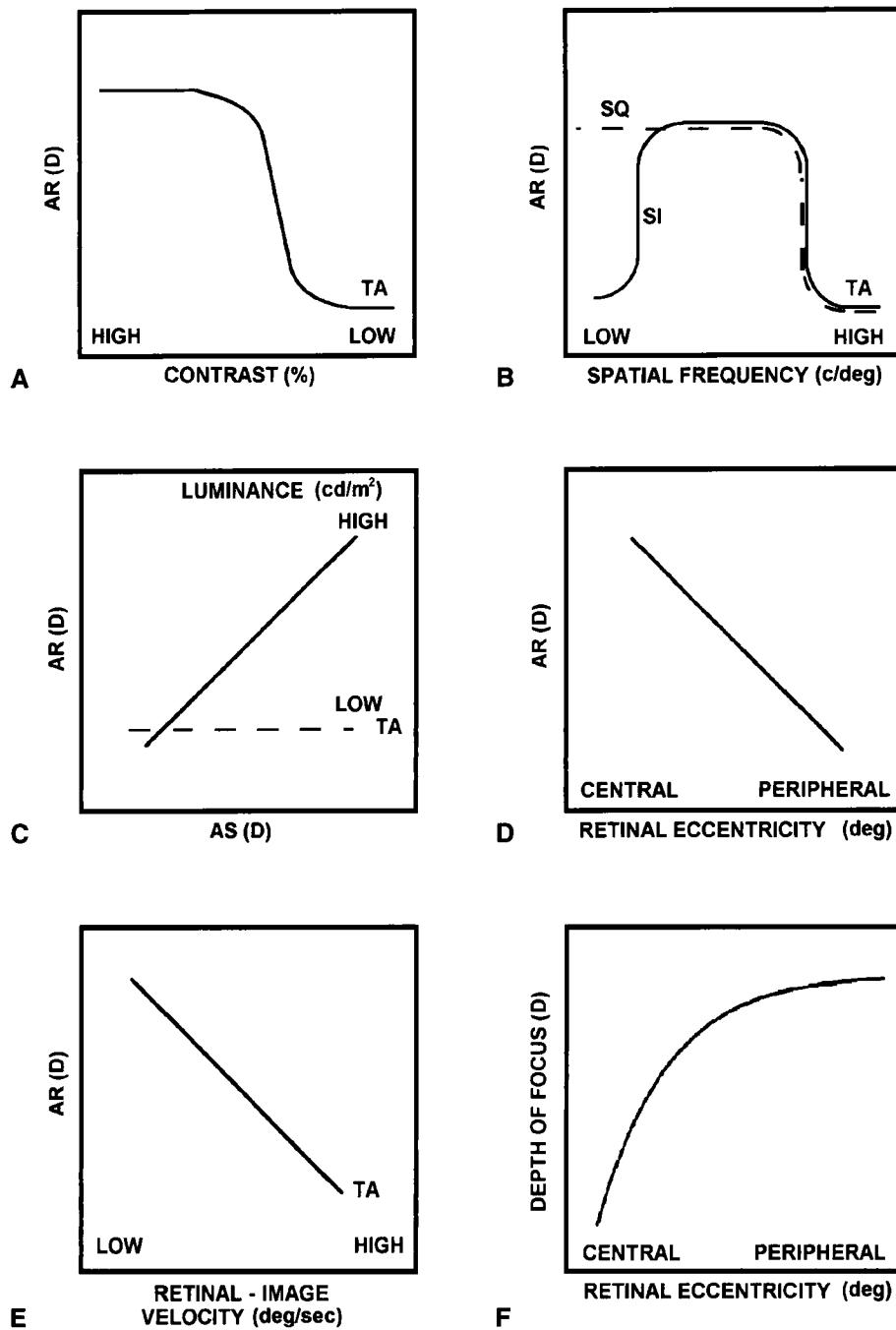
A useful static model of the accommodative system and its motor interactions has been developed over the years by Hung and his colleagues^{23,27,68,72,90-93} for both basic and clinical studies. The latest, amplified version is presented in Figure 4-8.²³ Moving from left to right in the figure, it may be seen that the accommodative and disparity loops have similar component structures.

Input

The input or stimulus change for accommodation (target distance in diopters later converted to retinal defocus/blur) and disparity vergence (target distance in meter angles later converted to retinal disparity) sum with the negative feedback response of the respective system at that moment. This difference represents the initial system error. The input for the proximal branch is target distance. This becomes converted to perceived distance, which is multiplied by the two subsequent gain terms and is then finally input to both the accommodative and vergence forward pathways. Note that this perceptually driven component does not have a visual feedback pathway of its own, because it constitutes a nonretinal (versus retinal, i.e., defocus and disparity) input.

Threshold "Deadspace" Operator

This represents the depth of focus for accommodation and Panum's fusional areas for disparity vergence. This component allows some small neurosensory-based system error to be tolerated without adverse perceptual consequences, such as blur and diplopia, respectively. If such neural tolerance were not allowed, we would be forced to have perfect motor system responses at all times, which is clearly an unrealistic expectation. Only

**Figure 4-6**

A-F, The effects of changes in several important target characteristics on the steady-state accommodative response (AR). SQ, square wave; SI, sine wave; TA, tonic accommodative level; AS, accommodative stimulus; c/deg, cycles per degree.

if the input error exceeds this threshold level does it proceed to drive either system.

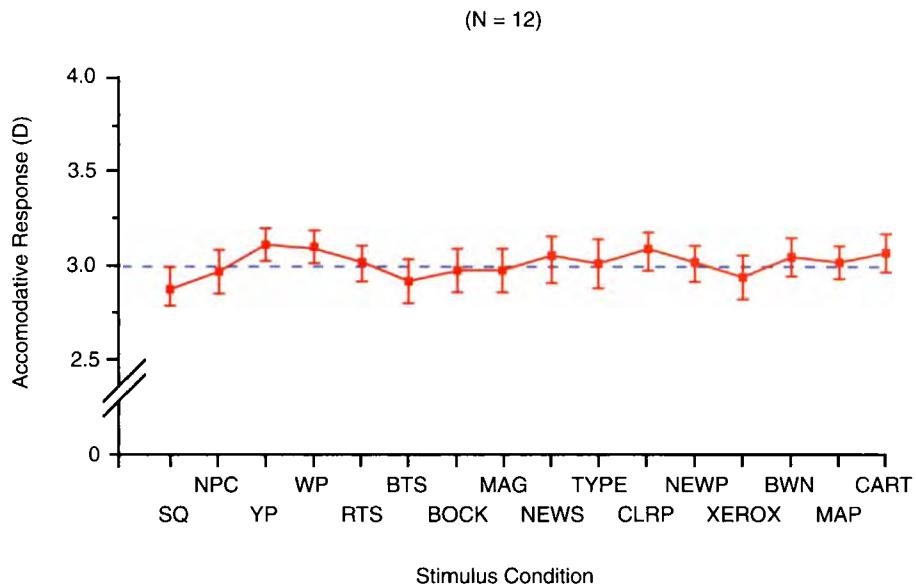
Gain

The gain represents the experimentally derived open-loop gain of the system. It multiplies its input error signal. The gain term and all the elements in its direct forward pathway dynamically represent the "fast" sub-

system. That is, this complex is responsible for generating the initial 1 sec or so response to a blur or disparity input to obtain an immediately clear and/or fused retinal image.

Adaptive Loop

Once the "fast" system response is completed and only a small steady-state error exists, the adaptive loop is

**Figure 4-7**

Mean accommodative response for 12 subjects. The broken horizontal line indicates the accommodative stimulus level. Error bars indicate ± 1 standard error of the mean. *SQ*, square-wave grating; *NPC*, reduced Snellen near-point card; *YP*, yellow pages telephone directory; *WP*, white pages telephone directory; *RTS*, red train schedule; *BTS*, blue train schedule; *BOOK*, paperback novel; *MAG*, magazine; *NEWS*, newspaper; *TYPE*, typed text; *CLRP*, instant color photograph; *NEWP*, color newspaper photograph; *XEROX*, photocopy of *NEWP*; *BWN*, black and white newspaper photograph; *MAP*, multicolored street map; *CART*, black and white newspaper cartoon. (Reprinted from Ciuffreda KJ, Rosenfield M, Rosen J, Azimi A, Ong E. 1990. Accommodative responses to naturalistic stimuli. *Ophthal Physiol Opt* 10:168. With permission from Elsevier Science.)

activated. Its input is the output of the gain element, and its output goes back into the same gain element. This adaptive, or "slow," subsystem acts to sustain the motor response for a prolonged period, presumably preventing or minimizing system fatigue and correlated near-work symptoms⁹⁴⁻⁹⁶ and possibly even myopia development and its progression.⁹⁶⁻¹⁰⁰

Crosslink Gain

This crosslink gain term multiplies the output of the direct pathway gain term. For accommodation, this new value represents the effective AC/A ratio, whereas for convergence it represents the effective CA/C ratio. For example, if the crosslink gain from accommodation to vergence were abnormally high, the vergence system would be overdriven, and an esotropia might result. Conversely, reduced gain might result in exotropia.

Tonic Input

Tonic input presumably reflects midbrain baseline neural innervation. These tonic terms have negligible influence on the overall closed-loop near response and only modest influence on the far response.⁷² As described earlier (see Components of Accommodation), their influence primarily occurs in the absence of any visual feedback.

Summing Junction

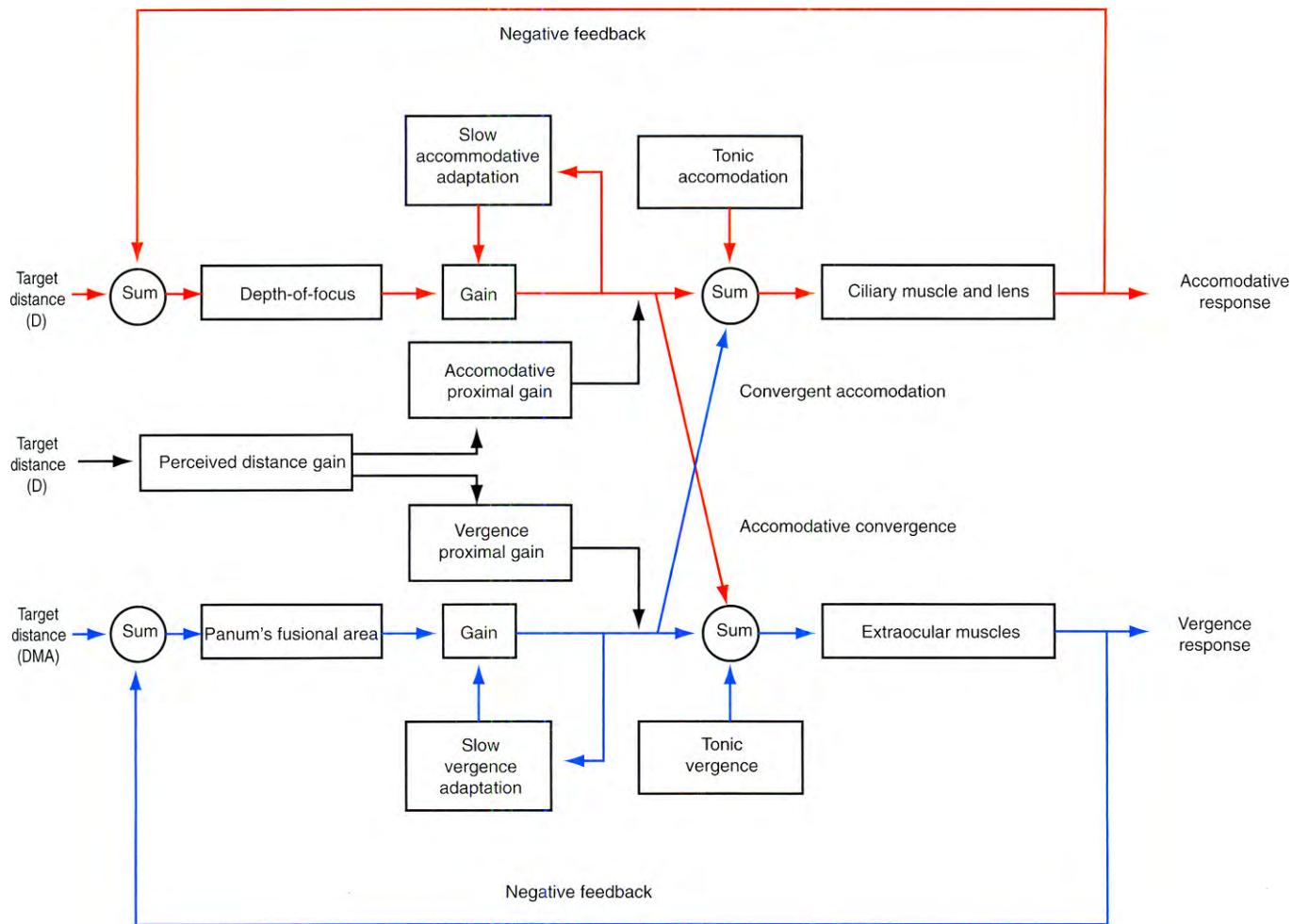
The gain output is directed to the summing junction, where it sums with the crosslink output, the proximal output, and the tonic input to form the final combined signal to drive the respective system.

Peripheral Apparatus

The output of the summing junction proceeds to cortical and subcortical centers related to accommodation to formulate the basic neural signal¹ and then advances to innervate the appropriate peripheral apparatus—the ciliary muscle and lens complex for accommodation and the extraocular muscles for vergence. These motor changes are then returned to the initial summing junction via the negative-feedback pathways. If a relatively large residual error remains, the cycle is repeated until an acceptably small, stable steady-state error for both systems is attained.

Dynamic Aspects of Accommodation

The accommodative system can respond reasonably quickly and accurately to a variety of blur stimuli (Figure 4-9). These stimuli include steady-state inputs, step and pulse inputs, sinusoidal inputs, and ramp inputs.

**Figure 4-8**

Simplified version of Hung et al.'s²³ comprehensive static model of accommodation and vergence. See text for details. MA, Meter angle.

Steady-State Inputs

During attempted steady focus on an object at a fixed distance from the eyes, the steady-state accommodative response actually exhibits variable frequency (0.05 to 5.00 Hz, with prominent bands at 0.05 to 0.50 Hz and 1.50 to 2.00 Hz), small-amplitude (± 0.02 to 0.20 D) oscillations, or microfluctuations, about the mean response level^{1,101,102} (Figure 4-9, A). These oscillations have a maximum amplitude at midrange and a minimum amplitude at the accommodative range limits, suggesting biomechanical involvement. At the far point, zonular tension is too great to allow easy transmission of the small band of high-frequency energy, and at the near point, the zonules are too relaxed to transmit such small perturbations faithfully. At the midrange, the moderate degree of zonular tension is optimal to allow these oscillations to manifest themselves fully. Correlation of oscillations between the two eyes suggests a central origin.¹⁰³ The precise role of these microfluctuations in accommodative control remains

controversial, with theories running the gamut from simple system feedback instability and biological noise to periodic blur feedback to the eye to help maintain steady-state accuracy.¹ However, current thinking is that only the lower frequency oscillations play a direct role in accommodative control,¹⁰² with the higher frequencies being an epiphenomenon related to arterial pulse.¹⁰⁴

Step and Pulse Inputs

The accommodative response to a step input can be approximated by an exponential (Figures 4-9, B, and 4-10)¹⁰⁵ having a time constant (i.e., the time to reach 63% of its final response amplitude) of approximately 200 to 250 msec.⁷⁶ Average latency (i.e., reaction time) is 370 msec; latency from far to near is 360 (± 90) msec, and that from near to far is 380 (± 80) msec.⁷⁶ Reaction time to unpredictable step inputs is less (around 180 ms) than for predictable inputs, with much greater variability, including "negative" values when one actually

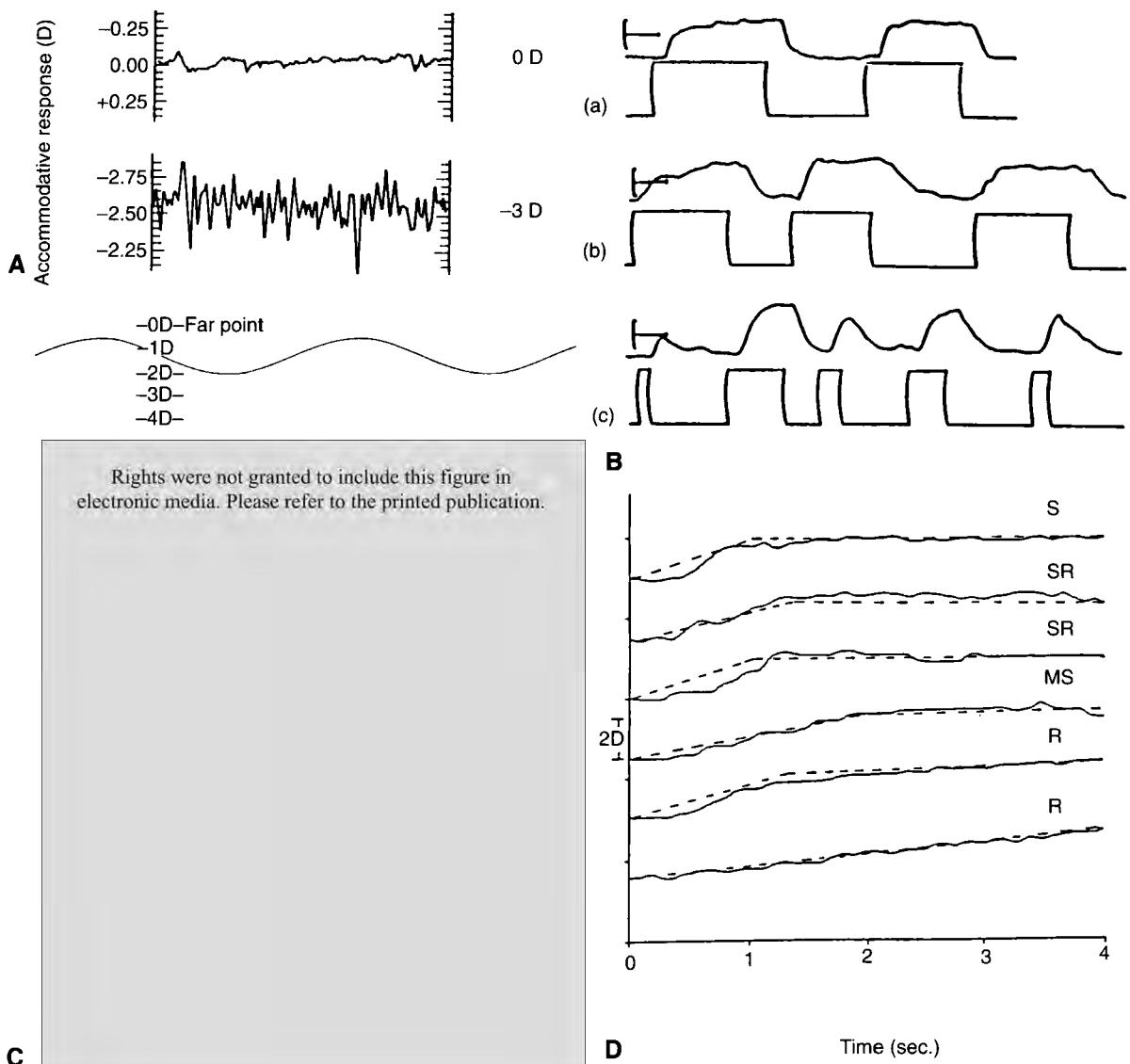


Figure 4-9

A, Typical responses of accommodation for two target vergences during steady-state stimulus conditions. The ordinate for each trace is an absolute dioptric scale. **B**, Graph (a) shows accommodative responses to a 2 D step stimulus and return to zero level of accommodation. Allowance should be made for the arc of the pen. The top line shows accommodation (the length of the horizontal line represents 1 sec, and the height of the arc represents 1 D; upward movement represents far-to-near accommodation); the bottom line shows the stimulus signal on the same scale. This record is an example of the single-sweep accommodative responses. Graph (b) shows typical accommodative responses to a 2 D step stimulus and return when targets change only in focus and not in size. Note the increased variability in response relative to that in (a). The length of the horizontal line represents 1 sec, and the height of the arc 1 D. Graph (c) shows the accommodative responses when a far visual stimulus is replaced by an identical one at a nearer optical distance for various time intervals presented in random order (rectangular pulse stimuli). The length of the horizontal line represents 1 sec, and the height of the arc 1 D. **C**, Sinusoidal responses at various temporal frequencies. In each case, the upper line shows the stimulus changes, the middle line traces the corresponding response, and the bottom line is marked in seconds. **D**, Representative dynamic accommodative responses for different ramp velocity stimuli (0.5–2.5 D/sec). Shown are individual accommodative responses (*solid lines*) and stimuli (*dashed lines*). Response type is indicated to the right of the response traces. Note that in the third trace from the top, the step-ramp (SR) response is followed by another step. S, Steplike; MS, multiple step; R, ramp. (A, Adapted from Miege C, Denienl P. 1988. Mean response and oscillations of accommodation for various stimulus vergences in relation to accommodation feedback control. *Ophthal Physiol Opt* 8:165; B, From Campbell FW, Westheimer G. [1960]. Dynamics of accommodative response of the human eye. *J Physiol* 151:285; C, From Kasai T, Unno M, Fujii K, et al. 1971. Dynamic characteristics of human eye accommodation system. *Osaka Univ Tech Report* 21:569; D, From Hung GK, Ciuffreda KJ. 1988. Dual-mode behavior in the human accommodation system. *Ophthal Physiol Opt* 8:327. With permission from Elsevier Science.)

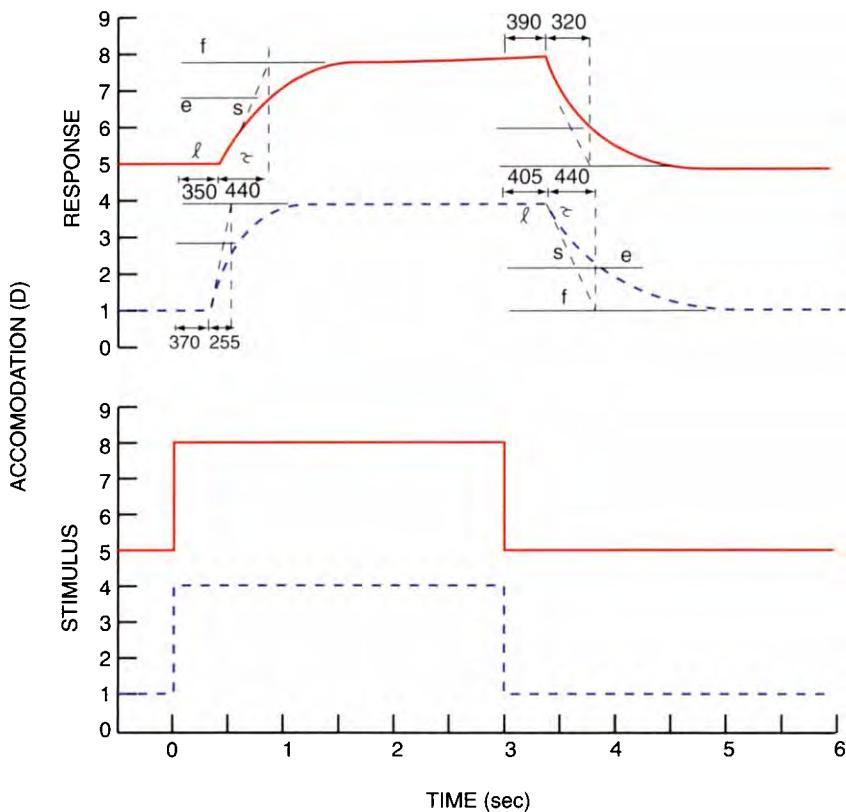


Figure 4-10

Analysis of accommodative dynamics. Idealized responses are shown for stimulus changes in near (solid lines) and far (dashed lines) range. Latencies (*l*) can be directly measured. Time constants (τ) are determined by two methods: time for the tangent (*s*) to reach the final response value (*f*) and time for the response to reach 63% (*e*) of the final response value. (From Shirachi D, Liu J, Lee M, et al. 1978. Accommodation dynamics: Range nonlinearity. Am J Optom Physiol Opt 55:631.)

responds before the target position changes.¹⁰⁶ Such responses clearly demonstrate the presence of a predictor operator, with maximum effectiveness over the stimulus range of 0.1 to 0.7 Hz. Total response time (latency plus actual lens movement time) is approximately 1 sec. Peak accommodative velocity increases in proportion to response amplitude, reaching 10.00 D/sec for the largest movements.^{20,107,108} Responses exhibit more variability when only blur information is present than when blur plus size information is present,⁷⁶ confirming the notion of blur dominance and response enhancement with addition of the various cues to accommodation. Subtle dynamic differences in accommodation may exist between the dominant and nondominant eyes.¹⁰⁹

Regarding pulse inputs, responses suggest the presence of continuous visual feedback control, in that response durations approximate stimulus durations, with a delay of one reaction time.⁷⁶ If the system had solely discontinuous or "sampled-data" control, the response duration would be fixed and independent of stimulus duration.²⁸ (But see Ramp Inputs for discus-

sion of the notion of "dual-mode" control of accommodation.)

Sinusoidal Inputs

The accommodative response to a sinusoidal input exhibits a sinusoidal profile whose gain (i.e., response amplitude divided by target amplitude) reduces and whose phase lag (i.e., tracking position error) increases with higher stimulus frequencies (Figure 4-9, C). Basically, the investigator inputs a range of sinusoidal frequencies at a fixed amplitude, such as 3.00 (± 1.00) D, assesses system gain and lag (or lead, especially for predictable inputs, thus demonstrating the presence of a predictor operator for such stimuli), and constructs Bode plots (Figure 4-11). Such plots reveal optimal tracking over a range of approximately 0.05 to 0.40 Hz, with a total response range from 0.04 to 4.00 Hz.^{76,110}

Ramp Inputs

Ramp, or constant-velocity, stimuli have revealed several interesting aspects of accommodative control¹⁰⁷ (Figure

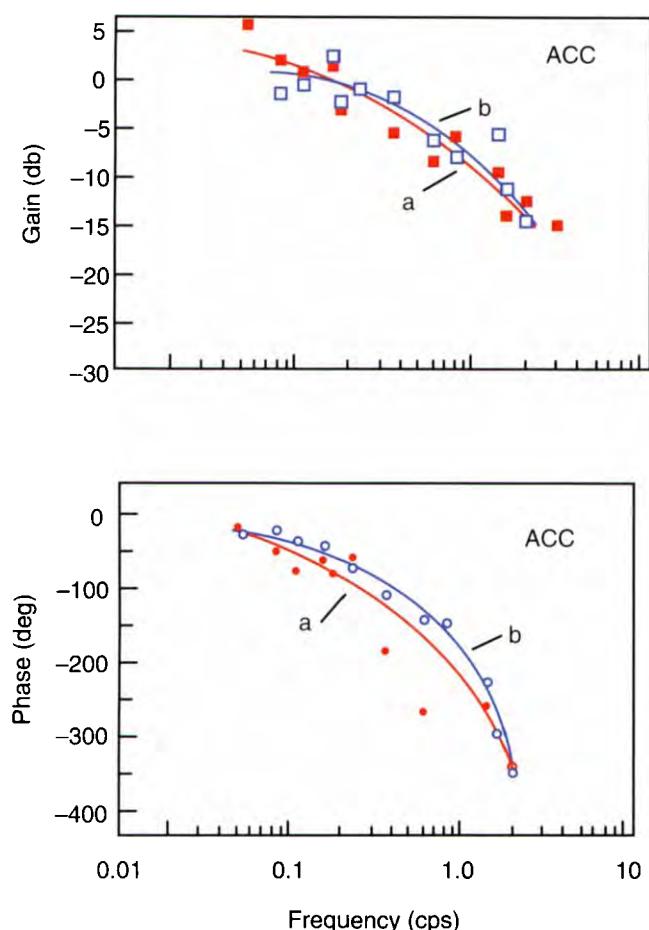


Figure 4-11

Frequency characteristics of the prediction operator for accommodation (ACC). The responses to predictable sinusoidal stimuli are indicated by thin lines with data points consisting of *open circles and squares*, and the responses to unpredictable sinusoidal stimuli are indicated by *thick lines* with data points consisting of *filled circles and squares*. The difference between the two sets of responses is a definition of the prediction operator. (Reprinted from Krishnan VV, Phillips S, Stark L. 1973. Frequency analysis of accommodation, accommodative vergence, and disparity vergence. *Vision Res* 13:545. With permission from Elsevier Science Ltd.)

4-9, D). For slow ramps, the responses are ramp-like, whereas for fast ramps, the responses are exponential in nature and similar to that found for step inputs. This suggests a "dual-mode" control of accommodation, with preprogrammed (i.e., not based on visual feedback) responses occurring for the fast ramps and slow responses involving continuous visual feedback control occurring for the slow ramps. Such dual control would allow for more effective and stable target tracking over a full range of target velocities.

Although some have attempted to approximate the dynamic accommodative system as linear in nature, it is

really nonlinear.¹¹ For example, it has dual-mode control and presence of additional harmonic components in the sinusoidal response.

A Dynamic Model of the Accommodative System

Figure 4-12 presents a dynamic model of the accommodative system. Similar to the static model presented earlier, this model, adapted from Krishnan and Stark,¹¹² provides a comprehensive, organizational framework for logical thinking and understanding of its elemental system components. An updated version of this model has recently been proposed.¹¹³ The various model elements, moving from left to right in the figure, are discussed in the next section.

Input

The input is the target accommodative stimulus level, that is, the target distance in diopters. It sums with the instantaneous accommodative level of the system via the negative-feedback loop. The difference between these two (i.e., target versus lens diopters) represents the initial system error.

Threshold "Deadspace" Operator

This represents the depth of focus (presumed 50% threshold criterion), which allows for some small neurosensory-based system error to be tolerated without the perception of blur. If such neural tolerance were not permitted, we would be forced to have a perfect motor response at all times for clarity of vision, obviously an unrealistic expectation. Only if the input error exceeds this threshold level does it proceed to drive the system.

Nonlinear Switching Element

Because blur is an even-error signal (i.e., it lacks directional information), this element uses the sign information from the derivative operator to determine its direction. It generates a signal that is directionally correct and proportional to the magnitude of blur.

Derivative Controller

This parallel, pseudoderivative controller component is a velocity operator. It generates the derivative of the error signal (i.e., the instantaneous velocity) for use by its control process. Such a controller improves the transient stability, as well as the speed, of the response.

Nonlinear Saturation Element

This element is a velocity-sensitive component that prevents the response velocity from exceeding a certain limit. This, too, facilitates dynamic response stability and limits the amplitude of oscillations of the accommodative response.

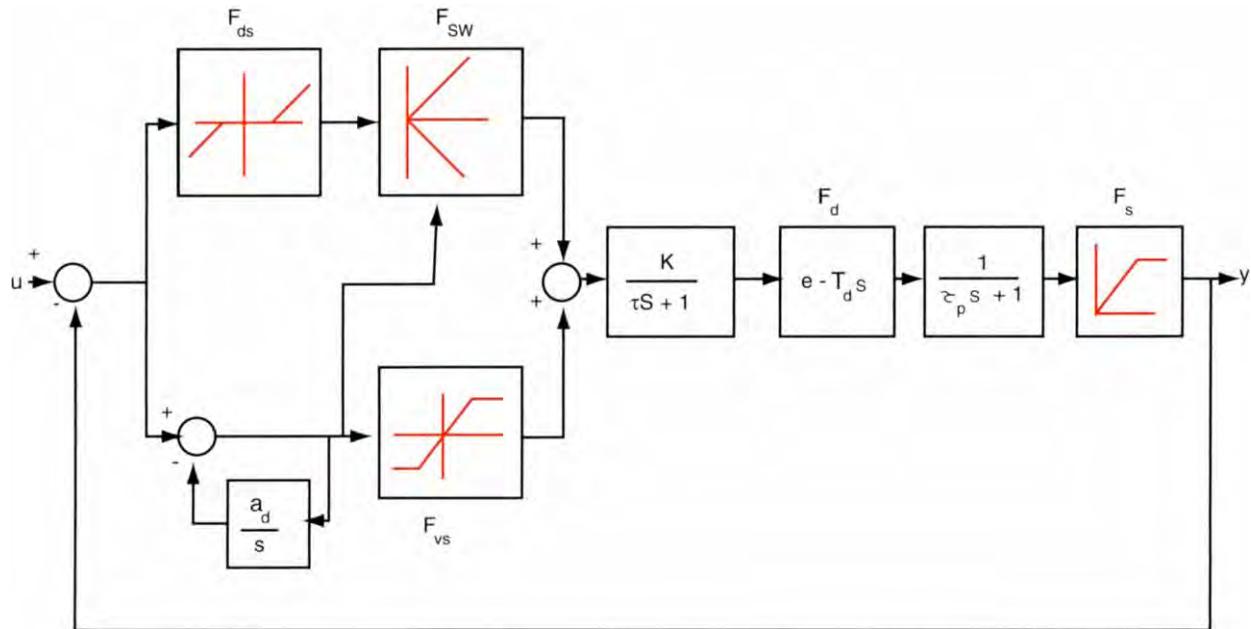


Figure 4-12

Simplified version of Krishnan and Stark's¹¹² dynamic model of accommodation. F_{ds} , Depth of focus; F_{sw} , switching component (even-error component); F_{vs} , velocity-sensitive saturation, or velocity operator, which limits the velocity change; u , input; $\frac{a_d}{s}$, lead/lag term (quasi-derivative controller, or velocity operator; $a_d = 10$ and involves dynamics and stability); y , accommodative amplitude, or "plant" saturation; K , gain; τ_p , time constant or decay for the accommodative peripheral apparatus ("plant") = 0.4 sec; F_d , time delay = $e^{-\tau ds}$, where T_d = accommodative latency = 0.38 sec; y , output; $\frac{1}{\tau s}$, integrator; $\frac{1}{1+\tau s}$, leaky integrator; τ , neural time constant, or accommodative decay = 10 sec. See text for details.

"Leaky" Integrator

The "leaky" integrator is a "charge/discharge" element. It represents a central neurological integrating circuit that is rapidly activated ("charged" like an electronic capacitor) by the visual input and stores this information, thus providing for steady-state maintenance of the response. In the dark without visual information related to the target, however, this circuit decays ("discharges") exponentially according to the value of its time constant, with the accommodative response shifting to the tonic accommodative bias level in 10 to 15 sec.

Time Delay

This represents the combined neural and biomechanical transmission time delays, or latency.

Ciliary Muscle/Lens Dynamics

This represents the response biomechanical characteristics of the ciliary muscle/zonules/lens/lens capsule complex, or "plant."

Saturation Element

The saturation element limits the accommodative response imposed by the lens elasticity. In effect, it represents the amplitude of accommodation.

Training the Accommodative System

A variety of studies over the past 45 years have demonstrated that the normal human accommodative system can be trained to improve response accuracy and time optimality. Such training primarily involves relatively rapid motor learning, as well as much slower perceptual learning to assess and respond appropriately to the blur signal.^{114,115}

Early on, Marg¹¹⁶ showed that steady-state accommodation could be varied easily by volitional control in the presence of a target and related blur feedback. These results were later confirmed and expanded by others^{20,21,117}; it was even found that such responses could be elicited in total darkness.²⁰ The dynamics of voluntary accommodation are the same as those of the more reflexive accommodation described by Fincham,¹¹⁹ suggesting similar basic neuromotor control despite very different modes and probable sites of initiation (i.e., retinal defocus versus higher level cortical control processes).²⁰ Cornsweet and Crane¹¹⁸ used an objective infrared recording technique and demonstrated that with only 3 hours of practice, subjects with normal vision could learn to use audio information related to accommodative state to control their accommodation under blur-free conditions (i.e., with open-loop accom-

modation via a pinhole) and match various standard tones. This voluntary accommodative ability, once learned, exhibited immediate transfer to a new task involving a visual-matching paradigm based on voluntary accommodative response level.

Using an objective recording system, Randle and Murphy¹¹⁹ showed that with repeated testing (every 3 waking hours for 7 days; 3 hours total per day) of dynamic accommodative ability (predictable step and sine inputs), performance improved considerably (Figure 4-13, A). Velocity to step inputs increased, and gain increased and phase lag decreased to the sinusoidal targets. However, there was no change in either latency or total response amplitude to the blur steps. These results are consistent with a clinical study by Levine et al.,¹²⁰ which showed that only a few minutes per day of testing accommodative facility with ± 2.00 D flippers produced considerable improvement in overall responsiveness in asymptomatic young adults.

Several studies have demonstrated that it is also possible to train and improve accommodation in symptomatic patients manifesting slowed dynamics. The first study was that of Liu et al.,¹²¹ who treated three optometry students with symptoms related to focusing difficulties at near using standard vision training procedures, including jump focus, plus-and-minus lens flippers, and pencil push-ups.¹²² Subjects trained themselves at home for 20 minutes each day for 4.5 to 7 weeks, and objective measurements of dynamic accommodation were made each week. Initially, these measurements showed prolongation of the time constant and latency of accommodation. During treatment, the patients exhibited significant reductions in these two parameters that correlated well with reduction of symptoms (Figure 4-13, B). Flipper rates increased and symptoms were either markedly diminished or no longer present at the termination of therapy. These results clearly demonstrate that vision training in this small sample of young adult patients resulted in objective improvement of accommodative function. The reduction in time constant suggested revision and improvement in the neuromotor control program,¹²³ leading to a more efficient, time-optimal response. This might involve greater synchronization of neural signals related to the improved blur information processing. The reduced latency also suggested more efficient signal processing of blur information. Two years later, these results were reproduced in children.¹²⁴ The adult results were also later confirmed and extended by Bobier and Sivak¹²⁵ using a different objective recording technique (photorefraction). They found no regression of improvement 4.5 months after cessation of training. In addition, subjective (minus lens) and objective (i.e., visual-evoked-response amplitude) increases in the amplitude of accommodation were recorded during a 4-month course of vision training in one patient¹²⁶ (Figure 4-14). Lastly, the slope of

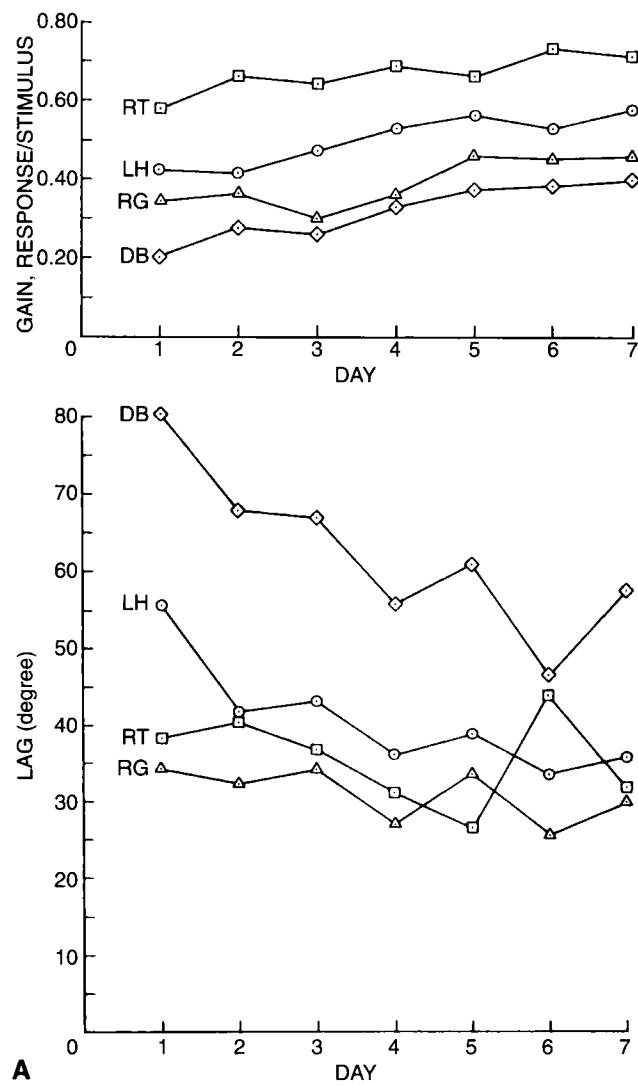
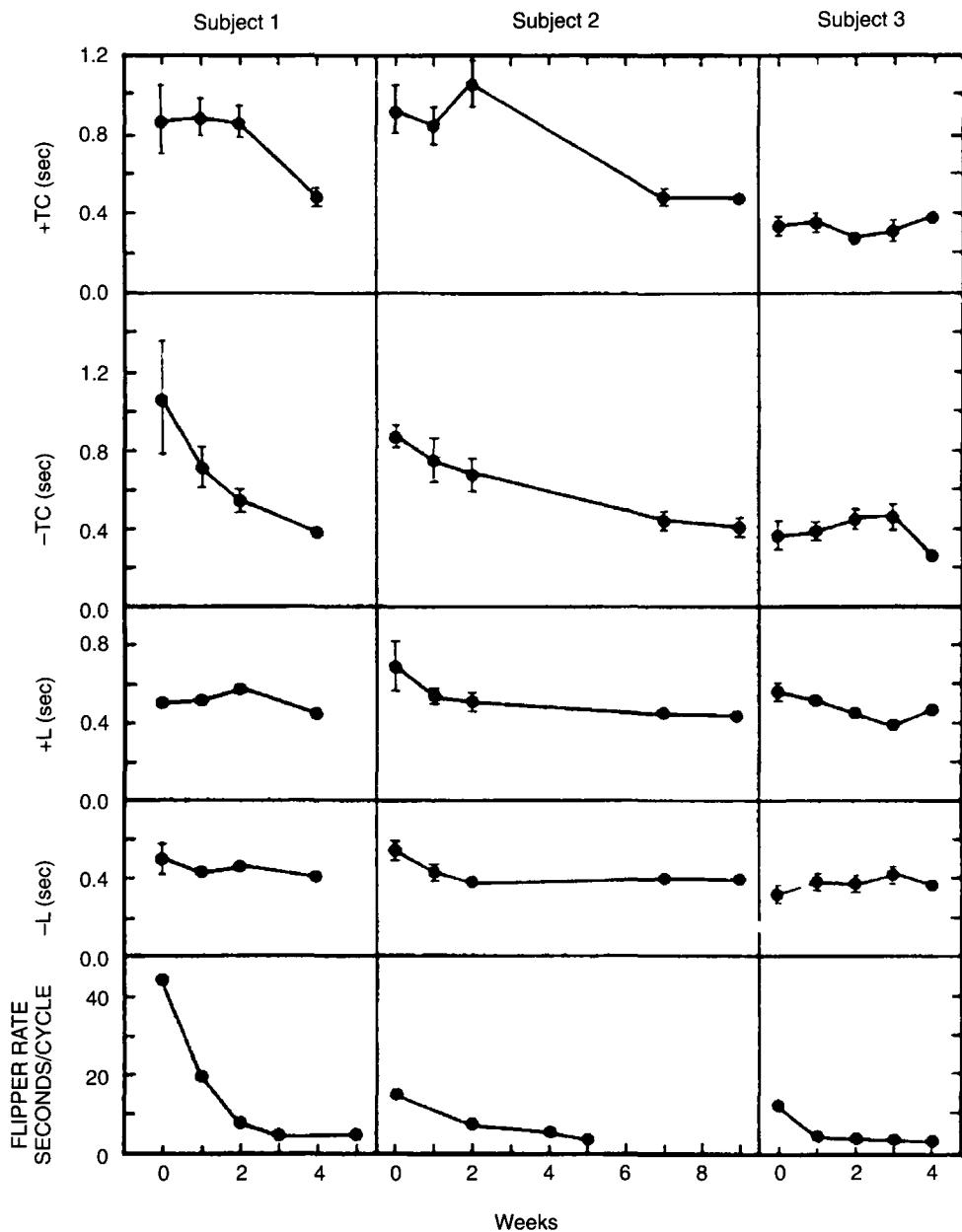


Figure 4-13

A, The improvement in gain for tracking a 0.5-Hz sine wave as a function of experimental day is shown at the top, and the decrease in phase lag for tracking a 0.5-Hz sine wave as a function of experimental day is shown at the bottom.

the accommodative stimulus-response function showed improvement after 8 to 16 weeks of basic accommodative therapy in a group of college students. This normalization was maintained when patients were retested 6 to 9 months later.⁹²

Together, results of the foregoing studies clearly demonstrate that symptoms related to near focusing were correlated with the clinical accommodative lens flipper rate.¹²⁷⁻¹³⁰ Furthermore, objectively determined improvement in accommodative dynamics was paralleled by similar changes (i.e., increased timed cycles) in accommodative lens flipper rate. Thus, in the clinical environment, the lens flipper ("accommodative rock") provides a simple, inexpensive, effective, and valid diag-

**Figure 4-13, cont'd**

B, Change of accommodative characteristics in the three subjects as measured weekly through changes in time constant (TC), latency (L), and flipper rate during their orthoptic therapy program. For time constants and latencies, mean values are plotted and standard errors denoted. Flipper rates are self-reported by subjects. (A, From Randle RJ, Murphy MR. 1974. *The dynamic response of visual accommodation over a seven-day period*. Am J Optom Physiol Opt 51:530; B, From Liu JS, Lee M, Jang J, et al. 1979. *Objective assessment of accommodation orthoptics: Dynamic insufficiency*. Am J Optom Physiol Opt 56:285.)

nostic and therapeutic indicator of overall accommodative dynamic ability. Combining this with careful static measures of accommodative amplitude (minus-lens technique or dynamic retinoscopy) and steady-state error of accommodation (i.e., near lag/lead, again using dynamic retinoscopy), practitioners can begin to obtain comprehensive static and dynamic clinical profiles of their patients' accommodative abilities.^{68,131}

A few studies have shown that it is possible to train and improve accommodation in patients with other clinical conditions with verification using objective recording techniques or psychophysical test paradigms. In the area of amblyopia, my colleagues and I showed that both static accommodation and dynamic accommodation (Figure 4-15) normalized after conventional vision therapy (part-time occlusion, eye-hand sensori-

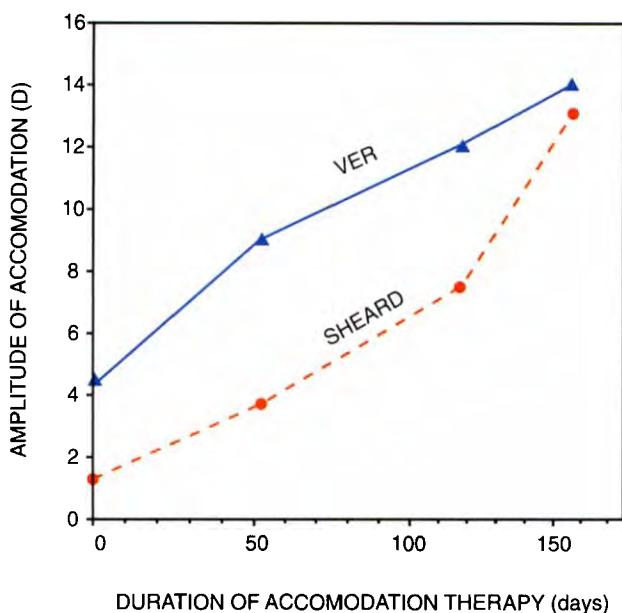


Figure 4-14

Changes in the amplitude of accommodation measured objectively (VER) and subjectively (Sheard's technique of minus lens to first blur) as a function of the duration of accommodative therapy. Note that although the two techniques revealed similar progressive increases in the amplitude of accommodation with therapy, the VER nearly always predicted a higher amplitude and indicated good concordance with the Sheard value at the latest measurement. (From Lovasik JV, Wiggins R. 1984. Cortical indices of impaired ocular accommodation and associated convergence mechanisms. Am J Optom Physiol Opt 61:150.)

motor exercises, and lens flipper).¹³² With regard to static accommodation, therapy resulted in reduced accommodative lag (and thus increased, more accurate response amplitude), reduced depth of focus, and increased accommodative amplitude.^{68,81,83,133–136} With respect to dynamic accommodation, therapy resulted in reduced latency, increased response amplitude (i.e., increased system gain), and more accurate accommodation, with less variability and improved response sustaining ability.¹³² The amblyopia therapy improved neurosensory sensitivity and processing, as well as reduced the unsteady and eccentric fixation, all of which acted to improve overall static and dynamic accommodative function.¹³² Similar findings were reported in a case of myasthenia gravis.¹³⁷ Also, one patient with congenital nystagmus achieved more accurate accommodation after eye movement auditory feedback therapy.¹³⁸ This probably resulted from reduced retinal-image motion and therefore a higher contrast retinal image with more distinct edges to stimulate accommodation more effectively.

A helpful clinical classification of accommodative anomalies was developed by Duane¹³⁹ and later adopted with some modification by others.^{140–142} Using his classification, Duane found that more than 10% of the patients in his general ophthalmology practice had non-pathological accommodative dysfunction. He proposed simple accommodative "exercises" for alleviation of symptoms in some cases. Duane's classification scheme can be used for both the diagnosis and treatment of an accommodative problem. With some updating and modification,^{141,142} it includes the following anomalies:

1. **Accommodative insufficiency**—Accommodation is persistently lower than expected for the patient's age. Reduction of the accommodative amplitude by 2.00 D or more is the hallmark. Its main symptom is general asthenopia related to near work. Subcategories are as follows:
 - a. Ill-sustained accommodation—Accommodation, especially its amplitude, is initially sustained only with considerable effort. Over time, it cannot be maintained. This may be the first stage of accommodative insufficiency. It has also been referred to as *accommodative fatigue*.
 - b. Paralysis (or paresis) of accommodation—The accommodative amplitude is either markedly reduced (*paresis*) or totally absent (*paralysis*), once compensation for the depth of focus is considered. It is frequently the result of an organic condition or head trauma.
 - c. Unequal accommodation—There is a persistent interocular difference in monocular accommodative amplitude of at least 0.50 D. This could result from organic disease, head trauma, or functional amblyopia.
2. **Accommodative excess**—Accommodative excess has traditionally been defined as accommodation that is persistently higher than expected for the patient's age. Using this definition, it is a rare condition to occur in isolation (i.e., without being secondary to convergence insufficiency). Modern definitions simply regard it as an inability to relax accommodation readily, one end of a continuum leading to frank spasm of accommodation. Using this newer definition, it is a relatively common condition.
3. **Accommodative infacility**—In this condition, accommodative dynamics (latency, time constant, and peak velocity) are slowed, with change in accommodation only occurring with effort and difficulty, in the presence of normal response magnitude (including the accommodative amplitude). This has been called *inertia of accommodation*. It is a moderately common condition. The most frequent symptom is difficulty changing focus to various near and far distances.

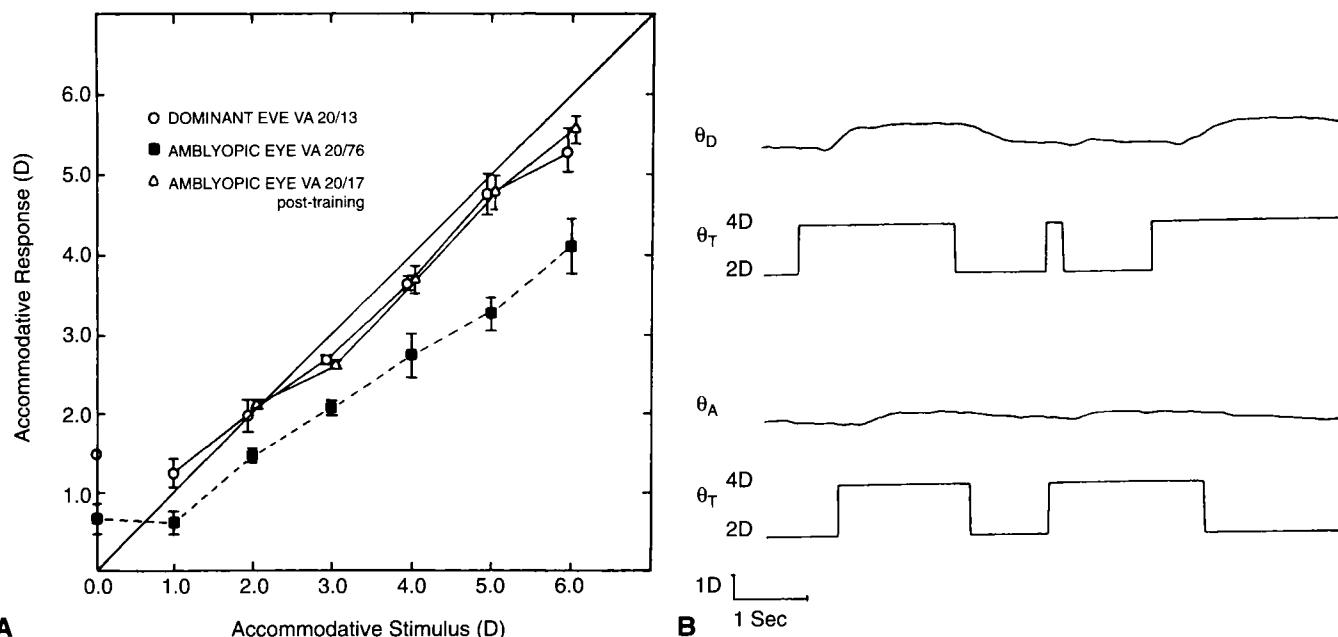


Figure 4-15

A, Accommodative stimulus–response curves in a 12-year-old patient with strabismic amblyopia before and after intensive orthoptic therapy. After therapy, monocular accommodative responses were similar in the two eyes. In the dominant eye, visual acuity (VA) = 20/13, accommodative controller gain (ACG) = 21, and slope = 0.88. In the amblyopic eye before therapy, VA = 20/76, ACG = 7.8, and slope = 0.68. In the amblyopic eye after therapy, VA = 20/17, ACG = 18, and slope = 0.87. Means and 1 standard deviation are plotted. **B**, Accommodative dynamics in a person with amblyopia (20/50). Step and pulse inputs are shown. θ_D , dominant eye response; θ_T , target positions; θ_A , amblyopic eye response. (A, From Ciuffreda KJ, Hokoda SC, Hung GK, et al. 1983. Static aspects of accommodation in human amblyopia. Am J Optom Physiol 60:436; B, from Ciuffreda KJ, Levi DL, Selenow A. 1991. Amblyopia: Basic and Clinical Aspects, p 292. Boston: Butterworth-Heinemann.)

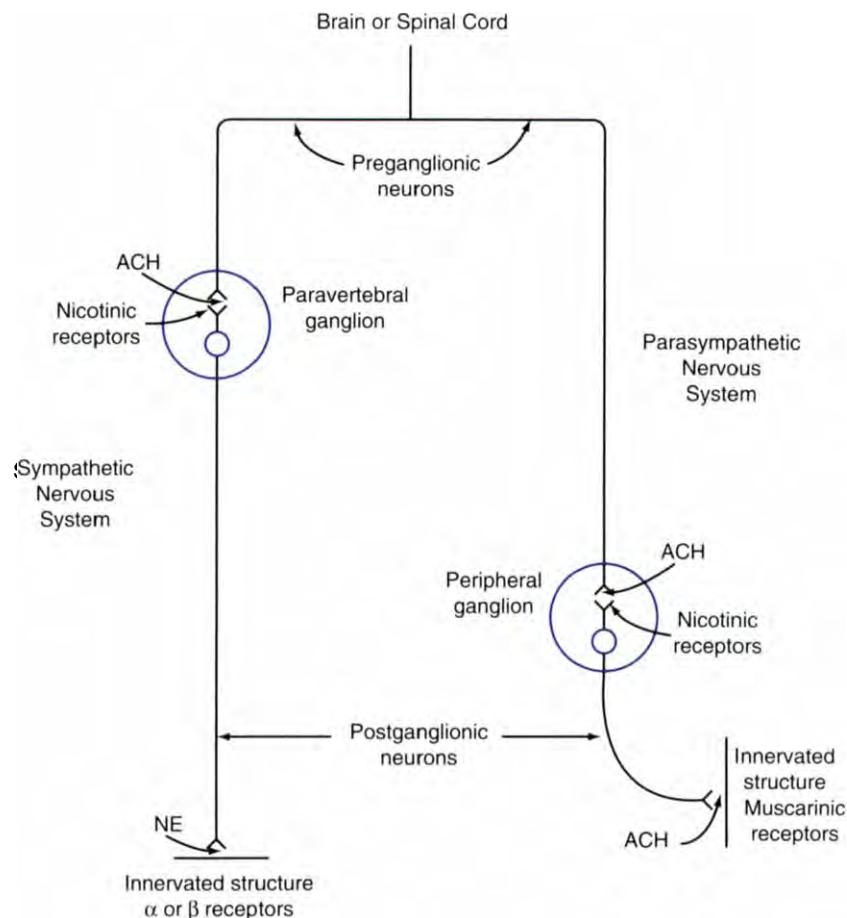
Effects of Drugs on Accommodation

Topically Applied Diagnostic Ophthalmic Drugs
The autonomic nervous system, upon which most topically applied diagnostic ophthalmic drugs act, can be subdivided into the parasympathetic and sympathetic branches. Each branch has distinct anatomical features and pathways, as well as its own physiological effects (Figure 4-16; see also Figure 4-3).^{15,94,143,144}

The *parasympathetic* branch provides the primary neural control over the entire range of accommodation. It has an excitatory effect—an increase in its activation level results in an increase in accommodation, and a decrease in its activation results in a decrease in accommodation. It is a rapidly acting system that participates in all general dynamic changes in accommodation that typically have a total response time of 1 second or so. Stimulation of this system also affects pupil diameter. There is an inverse relation between level of excitation and pupil size, thus producing pupillary miosis with high amounts of accommodation. The parasympathetic branch releases acetylcholine at the ciliary muscle receptor site.

In contrast, the *sympathetic* branch plays a secondary role in the control of accommodation. It has an inhibitory effect—an increase in its activation level results in a reduction in accommodation (although its effect is no greater than 2.00 D in primates).^{145,146} Initiation of sympathetic activation takes 5 to 10 seconds, and maximal stimulation requires 10 to 40 seconds. Furthermore, the sympathetic level of activity and its effect on refractive state are directly related to parasympathetic level of activity.^{94,95} The sympathetic branch releases noradrenaline at the ciliary muscle receptor site. Although it was believed to be too slow-acting to affect accommodative dynamics, recent evidence^{99,147-149} suggests that the sympathetic system may be involved in the tracking of slow-moving stimuli, such as a low-frequency sinusoid.

The foregoing characteristics of the sympathetic system have led some researchers^{94,95,150} to speculate that its role involves sustained, rather than transient, accommodative near-point activities, especially when the parasympathetically driven accommodative levels are relatively high for extended periods of time. Such beta-inhibitory adrenergic sympathetic activation (see below) to the ciliary muscle may act to limit the amount

**Figure 4-16**

The autonomic nervous system. The sympathetic system characteristically has short preganglionic and long postganglionic neurons synapsing in the paravertebral ganglia. The ganglia use acetylcholine (ACh) as the neurotransmitter and have nicotinic receptors on the associated postganglionic neurons. The sympathetic postganglionic neurons release norepinephrine (NE), which acts on either alpha or beta receptors in the innervated structure. The parasympathetic system generally has long preganglionic neurons and short postganglionic neurons synapsing in peripheral ganglia located near the innervated structure. As in the sympathetic system, impulses are transmitted between preganglionic and postganglionic neurons via ACh acting on nicotinic receptors. The parasympathetic postganglionic neurons release ACh, which acts on muscarinic receptors in the innervated structure. (From Jose J, Polse KA, Holden EK. 1984. Optometric Pharmacology, p 48. New York: Grune & Stratton.)

of accommodative adaptation (i.e., accommodative aftereffect or hysteresis) after prolonged near work.³³ This may be of considerable importance and benefit in reducing near-work symptomatology and visual fatigue.^{94,95} In addition, abnormally low beta inhibition may play a role in the development of myopia induced by near work.^{94,96,97}

Sympathetic Receptors in the Ciliary Muscle

The sympathetic receptors in the ciliary muscle include alpha receptors, beta-2 receptors, and beta-1 receptors.¹⁴³

Alpha Receptors. Early studies¹⁵¹⁻¹⁵³ suggested that the alpha-adrenergic effect, which acted to relax the ciliary muscle, was small and indirect. Peripheral vasoconstriction reduced the volume of the ciliary muscle, which in turn acted in a mechanical manner to reduce its

effective impact on the crystalline lens. However, more recent work using isolated ciliary muscle preparations has revealed a direct pharmacological effect.¹⁵⁴ Furthermore, alpha receptor activation produces a reduction in the amplitude of accommodation^{148,155} and midrange slope of the accommodative stimulus-response function,¹⁵⁶ without any effect on tonic accommodation.¹⁵⁷ These findings can be explained by the fact that the sympathetic effect is maximal where the parasympathetic stimulation is greatest, namely, the near-point of accommodation, and therefore is minimal at the far-point and nearby tonic level.

Beta-2 Receptors. These adrenergic receptors provide the primary sympathetic stimulation to the ciliary muscle. About 90% of the beta receptors are of the type 2 variety.¹⁵⁸ However, in contrast to the alpha

receptors, these can have an influence on tonic accommodation. Gilmartin and Hogan¹⁵⁹ found that with a beta agonist, tonic accommodation decreased 0.50 D, whereas with a beta blocker, it increased 1.00 D. They may also influence the far point of accommodation and accommodative adaptation.¹⁴⁸

Beta-1 Receptors. Microdissection has shown that these adrenergic receptors comprise only 10% of the total beta receptor population in the iris/ciliary body complex.¹⁵⁸ They appear to be confined primarily to the ciliary muscle itself, with only 30% of the beta receptors here being type 1.¹⁵⁸ They therefore play a secondary role in accommodation. However, they may affect accommodative adaptation.¹⁴⁸

Parasympathetic Receptors in the Ciliary Muscle

Although several types of muscarinic (cholinergic) receptors have been reported (M_1 , M_2 , and M_3), recent evidence suggests it is the M_3 receptor that is primarily involved in basic ciliary muscle contraction.¹⁶⁰ The other two receptors are primarily involved in aqueous outflow dynamics.

Categories of Drugs that Act on the Parasympathetic and Sympathetic Branches

The four categories of drugs that act on the parasympathetic and sympathetic branches of the autonomic nervous system are as follows.^{1,144,161,162}

Parasympathomimetics. These mimic the action of the parasympathetic system and have been used in the treatment of accommodative esotropia. These can be either indirect- or direct-acting. Indirect-acting acetylcholinesterase inhibitors prevent the breakdown of acetylcholine by binding with the enzyme acetylcholinesterase, resulting in an increased amount of acetylcholine at the nerve terminals and therefore greater

activity. Examples include physostigmine (eserine sulfate), neostigmine, echothiophate (phospholine iodide), and diisopropyl fluorophosphate. Direct-acting drugs bind with the muscarinic acetylcholine receptors, thereby producing the same effect as acetylcholine itself. They also produce pupillary miosis. Examples include pilocarpine, carbachol, and methacholine.

Parasympatholytics. These antimuscarinic drugs inhibit the parasympathetic system by binding to the muscarinic acetylcholine receptors and thereby preventing acetylcholine from acting. They include atropine, cyclopentolate (Cyclogyl), homatropine (Homatropel), and tropicamide (Mydriacyl). These drugs produce mydriasis and loss of accommodation.

Sympathomimetics. These alpha-receptor agonist drugs mimic the action of the sympathetic system by either imitating (direct acting) or potentiating (indirect acting) the action of noradrenaline, primarily on the dilator muscle of the iris. Such drugs include phenylephrine (Neo-Synephrine), hydroxyamphetamine (Paredrine), and cocaine. With the exception of cocaine, instillation of these drugs results primarily in pupillary mydriasis and little, if any, cycloplegia.

Sympatholytics. These beta-receptor antagonist drugs block the action of the sympathetic system. The primary drug in this category is the nonspecific beta blocker timolol, which binds to these receptors in general and prevents their stimulation. It also produces a myopic shift in tonic accommodation.^{32,33} More recently, betaxolol has gained greater acceptance, because it is a specific beta-1 receptor blocker.

Systemic Drugs

A variety of systemic drugs can affect accommodation adversely, with most producing reduced and variable responses. The mechanisms vary and are beyond the scope of this chapter. However, a list of such drugs and their accommodative effects is provided in Box 4-5.¹⁴¹

Box

Accommodative Disorders Related to Systemic Drugs

Infacility

- Alcohol
- Artane
- Lystrone
- Ganglion blockers
- Phenothiazides
- Antihistamines
- Central nervous system stimulants
- Marijuana
- Digitalis
- Sulfonamides and carbonic anhydrase inhibitors

Insufficiency

- Alcohol
- Artane
- Lystrone
- Ganglion blockers
- Phenothiazides
- Antihistamines
- Central nervous system stimulants
- Marijuana

Excess

- Morphine
- Digitalis
- Sulfonamides and carbonic anhydrase inhibitors

Box 4-6 Disease-Related Causes of Accommodative Insufficiency

Bilateral	Unilateral
General disease	General disease
Adults	Sinusitis
Anemia	Dental caries
Encephalitis	Posterior communicating artery
Diabetes mellitus	Aneurysm
Head trauma	Parkinsonism
HIV	Wilson's disease
Multiple sclerosis	Midbrain lesions
Myotonic dystrophy	
Myasthenia gravis	
Malaria	Neuro-Ophthalmic
Typhoid	Fascicular nerve III lesion
Toxemia	Herpes zoster
Botulism	Horner's syndrome
Children	
Anemia	Local Eye Disease
Down's syndrome	Iridocyclitis
Mumps	Glucoma
Measles	Choroidal metastasis
Scarlet fever	Tear in iris sphincter
Whooping cough	Blunt trauma
Tonsillitis	Ciliary body aplasia
Diphtheria	Scleritis
Lead and arsenic poisoning	Adie's syndrome

Neuro-Ophthalmic

- Lesions in Edinger-Westphal
- Lesions in rostral superior colliculus
- Trauma to craniocervical region (whiplash)
- Pineal tumor
- Parinaud's syndrome
- Agenesis of posterior cerebellar vermis
- Polyneuropathy
- Anterior poliomyelitis

Adapted and modified from London R. 1984. Accommodation. In Barresi BJ (Ed), Ocular Assessment: The Manual of Diagnosis for Office Practice, p 123. Boston: Butterworth.

Drugs that influence accommodation are discussed further in Chapter 12.

Effects of Disease on Accommodation

A variety of disease-related peripheral and central neurological conditions, as well as systemic and ocular-based conditions, can adversely affect both the static and dynamic aspects of accommodation.¹ The mechanisms involved vary, and discussion of them is beyond the scope of this chapter. However, Boxes 4-6 and 4-7 provide a brief summary of disease-related conditions

Box 4-7 Disease-Related Causes of Accommodative Excess

Bilateral	Unilateral
General disease	General disease
Adults	Trigeminal neuralgia(s)
Encephalitis	Head trauma
Syphilis	
Head trauma	
Children	
Influenza	
Encephalitis	
Meningitis	
Head trauma	

Adapted from Scheiman M, Wick B. 1994. Clinical Management of Binocular Vision, p 359. Philadelphia: JB Lippincott.

that produce abnormalities of accommodation.¹⁴¹ In some cases, optometric vision therapy has improved accommodative function to a moderate degree; however, the use of plus lenses for near vision is the most common treatment.¹

PUPIL EFFECTS ON THE RETINAL IMAGE AND ACCOMMODATION

The pupil performs three primary functions, and each affects the quality of the retinal image.¹⁶³ The pupil:

1. Controls the entering light flux
2. Modifies the depth of focus
3. Varies the extent of optical aberrations present

In this section, each of these areas is considered in detail. See other sources for detailed anatomical descriptions of the pupil.^{7,164,165}

Light Flux

Perhaps the most prominent role of the pupil is the control of light flux entering the eye and impinging upon the rod and cone retinal elements.¹⁶⁵ The basic pupillary responses to stimuli of different light intensities are shown in Figure 4-17, A-C.¹⁶⁶ As the light intensity increases, the reaction time or latency decreases (by up to 30 msec), and the response amplitude increases. Note the equality of response in each eye. Figure 4-17, D, shows pupillary responses to prolonged step stimuli of different light intensities. In each case, there is the initial constriction, followed by "pupillary escape," or the gradual and partial redilation of the pupil without any change in the light intensity, presumably as a result of rapid retinal adaptation. Also note the pupillary unrest, or "hippus," the small oscillations in pupillary diameter that occur during maintained stimulation,¹⁶⁷ presum-

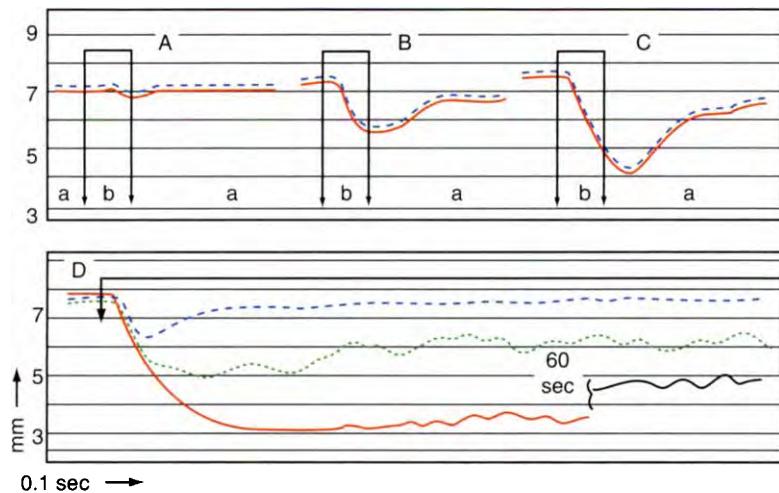


Figure 4-17

A–C, Dark-adapted normal subject. In (a), the light is off. Light flashes (b) of increasing intensity produce increasing pupillary constriction. The latent period decreases with the intensity of the flash. The right eye (solid line) was stimulated; the left eye (broken line) remained in darkness. Reactions were equal in the two eyes. D, The pupil's reaction to prolonged light of different intensities. (From Lowenstein O, Loewenfeld IE. 1959. Influence of retinal adaptation on the pupillary reflex to light in normal man. Am J Ophthalmol 48[Part II]:536.)

ably because of normal fluctuations in the sympathetic/parasympathetic equilibrium.^{163,168} It should be noted that for light,^{167,169} as well as for blur and disparity stimuli,¹⁷⁰ the pupillary system was found to exhibit an important range nonlinearity. Its response amplitude (reflecting system gain) to the same change in light intensity was greatest for midrange pupil sizes (approximately 4.0–5.5 mm), with responsivity reduced precipitously for either smaller or larger initial pupillary diameters. This has important clinical implications, because a relatively reduced pupillary response to various stimuli would be expected for normal patients with habitually small or large pupils, presumably because of mechanical, and not neurological, limitations or compromise.¹⁷⁰ Over this midrange, a normal peak velocity/amplitude relationship was found,¹⁷¹ with the peak velocity of the pupillary response being directly related to its amplitude. Overall, larger amplitude responses were faster and had proportionally higher peak velocities, reflecting the underlying neurological control properties. Pupillary responsivity was also found to display spectral sensitivity in accordance with the Purkinje shift.¹⁷²

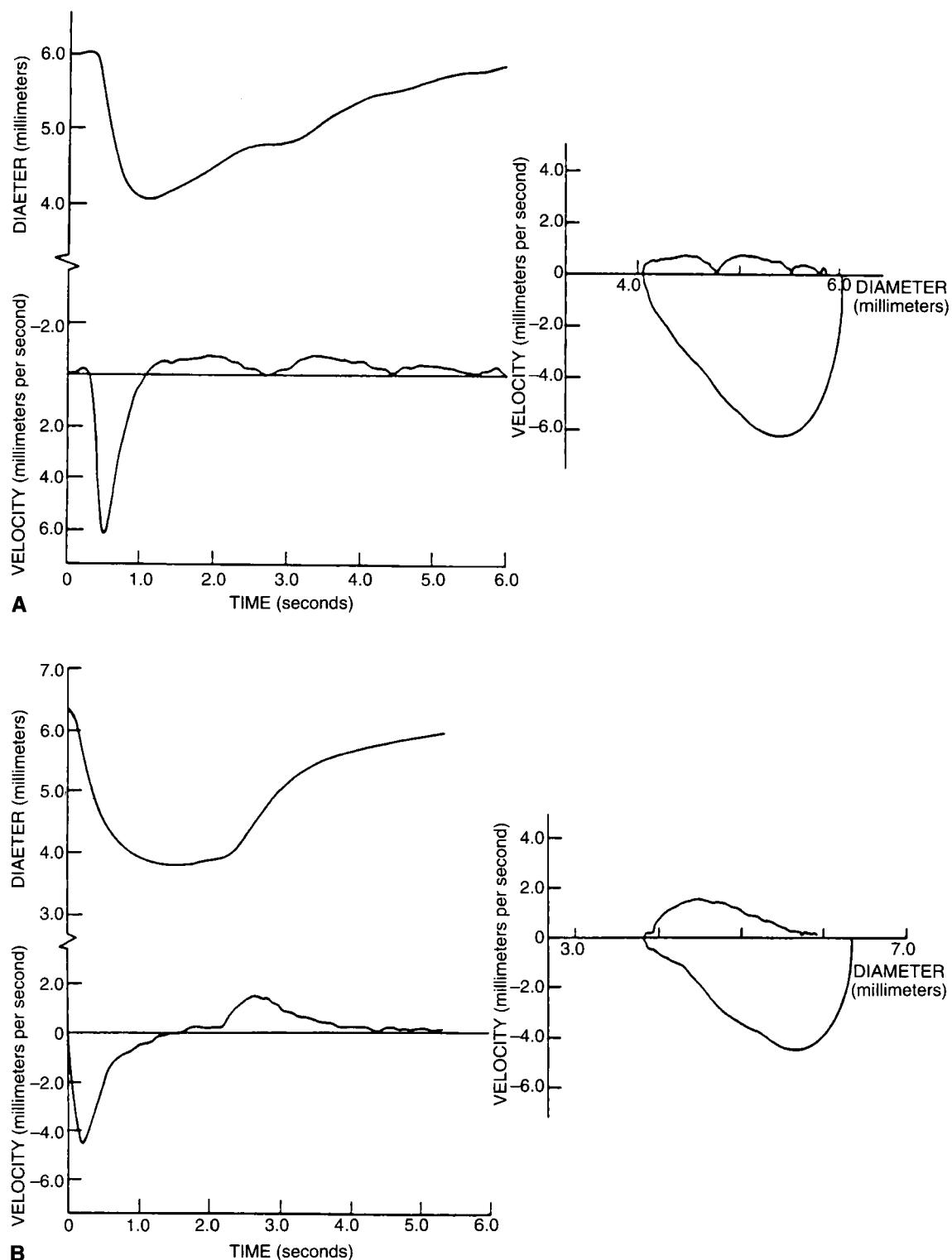
Thompson¹⁶⁸ found that the pupillary latency ranged from 180 to 500 msec, increasing as light intensity was decreased. It was also found to exhibit a statistically significant but normal age-related increase of approximately 1 msec/year, from a latency of 235 msec at 20 years old to 280 msec at 70 years old.¹⁶⁴ This age-related increase is consistent with other reaction time measures.¹⁷³ Although this latency change is important in clinical laboratory investigations involving high-speed

objective infrared pupillometers, it is too small to be detected clinically. In addition to the latency increase with age, mean pupillary diameter was shown to decrease approximately 0.3 mm/decade,^{164,174} probably as a result of increased stiffness of the iris.¹⁶⁴

Dynamic pupillary responses to light are shown in greater detail in Figure 4-18, A.^{170,175} Displayed are changes in pupillary diameter and velocity as a function of time, as well as the phase-plane plot showing the correlated changes in pupillary diameter and velocity, from which the pupillary diameter at which the peak velocity occurred (as well as other aspects of its transient dynamic behavior) can immediately be determined. Note the rapid constriction in response to the 2-second step of light intensity increase, the slow pupillary escape phenomenon occurring during the latter part of the step, and the subsequent slow redilation with its 4-second return to the original light intensity. Clearly, the initial constriction, with a peak velocity of 6 mm/sec, was much faster than either the pupillary escape or the light-off response, the peak velocity of each of which was less than 1 mm/sec. Thus, the pupil's nonlinear, dynamic, response directional asymmetry was evident. Figure 4-18, B, presents similar graphs for a blur input.^{170,175} Note the overall relative slowness of the response compared with the light response.

Depth of Focus/Depth of Field

Depth of focus is "the variation in image distance in a lens or an optical system which can be tolerated without

**Figure 4-18**

A, Averaged pupil light reflex movements to a 2-second on, 4-second off step change in light intensity from 3.5 to 4.5 log trolands presented as time plots (left) and phase-plane trajectory (right). Note the marked overshoot in the on response and the slower dilation movements. B, Averaged response of the pupil to a 2-second on, 4-second off, 6-D (1–7 D) step change in accommodative stimulation. Note the marked direction-dependent dynamic behavior. (Reprinted from Semmlow JL, Stark L. 1973. Pupil movements to light and accommodative stimulation: A comparative study. *Vision Res* 13:1087. With permission from Elsevier Science Ltd.)

incurring an objectionable lack of sharpness in focus.¹⁷⁶ Projected into free space, this dioptric interval defines the depth of field of the eye¹⁷⁷ (Figure 4-19, A). According to a simple geometrical optics model, depth of focus is inversely proportional to ocular focal length and pupil size and directly proportional to the just-detectable retinal blur circle.⁴⁵

Depth of focus can be conceptualized as reflecting a neurological tolerance for system error. Some small amount of accommodative error is thereby allowed to be present without adverse perceptual consequences. That is, a small amount of retinal defocus is tolerated without producing the perception of blur. However, after a certain point, further increase in retinal defocus

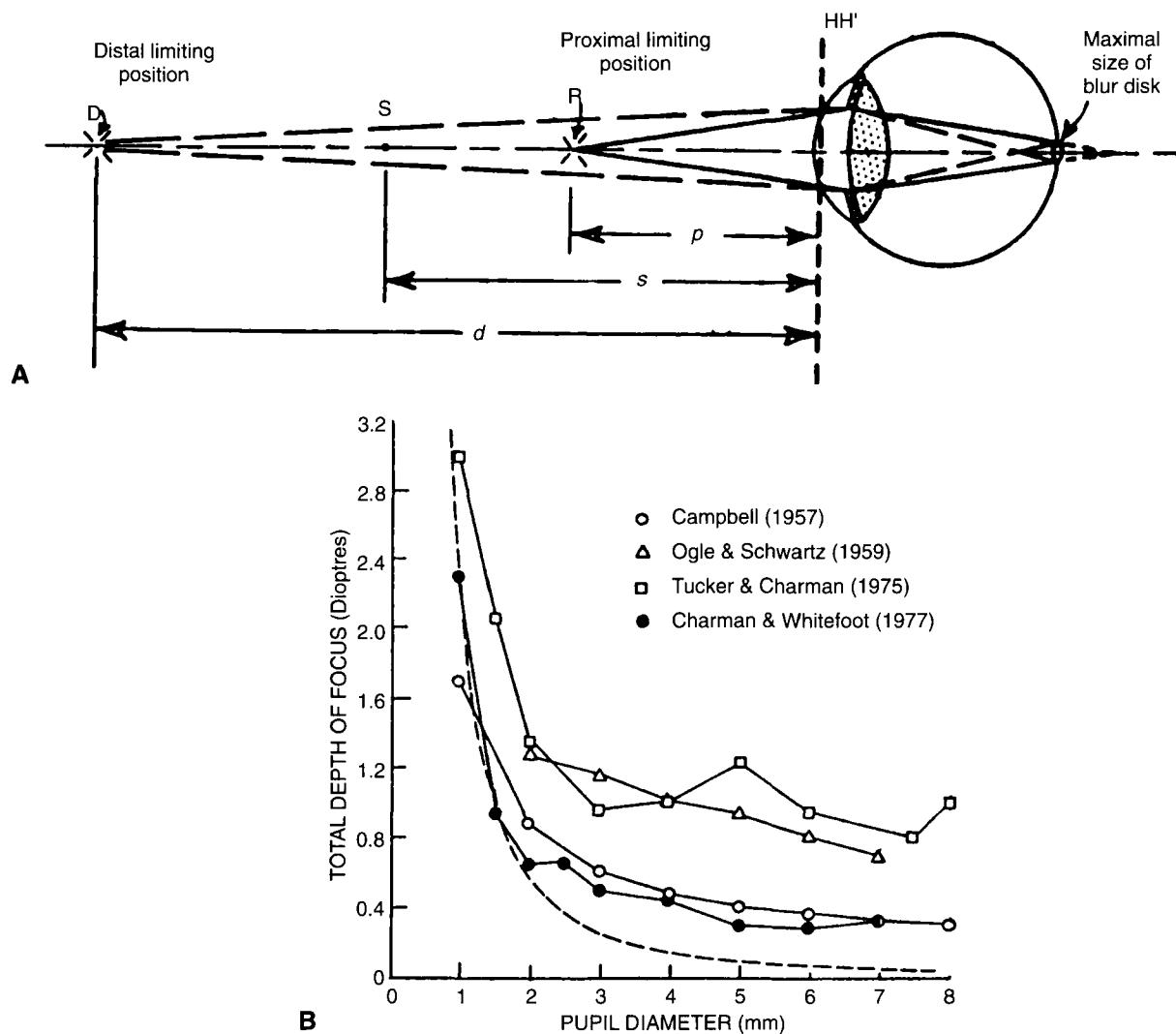


Figure 4-19

A, Depth of focus/field of the human eye. S , point in space conjugate to the retina; HH' , principal planes. $d - p$ = depth of field, or range in space over which a target can be moved and still be seen clearly with focus maintained at S . B, Examples of experimental measurements of photopic, total, monocular depth of focus as a function of pupil diameter. The optimal focus lies midway dioptrically through the total depth of focus. Campbell's⁸⁵ measurements were based on the just perceptible blur for a small disc viewed by one subject in white light. Ogle and Schwartz's³¹³ measurements were based on 50% probability of resolving a 20/25 checkerboard, and data are the mean of three subjects viewing in white light. Tucker and Charman's¹⁹⁶ measurements were based on 80% probability of achieving 90% of the optimal Snellen acuity; data are the mean of two subjects viewing in white light. Charman and Whitefoot's³¹¹ measurements were based on the detectable movements of laser speckles; data are the means of six subjects viewing in light of 633 nm. The dashed line gives the depth of focus based on Rayleigh's quarter-wavelength criterion for an aberration-free eye in monochromatic light of wavelength of 555 nm. (A, From Ogle KN. 1968. Optics: An Introduction for Ophthalmologists, 2nd ed, p 232. Courtesy of Charles C Thomas, Publisher, Ltd, Springfield, IL; B, From Charman WN. 1991. Optics of the human eye. In Charman WN (Ed), Vision and Visual Dysfunction, vol 1, p 1. London: Macmillan Press.)

results in a slightly blurry percept. If there were no such tolerance for accommodative error, and precise conjugacy of focus were lacking, a blurred percept would result. This is clearly an undesirable situation. Furthermore, the depth of focus allows for normal, small, neurological- and biomechanical-based fluctuations in system gain (as reflected in variation of response amplitude) of both blur- and disparity-driven accommodation to occur, again without the sensation of blur.

One of the best and most comprehensive studies on the depth of focus and depth of field of the human eye was conducted by Campbell.⁸⁵ Subjects judged the occurrence of the first slight blurring of black circular contours against a white background as they stimulated retinal regions about the central fovea. Under optimal test conditions, the depth of focus was approximately ± 0.30 D (for a 3-mm pupil). This value agrees well with those found in most other studies¹⁷⁸ (Figure 4-19, B), although a relatively wide range of normal values has been found—as small as ± 0.02 D¹⁷⁹ and as large as ± 1.25 D.¹⁸⁰ In addition, objective, defocus/blur-driven accommodative responses to step and sine inputs as small as 0.10 D have been recorded.^{181,182} Furthermore, Campbell found that maximum blur sensitivity was obtained for light with a wavelength of 550 nm. He also found that depth of field reduced with increases in target luminance, contrast, and pupil size, as well as with the use of an achromatizing lens.

An additional factor that can influence the depth of focus is retinal eccentricity. Eccentric retinal stimulation in the far retinal periphery (up to 60 degrees) increases it.¹⁸³ More recent work over the near retinal periphery (fovea to 8 degrees) revealed a linear increase in the depth of focus of 0.29 D per degree, with a total depth of focus of 0.89 D at the fovea progressing to 3.51 D at 8 degrees of retinal eccentricity.⁸⁶ When the results at the fovea, near retinal periphery, and far retinal periphery are combined across most of the visual field, the overall depth of focus change can be best represented by a decreasing exponential (see Figure 4-6). Based on these and other investigations on blur sensitivity (i.e., blur detection and blur discrimination) at the fovea and across the near retinal periphery up to 8 degrees,^{86,184–189} a schematic conceptualization of blur perception has been proposed (Figure 4-20).¹⁸⁷ It is represented by the spatial distribution of the dioptric depth-of-focus zone of clarity and surrounding equiblur zones, both in depth and across the near retinal periphery. The width of both zones increases with retinal eccentricity, which demonstrates the adverse effect of peripheral vision on both the blur detection and discrimination thresholds as compared with the more sensitive fovea. A target within any particular zone of these clarity/blur limits will be perceived with equal clarity or blur, respectively, and only when these limits are exceeded would the blur perception change.

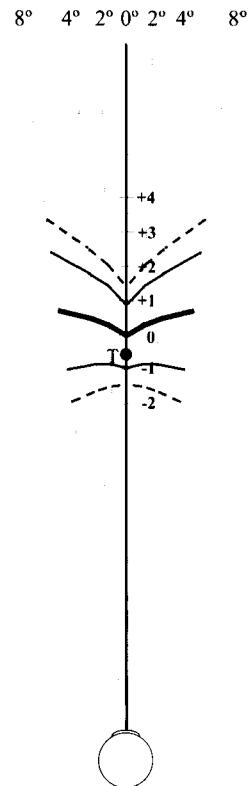


Figure 4-20

Schematic representation of the blur detection and initial blur discrimination regions in visual space. T, target; solid heavy line, zero retinal defocus plane; solid thin lines, proximal/distal limits of the blur detection region; dashed lines, proximal/distal limits of the initial blur discrimination region. Numbers represent diopters of defocus, and numbers denoted in degrees represent retinal eccentricities. (From Wang B, Ciuffreda KJ. 2005b. *Blur discrimination of the human eye in the near retinal periphery*. Optom Vis Sci 82:52.)

Knowledge regarding these equiclear and equiblur zones across the near retinal periphery has important basic and clinical implications. From a basic point of view, it provides further insight into the general area of blur perception and image processing (e.g., blur sensitivity to a naturalistic target¹⁸⁴) and target/size-dependent accommodative responsivity.¹ From a clinical point of view, it provides a better understanding of the blur-related symptoms and possible treatments for those patients with central retinal diseases (e.g., macular degeneration) in which the fovea and contiguous regions are dysfunctional and hence adversely affected.^{190,191} In addition, blur sensitivity in the retinal periphery should be taken into consideration in the design of ophthalmic lenses, especially progressive addition lenses,^{192,193} as well as in refractive surgery expectations.¹⁹⁴ In these conditions, maximizing optical quality at the edge of the near peripheral field may not be as critical as in the fovea and immediately adjacent

regions, as all aspects of blur sensitivity decline with retinal eccentricity.

Another important factor is age. The depth of focus is relatively large (at least ± 1.00 D) in a 1-month-old infant, but it reduces rapidly over the next 2 months,⁴⁵ presumably reflecting developmentally related changes (e.g., improved contrast perception) in neurologically imposed tolerances for the appreciation of blur. The depth of focus increases slightly again with advanced age, first during early presbyopia^{36,37} (see Age, Accommodation, and Presbyopia) and later due to normal, anatomically related pupillary miosis.^{168,195} It also increases with either a reduction in visual acuity demand¹⁹⁶ or a decrease in spatial frequency of a sinusoidal grating.¹⁹⁷ Jacobs et al.¹⁸⁵ found that: (1) blur thresholds depended on target size, (2) thresholds for perceived change in blur were independent of initial defocus level, and (3) the threshold for perceived change in blur was considerably smaller than the initial blur threshold value (e.g., the just-noticeable difference in blur was easier to perceive than the first blur of an initially in-focus target).

The effect of pupil size and correlated depth of focus on accommodation has been investigated. In general, the results revealed that a slight reduction in the normal slope of the static accommodative stimulus-response function first occurred at pupil diameters of 1 to 2 mm, with little or no change in mean level of accommodation after a change of several diopters in accommodative stimulus for a pupil diameter of 0.5 mm.^{196,198,199} Thus, we need to have a pupil about 0.5 mm in diameter to obtain true blur-free, open-loop viewing conditions.

There is also a static, linear interactive effect between light and blur stimuli²⁰⁰; the effect of the interaction of fusional (i.e., disparity) vergence and pupillary size is negligible (approximately 1 mm/25° of convergence)²⁰¹ and is not considered further here. The notion of a linear summation of inputs is appealing and represents an important simplifying factor, inasmuch as under isolated stimulus conditions, pupillary diameter varies directly as the log of retinal illuminance and target distance, with both effects acting on the iris muscles, which themselves exhibit a muscle length-tension nonlinearity.²⁰⁰ The experimental results are shown in Figure 4-21, A, and for comparison the linear model results are shown in Figure 4-21, B. Note the close correspondence between the experimental and modeling findings, validating the simpler linear interactive model.

Dynamic changes in the depth of focus also occur during the process of accommodation itself¹⁷⁰ (see Figure 4-18, B), resulting in a new blur-driven, steady-state pupillary diameter (around 0.25 mm/D change over the linear accommodation-pupillary region).²⁰² After the blur-driven accommodation latency (i.e., reaction time) of approximately 350 msec,²⁰³ a moderately

fast, damped decrease in pupillary diameter occurs with a latency of 400 msec in conjunction with the exponential increase in accommodation.²⁰³ This is followed by a slow redilation of the pupil when the step of blur stimulus is subsequently reduced. Moreover, similar to the light reflex (see Figure 4-18, A), but to a lesser extent, there is a nonlinear, directionally dependent, dynamic response asymmetry. These dynamic differences are especially evident in the velocity and phase-plane plots; the peak velocities are considerably higher for pupillary constriction than for dilation.

Optical Aberrations

There has been considerable work related to the classically-related aspects of optical aberrations (i.e., Seidel aberrations) and their effects on accommodation.^{177,178,204-207} In addition, more recent concepts using wavefront technology and Zernike analysis will be considered.²⁰⁸ The primary aberrations involved longitudinal spherical aberration, axial chromatic aberration, radial astigmatism, and curvature of field.

Longitudinal Spherical Aberration

Longitudinal spherical aberration (SA) refers to the lack of coincidence of focus between the off-axis (peripheral, marginal) rays and the on-axis (central) rays (Figure 4-22, A), resulting from basic geometrical optics. Typically, the peripheral rays come to a focus in front of the central rays. This is referred to as *positive* or *undercorrected* SA. In some unusual cases, the opposite, *negative* or *overcorrected* SA, is found. SA tends to produce slight symmetric blurring of an on-axis image point. The envelope of all emerging rays is referred to as the *caustic surface*, with its circle of least aberration occurring at the dioptric midpoint of the SA range. SA is considered to be the dominant monochromatic optical aberration of the human eye.

SA is measured experimentally by determining changes in focus (or basic refraction) using either annular apertures of various radii or small dual-circular apertures positioned symmetrically at various eccentricities from the center of the pupil to restrict the incoming marginal rays with respect to their distance from the central axis of the eye.²⁰⁹ The amount of SA is generally less than 1.00 D, with the contribution from the cornea occurring only for rays 2 mm or more off axis (Figure 4-23, A).²¹⁰ This is considerably less than would be expected on the basis of calculations from the Gullstrand-Emsley schematic eye, which predicts an increase in SA in a decreasing, third-order, parabolic manner, with an asymptote of 10.00 D at a 4-mm pupil radius.²⁰⁵ The empirically derived profile of SA as a function of pupil radius is displaced dioptrically (as expected) and altered in shape with increased accommodation. Variation in the shape of the curve is

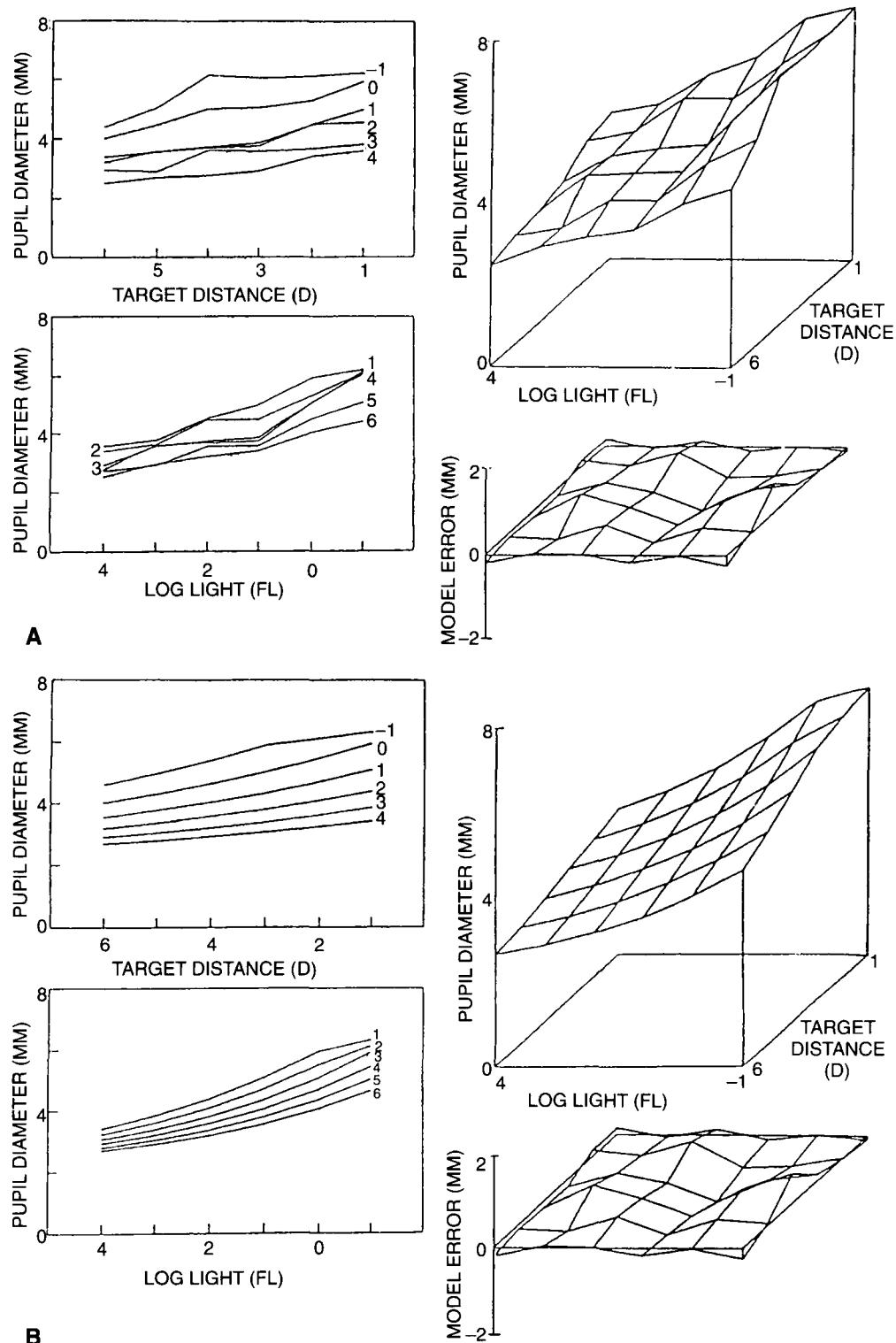
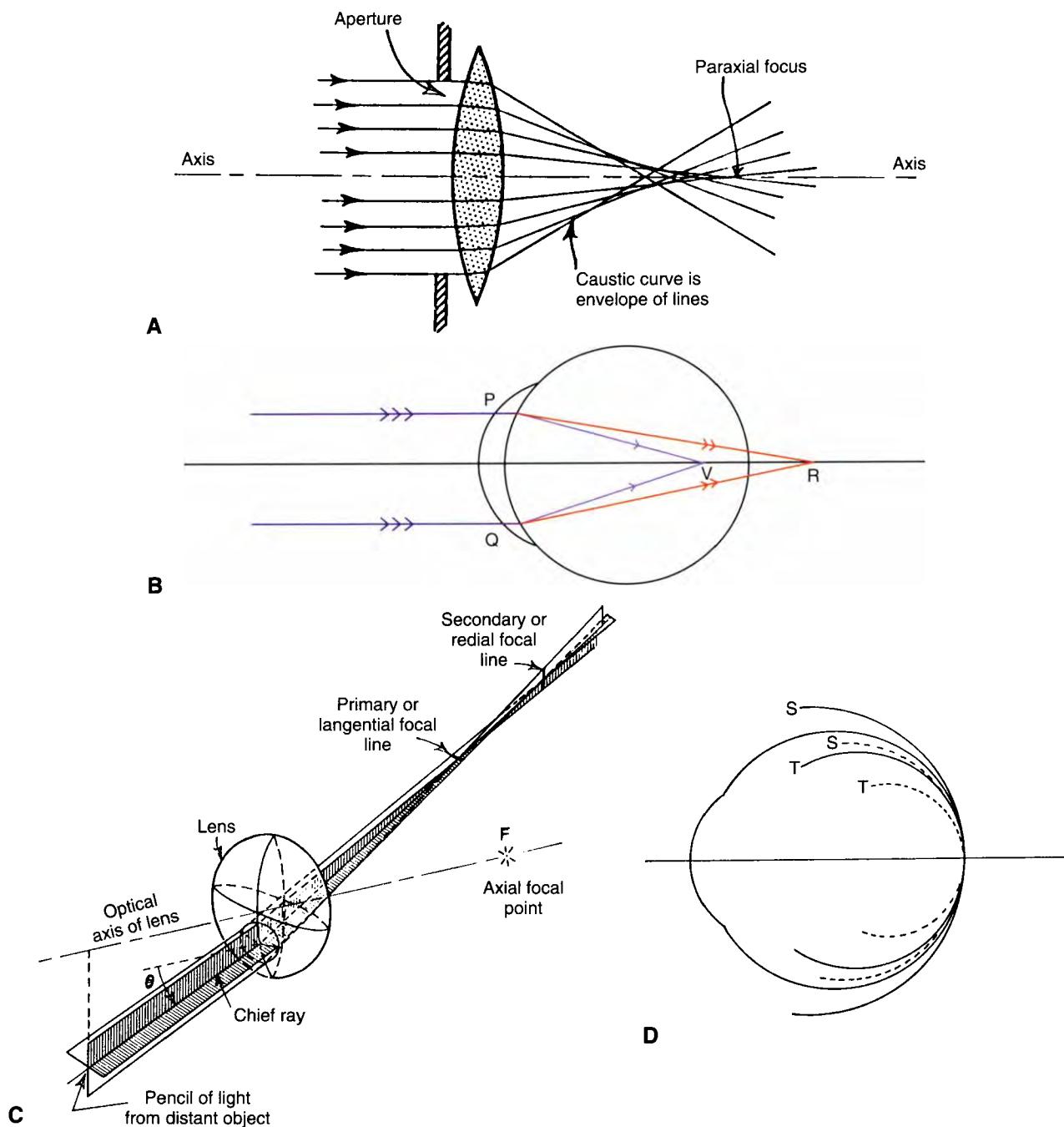
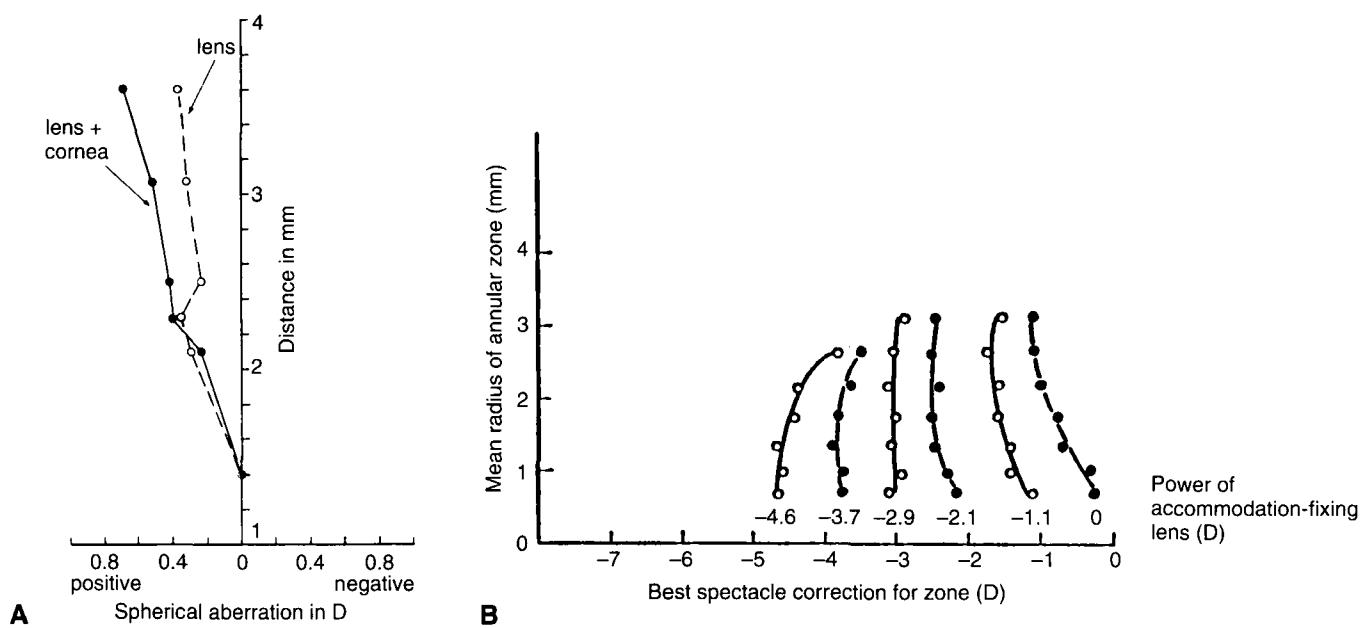


Figure 4-21

A, Pupil diameter controlled by light and target distance. The top left graph shows the pupil diameter versus target distance for a range of light levels. Top curve to bottom curve areas are $-1, 0, 1, 2, 3$, and 4 log FL (foot-Lambert). These data represent the average of three experimental runs for one subject (S.B.). The bottom left graph shows the pupil diameter versus light level for a range of target distances. The six curves are denoted according to target distance in diopters, from 1 to 6 D. The graph on the right shows pupil diameter versus light versus target distance. Although these lines sometimes touch, they do not cross. **B**, Using the parameters for subject S.B., the computer model yielded curves for pupil diameter versus target distance (top left) and pupil diameter versus light (bottom left). Combining these yields a three-dimensional control surface (top right), which shows pupil diameter versus light versus target distance. The bottom right graph shows the error, or the difference between the model and experimental results. (From Myers GA, Barez S, Krenz WC, Stark L. 1990. Light and target distance interact to control pupil size. Am J Physiol 258:R813.)

**Figure 4-22**

A, Positive spherical aberration of a lens. B, Chromatic aberration of the eye. P, Q, incoming light rays; V, violet; R, red. C, The astigmatism of rays at oblique incidence is limited to a small area of the lens. D, The sagittal (S) and tangential (T) image shells for the Le Grande theoretical schematic eye. The full lines are for the relaxed eye and the dashed lines for the accommodated eye. (A, From Ogle KN. 1968. Optics: An Introduction for Ophthalmologists, 2nd ed, p 227. Springfield, IL: Charles C Thomas; B, From Jenkins TCA. 1962. Aberrations of the eye and their effects on vision. I. Br J Physiol Opt 20:59; C, From Ogle KN. 1968. Optics: An Introduction for Ophthalmologists, 2nd ed. p 226. Charles C Thomas. Publisher, Ltd, Springfield, IL; D, From Smith G, Millodot M, McBrien N. 1988. The effect of accommodation on oblique astigmatism and field curvature of the human eye. Clin Exp Optom 71:119.)

**Figure 4-23**

A, Mean spherical aberration of the whole eye (lens and cornea) and of the lens alone as a function of the distance from the achromatic axis. B, Spherical aberration of the right eye for different levels of accommodative stimuli. (A, From Millodot M, Sivak J. 1979. Contribution of the cornea and lens to the spherical aberration of the eye. *Vision Res* 28:169. With permission from Elsevier Science; B, From Kooman M, Tousey R, Scolnik R. 1949. The spherical aberration of the eye. *J Opt Soc Am* 39:370.)

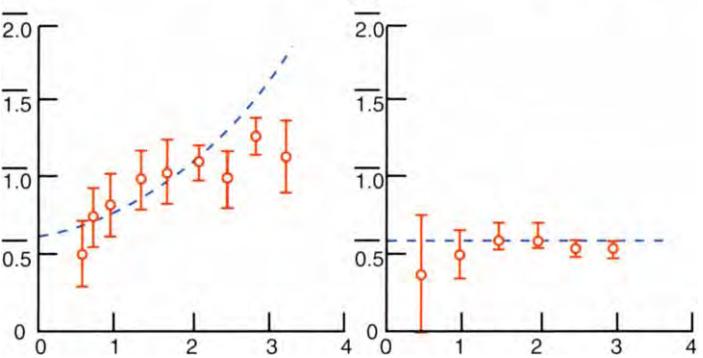
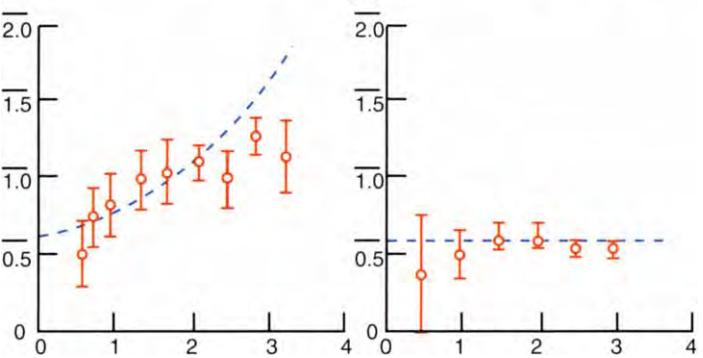
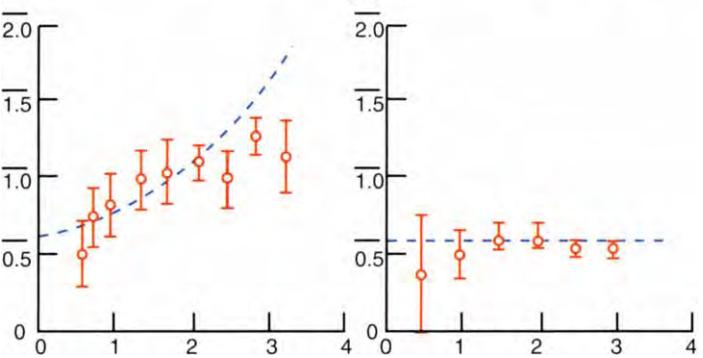
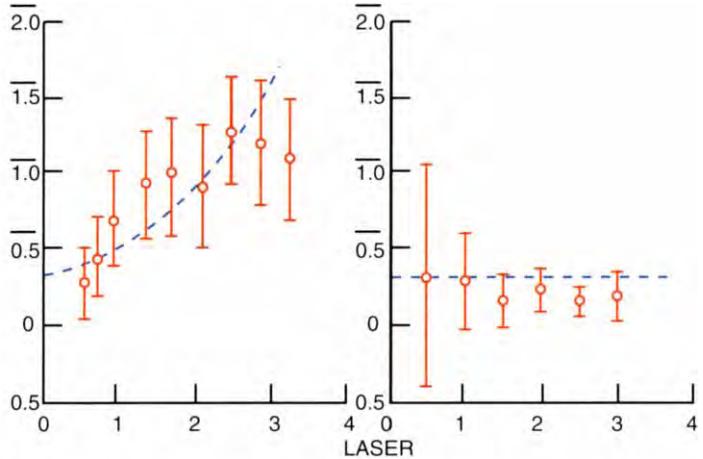
suggestive that changes of peripheral curvatures of the crystalline lens during accommodation influence the overall SA wave front^{207,211} (Figure 4-23, B). This finding of a lens contribution is consistent with the results presented in Figure 4-23, A.²¹⁰ However, it is not in agreement with the early results of Ivanoff,^{212,213} who was later shown to be incorrect.^{214–216} It is of interest that SA is invariant with pupil size (as reflected in the refractive correction) at the near accommodative stimulus level of 2.90 D, which approximates the typical distance at which near work is performed (Figure 4-23, B). Thus, any variations in pupillary diameter during near work at this distance would have minimal adverse impact on the overall quality of the retinal image.

Of what practical consequence is SA to the clinician? First, because up to 1.00 D of SA may be present with a very large pupil, the effective refraction and thus optimal focus under such conditions (as may be found during nighttime driving on a deserted road or after a dilated fundus examination) might be increased by up to 0.50 D of myopia (i.e., dioptric center of the circle of least aberration).²¹⁷ Second, some objective optometers use the peripheral portion of the pupil to determine the refractive state. This value may differ from that found subjectively, to which the more central pupillary rays provide the primary contribution.²⁰⁹ Fortunately, Charman et al.²⁰⁹ found the effect of SA on subjective refraction even to be less than predicted. Figure 4-24

shows subjective refraction results for the same individuals using annular pupils, which isolate and allow the full SA effect to manifest itself, and various-sized single circular pupils, which reflect the normal clinic test condition. With the annular pupils, the subjective refraction became progressively more myopic with increased annular radii, reflecting the progressive increase in positive SA. In contrast, with the standard circular pupils, the mean subjective refraction was essentially invariant with pupil size, although the precision was markedly decreased with the smaller pupils, because of the increased depth of focus. Charman et al. suggested that the robustness of the subjective refraction to SA probably results from three factors: (1) SA affects fine details (e.g., 20/20 Snellen letters) less than the gross details of targets, (2) the Stiles-Crawford effect reduces the "weighting" of information entering obliquely from the outer zones of the pupil, and (3) the presence of other aberrations reduces the impact of information coming from these outer pupillary zones.

Axial Chromatic Aberration

Axial chromatic aberration (CA) refers to the variation in focus with wavelength, which results from the variation in index of refraction with wavelength. That is, the refractive index of an optical system decreases as wavelength increases. The index of refraction for relatively long wavelength red light being less than that for rela-



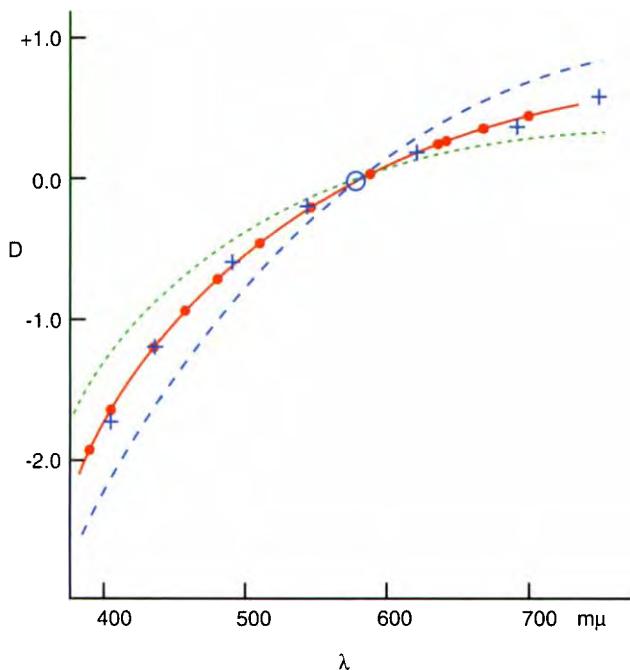


Figure 4-25

Axial chromatic aberration of the human eye. The ordinates are expressed in diopters necessary to correct the refractive error of the eye at each wavelength. The reference wavelength is 678 nm. The *solid line* through the dots is the average of 12 observers. The *crosses* are the average data obtained by Wald and Griffin.^{31,2} The *dotted lines* indicate the total range of the individual observers. (From Bedford RE, Wyszecki G. 1957. Axial chromatic aberration of the human eye. J Opt Soc Am 47:564.)

Other findings are also important. Experimental results relating accommodative level and chromatic aberration differed from those predicted by schematic eye calculations, which indicated that CA should increase by 2.4%/D of accommodative increase²⁰⁴ because of the increased power of the crystalline lens.²¹⁹ Steady-state accommodative accuracy in white light was not influenced by CA,²²¹ but dynamic accommodation tracking appeared to be slightly enhanced by the addition of such chromatic information.²²² However, given the considerable variation of the wavelength in focus on the retina at various distances,²²⁰ it is unlikely that CA plays a critical role under typical unpredictable and naturalistic viewing conditions, especially in the presence of the dominant blur stimulus.^{1,75} The effect of age on CA remains unresolved.¹⁷⁸

Of what practical consequence is CA to the clinician? Most important, it is the underlying principle in the duochrome (bichrome) test.^{74,204} Related to this, CA, along with SA, is involved in the determination of the subjective end-point distance refraction in white light. As Borish⁷⁴ stated, "The objective of accurate refraction is to reduce the diameter of the blur circle to the small-

est possible size and to place the hyperfocal curve so that the retina will intercept the narrowest portion of the caustic," with this overall on-axis caustic being due primarily to the combination of CA and SA. These aspects of chromatic aberration will be further discussed in Chapter 20.

Radial Astigmatism

Radial astigmatism (AST) refers to light's entering off axis at oblique incidence with respect to the central axis of the eye, thereby forming two separate focal lines at different distances, rather than a single focal point (see Figure 4-22, C). When light from a spherical wave front falls on a spherical optical surface in such an off-axis and oblique manner, the optical surface effectively has two different radii of curvature and thus two different orthogonally oriented powers. This produces a tangential focal line (perpendicular to the plane intersecting the optic axis and the incident ray) and an orthogonal sagittal (radial) focal line.

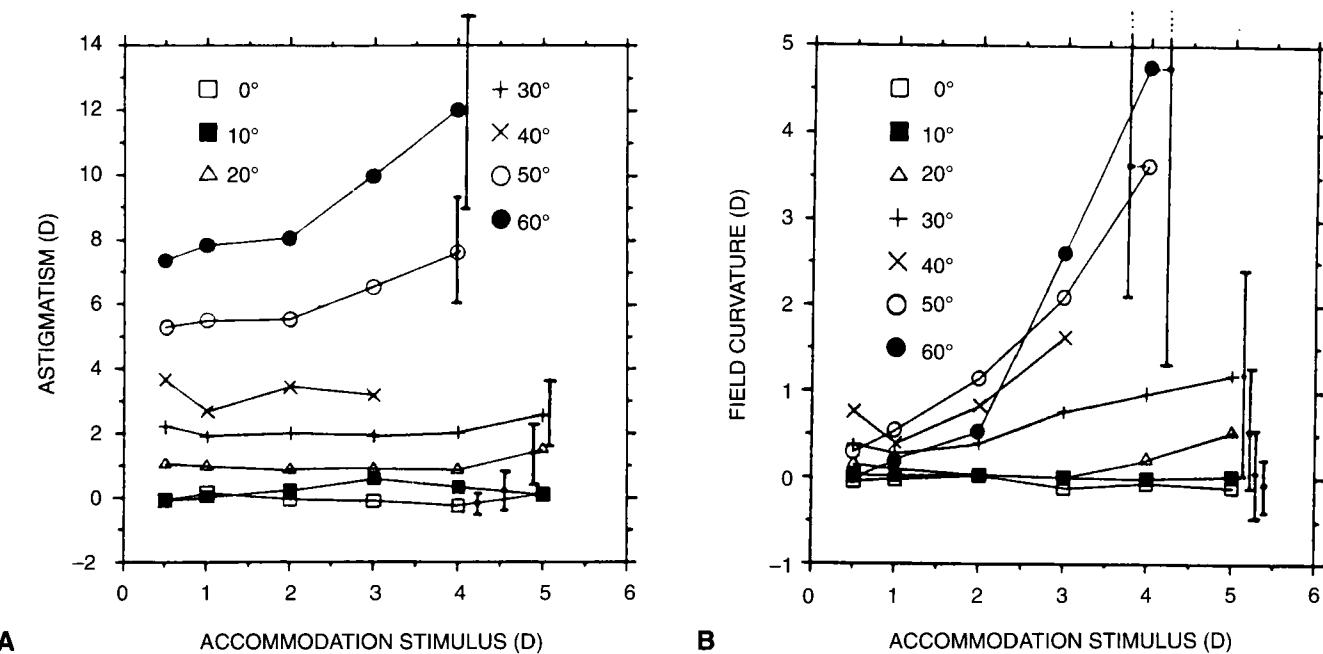
Other factors can affect AST. It increases with increased obliquity of the incident light, and Smith et al.²²³ found that it increased at the higher accommodative stimulus levels for the most oblique rays (Figure 4-26, A). Only the length of the focal lines, and not the magnitude of the astigmatic interval per se, was affected by pupil size. The greater the pupil diameter, the longer the focal line (see later).

Curvature of Field

Combining the pairs of astigmatic focal lines in AST across the entire retina forms two curved surfaces (see Figure 4-22, D). The surface formed by their dioptric midpoint, and thereby having the smallest blur circles, defines the curvature of field (CF) of the eye. Fortunately, this curved surface of minimal blur lies close to the spherical surface of the retina when accommodation is minimal^{177,223} (see Figures 4-22, D and 4-26, B). However, with increased accommodation, both the tangential and sagittal surfaces are displaced in front of the retina (see Figure 4-22, D), and thus CF increases, especially at the greatest obliquities (see Figure 4-26, B).²²³ This effect is large only for the greatest obliquities, which fortunately have the least impact because of poor peripheral neural-based visual resolution, reduced peripheral sensitivity to blur, and reduced weighting due to the Stiles-Crawford effect.^{178,183,224} In addition, visual attention to such peripheral stimuli is typically reduced. Thus, CF (and the related AST) generally pose no major problems under normal viewing conditions, including those of subjective clinical refraction.

Overall Aberration Effect

Clinicians typically use the standard Snellen visual acuity measurement as the yardstick against which they compare the influence of a parameter. Jenkins²²⁵ took

**Figure 4-26**

A, The group mean radial astigmatism as a function of accommodation for the various angles of eccentricity shown beside the symbols. Standard deviations are shown only for the highest levels of accommodation. For each angle of eccentricity, the standard deviations are approximately the same for each accommodation level. B, The group mean field curvature as a function of accommodation for the various angles of eccentricity shown beside the symbols. Standard deviations are shown only for the highest levels of accommodation. For each angle of eccentricity, the standard deviations are approximately the same for each accommodation level. (From Smith G, Millodot M, McBrien N. 1988. The effect of accommodation on oblique astigmatism and field curvature of the human eye. *Clin Exp Optom* 71:119.)

this approach to investigate the overall effect of pupil diameter on visual acuity, both with and without compensation for concurrent changes in retinal illumination (Figure 4-27). Fortunately, under the full range of pupil diameters and without compensation for related changes in retinal illumination, a situation representing natural viewing and the standard clinical test condition, variation in pupil diameter had relatively little effect on visual resolution. In contrast, when compensation was made for retinal illumination changes (i.e., retinal illumination was invariant with pupil diameter), a larger effect was observed under these unnatural viewing conditions. However, even this effect was relatively small and of little clinical consequence.

Wavefront Analysis

Recent advances in optical wavefront technology and refractive surgery have led to renewed interest in ocular aberrations and their interactions in the human eye using Zernike polynomials to assess simultaneously and quantitatively the individual aberrations, as well as their overall effect on visual performance.^{208,226} Of particular concern is how these aberrations change with variation in accommodation. Correlated changes are expected

due to predictable alterations in crystalline lens shape, position, and refractive index gradient with increased accommodation. Changes found in young adults²²⁷ included the following:

1. There was an increase in the overall aberrations.
2. With regard to the individual components, spherical aberration changed the most; it became more negative and was linearly related to the accommodative level. This finding is consistent with the fact that the crystalline lens changes more centrally than peripherally with increased accommodation.²²⁸ It is also consistent with results found in clinical cases of clinical accommodative spasm.²²⁸
3. In addition, both coma and astigmatism changed but only one-third as much as did spherical aberration. Vertical coma became more positive, and an astigmatic shift occurred in the with-the-rule direction ($-0.10\text{ D} \times \text{axis } 180$). These relatively small dioptric changes were attributed to tilt and/or vertical shift of the lens with accommodation.
4. The above changes primarily occurred at higher accommodative stimulus levels (i.e., 3 D or more).

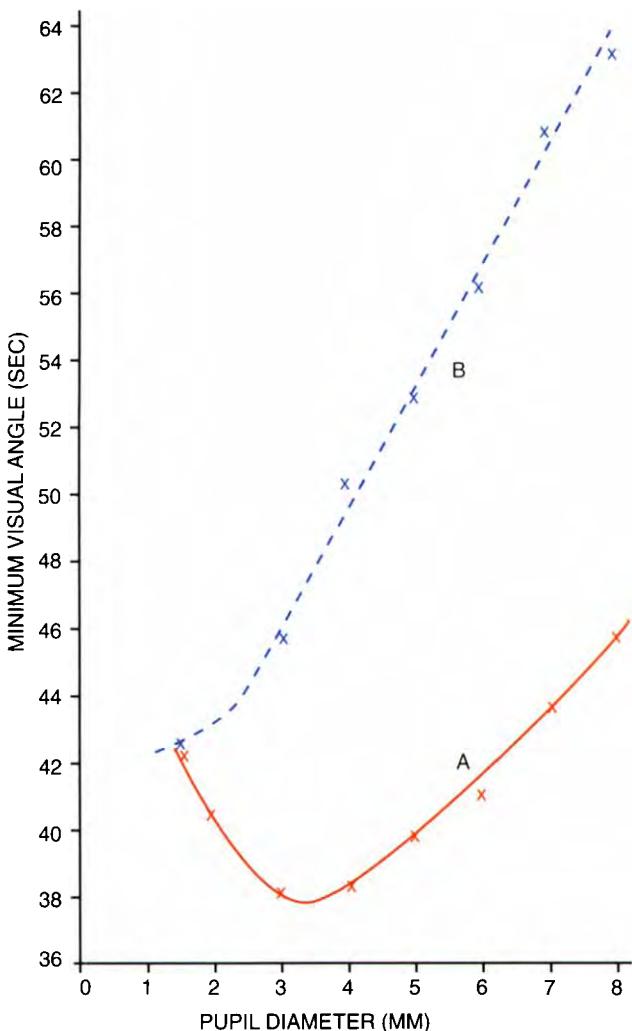


Figure 4-27

Changes in visual resolution with various pupil diameters. A shows change without compensation for retinal illumination changes; B shows changes with such compensation. (From Jenkins TCA. 1962. Aberrations of the eye and their effects on vision. I. Br J Physiol Opt 20:59. With permission from Elsevier Science.)

Other relevant findings include:

- With age, the overall aberrations increased for distance viewing, most abruptly after age 50 years, especially spherical aberration and to some extent coma. The wavefront aberrations that may change with increased accommodation as a function of age remain unknown.^{229,230}
- During dynamic changes in accommodation to a ramp stimulus (0–2.5 D over a 3-second period), many of the aberrations altered continuously, especially spherical aberration and coma.²³¹
- Microfluctuations in these wavefront-based ocular aberrations were not related either to the concurrent accommodative microfluctuations or to versional/translational eye movements. They were

speculated to be related either to dynamic changes in tear film thickness or to lens instabilities, or perhaps they may simply be a methodological artifact.²³²

- The shape of the retinal image changes slightly with increased accommodation, and this has been attributed to small changes in spherical aberration and coma.²³³

In addition to the crystalline lens-based aberration changes that occur with accommodation, correlated alterations in corneal shape occurred due to the biomechanical effects of the ciliary muscle forces on the cornea.²³⁴ Such anterior corneal-based changes affect spherical aberration and coma. Most interestingly, the accommodatively-derived, lens-based changes in aberrations may act to compensate for the corneal-derived ones,²²⁶ but this remains to be fully tested. The corneal aberrations did not change with age.²³⁰

AGE, ACCOMMODATION, AND PRESBYOPIA

Amplitude of Accommodation

The amplitude of accommodation represents the maximal accommodative level, or closest near focusing response, that can be produced with maximal voluntary effort in the fully corrected eye.¹ Clinically, it is measured from infinity to the nearest point of subjective clear vision with maximal accommodation expended, without compensation for the depth of focus (Figure 4-28; see also Figure 4-5). However, both theoretically and experimentally, it should be measured from the far point (the farthest point conjugate to the retina with exertion of minimum accommodation) to the near point (the closest point conjugate to the retina with exertion of maximal accommodation), incorporating appropriate compensation for the depth of focus at both focal extremes and thus effectively reducing its inflated clinical estimate by approximately 0.50 to 1.00 D in patients with normal vision. In patients with vision abnormalities such as amblyopia¹³² or macular disease,¹⁹⁰ the depth of focus is greater because of neurosensory insensitivity and, therefore, a larger compensation is warranted. On the accommodative stimulus-response curve (see Figure 4-4), the accommodative amplitude represents the dioptric difference between the actual response minimum and maximum, or the farthest to the nearest point of clear vision with the target conjugate to the retina.

From around 5 years of age^{55–57} to around 52 years of age,^{36,37,58,74,235,236} the accommodative amplitude progressively decreases at a rate of approximately 0.30 D/year²³⁷ (Figure 4-29 and Table 4-3). Thus, at age 10 years it is 13.50 D,⁵⁸ whereas at approximately 52 years

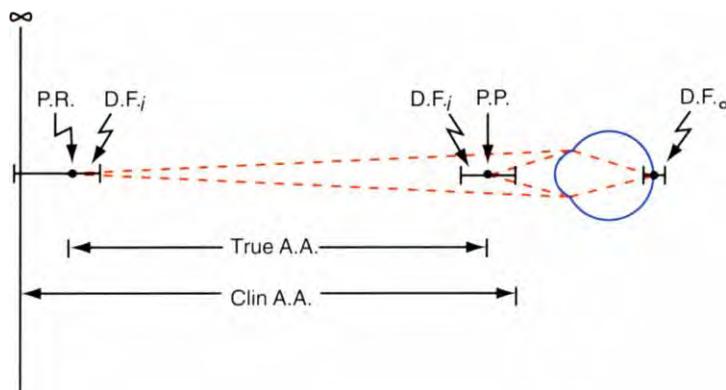


Figure 4-28

True versus clinical accommodative amplitude. Note that the depth-of-focus ranges at the near and far points are equal dioptrically but unequal linearly in free space because of the nonlinearity of the dioptric unit. AA, accommodative amplitude; DF_i , depth of field; DF_o , depth of focus; PR, punctum remotum; PP, punctum proximum. The figure is not drawn to scale.

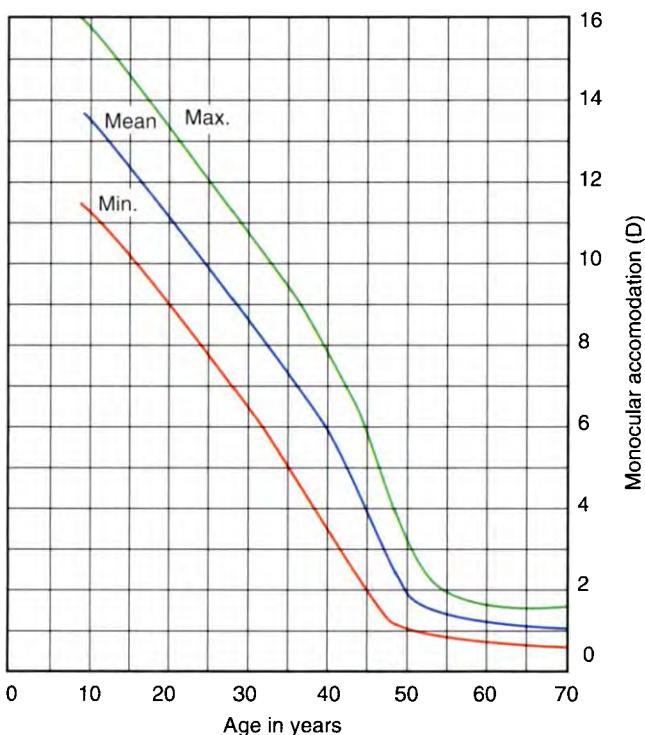


Figure 4-29

Variation of amplitude of accommodation with age. Monocular values are referenced to the spectacle plane.⁵⁸

of age it effectively becomes zero, with the apparent residual 1.00 D really reflecting the eye's depth of focus.²³⁸ Indeed, the amplitude changes with age are so predictable that reasonable clinical prescribing guidelines for presbyopia have been developed^{74,239,240}; tentative near adds at ages 45, 50, and 55 years are 1.00 D, 2.00 D, and 2.50 D, respectively.

It has been speculated²⁴¹ that various age-independent, lens-related factors, in combination with the normal complement of age-dependent lenticular factors, create a dual impact on the accommodative amplitude, which may explain why it declines so precipitously with age compared with all other physiological functions and biological system components²⁴² (Figure 4-30). These other functions do not decline or extrapolate to zero until ages 80 to 400 years.^{243,244} Most vision-related parameters decline to zero (by extrapolation) at approximately 120 years of age.²⁴⁴

There are a variety of other factors that should be considered with regard to the accommodative amplitude (Table 4-4). First, accommodative amplitude should be measured both monocularly and binocularly while the patient is viewing near threshold-sized, high-contrast test letters. The monocular value should be more or less equal in each eye (<0.25 D difference),⁵⁷ whereas the binocular value should be about 0.50 D greater (at least in children and young adults with considerable residual accommodation) because of the addition of vergence-accommodation.⁵⁸ Letter size does not affect the measured response in perceptive adults²⁴⁵ when specifically instructed to attend to an edge or border and judge when the first slight, sustained blur occurs that cannot be cleared with additional exertion of voluntary accommodative effort (e.g., the appropriate blur end-point criterion). Small letters (e.g., just above the visual acuity threshold) should be used with children and many adults who may interpret the word *blur* as meaning the inability to read the test letters, that is, total or near total blur out. Clearly, the accommodative amplitude would be grossly inaccurate and considerably inflated if the second criterion were used, especially if the test were performed with large letters. Thus, any type of "blur out" criterion is meaningless and should not be used.

TABLE 4-3 Comparison of Different Investigators' Age-Related Amplitudes of Accommodation

Age (yrs)	Donders	Duane	Jackson (binocular)	Sheard	Turner
10	19.70	13.50	14.00		13.00
15	16.00	12.50	12.00	11.00	10.60
20	12.70	11.50	10.00	9.00	9.50
25	10.40	10.50	9.00	7.50	7.90
30	8.20	8.90	8.00	6.50	6.00
35	6.30	7.30	7.00	5.00	5.75
40	5.00	5.90	5.50	3.75	4.40
45	3.80	3.70	4.00		2.50
50	2.60	2.00	2.50		1.60
55	1.80	1.30	1.25		1.10
60	1.00	1.00	0.50		0.70

From Borish IM. 1970. Clinical Refraction, 3rd ed, p 172. Chicago: Professional Press.

TABLE 4-4 Comparison of Push-Up and Minus-Lens Amplitudes of Accommodation

Push-Up ^a	Minus Lens
1. Retinal-image size increases greatly (up to 400%).	1. Retinal-image size decreases slightly (up to 10%).
2. Retinal-image size increases up to 3% due to the optics of accommodation.	2. Retinal-image size increases up to 3% due to the optics of accommodation.
3. Proximal stimulation to accommodation increases.	3. Proximal stimulation to accommodation remains constant.
4. Target change is more natural.	4. Target change is less natural.
5. Pupil size decreases.	5. Pupil size decreases.
6. Stimulus change is continuous.	6. Stimulus change is discrete.

^aThe same arguments could be made for the amplitude of accommodation as measured by dynamic retinoscopy.

adults by up to 0.60 D (<10% effect) when the test target presents both blur and large size increases (e.g., as are used in the push-up amplitude technique), as opposed to blur increases alone (e.g., as is done in the minus lens amplitude technique with relatively little lens-induced target minification),²⁴⁶ thus demonstrating slight response enhancement with cue addition.

Third, target velocity should be relatively slow (around 0.50 D/sec) to produce a smooth and continuous change in the accommodative response.¹⁰⁷ This is especially important as the target approaches the patient's eye, because diopters are nonlinear units that increase progressively more rapidly with constant linear inward movement. Therefore, the clinician should gradually reduce the linear speed (in centimeters per second) of the advancing target movement to keep the rate of the dioptric increase (in diopters per second) relatively constant.

Fourth, accommodative amplitude varies with the gaze angle of the eye,²⁴⁷⁻²⁴⁹ generally being greatest with the eye positioned down and in and least with the eye positioned in an upward gaze. This difference has been reported to be as large as 3.50 D. However, if the gaze-dependent difference in amplitude is due only to a slight forward movement of the crystalline lens as a result of gravity in the fully accommodated state, when the zonules are under the least tension, it should only be a few tenths of a diopter, as recently found by Atchison et al.²⁴⁷ Thus, it appears that other, as yet unknown, factors are involved. It seems logical and most functionally relevant to measure accommodative amplitude in both the normal reading position and the traditional clinical primary gaze position, for purposes of comparison and standardization.

Fifth, the amplitude may appear to be less in some very young children (<10 years of age)⁵⁷ than that pre-

Second, the static proximal accommodative contribution is small (around 4%) and equal under monocular and binocular test conditions,²³ because blur feedback and depth of focus both dominate and therefore limit the possible response range. Related to this, the monocular amplitude may be increased in young

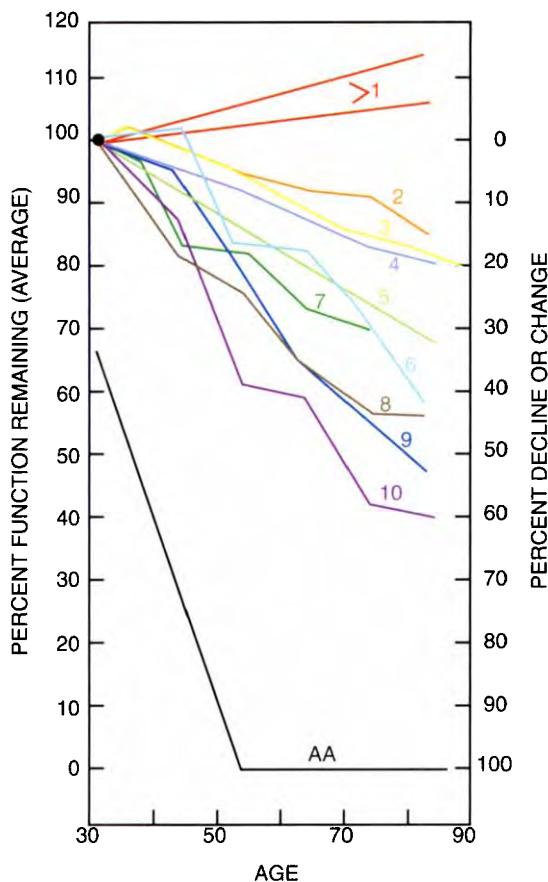


Figure 4-30

Changes in several physiological functions and one behavioral function in humans from 30 to more than 80 years of age. The amplitude of accommodation (AA) has been added to the original graph. 1, blood pressure (systolic and diastolic); 2, conduction velocity; 3, basal metabolic rate; 4, standard cell water; 5, hand grip strength; 6, glomerular filtration rate; 7, cardiac index; 8, vital capacity; 9, renal plasma flow; 10, maximal breathing rate. (Adapted from Ordy JM. 1975. Principles of mammalian aging. In Ordy JM, Brizzee KR [Eds], Neuropathology of Aging, p 12. New York: Plenum Press.)

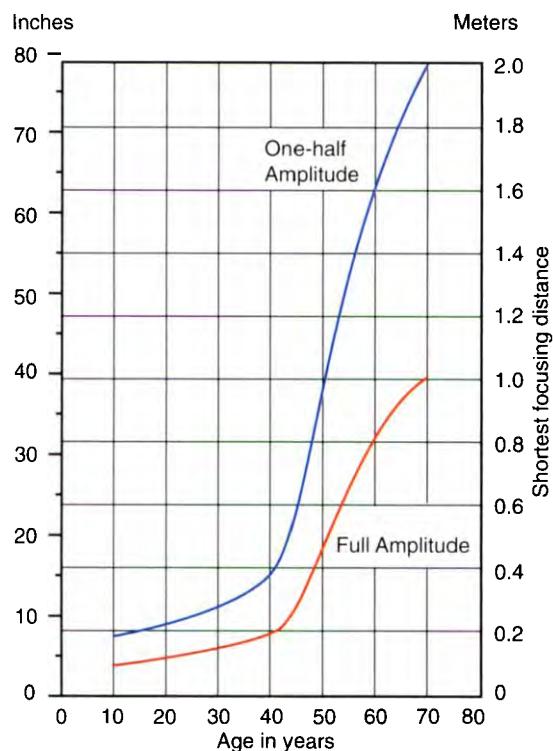
sented for 10-year-olds in most tables (see Table 4-3). Although this could be due to failure of mature accommodative development, it is more likely related to children's lack of full understanding of the concepts of slight blur and exertion of full voluntary effort, as well as their motivational and attentional factors. As discussed earlier, the amplitude is actually greater in these younger children and forms an upward continuum with the classic Duane's curve.^{58,250}

Finally, other factors, such as markedly reduced target illumination and poor testing technique, obviously would result in significant measurement error, as well as increased response variability.¹

Overview of Presbyopia

Presbyopia ("aged eye") refers to the slow, normal, naturally occurring, age-related, irreversible reduction in maximal accommodative amplitude (i.e., recession of the near point) sufficient to cause symptoms of blur and ocular discomfort or asthenopia at the customary near working distance. Essentially, the near point approaches and then becomes coincident with the far point. Presbyopia is generally first reported clinically between 40 and 45 years of age, with its peak onset between ages 42 and 44 years,²⁵¹ although its onset may occur any time from 38 to 48 years of age, depending on a variety of factors. From approximately age 52 years on, the prevalence of presbyopia is considered to be essentially 100%; however, its prevalence across all ages in the population is 31%.²⁵¹ Although there are a variety of potential risk factors for the development of presbyopia,²⁵¹ two are particularly important:

1. **Refractive error.** Because accommodative demand at the corneal plane in spectacle-corrected hyperopes is greater than that in myopes for the same accommodative stimulus and degree of ametropia at the spectacle plane (see Chapters 26 and 28), spectacle-corrected hyperopes exhibit apparent relatively reduced accommodative amplitudes and thus effectively become presbyopic a few years earlier than either myopes or emmetropes.²⁵²
2. **Ambient Temperature.** With the eyeball being peripheral to the body core, it may exhibit considerable surface temperature variations because of the influence of ambient temperature. There is an inverse relation between ambient temperature and age of onset of presbyopia.²⁵³ Clinically, when the near-work distance dioptrically equals half of an individual's residual accommodative amplitude, which occurs, on average, at 40 years of age²⁰⁴ (Figure 4-31), the gradual onset of symptoms will become manifest.²⁵⁴ These symptoms are as follows^{74,255,256}:
1. Vision at the customary near-work distance is blurred or can be sustained only with excessive accommodative effort and some ocular discomfort.
2. Drowsiness after a short period of reading or near work.
3. Reading material must be held farther away (e.g., closer to the receding near point and surrounding depth of field) to be seen more clearly. Thus, on average, smaller individuals with proportionally shorter arms develop presbyopic symptoms at an earlier age than do age-matched but proportionally taller persons. Some patients may actually complain, "My arms aren't long enough to see up close anymore." What they are describing is the fact that they can no longer keep the object of interest

**Figure 4-31**

Shortest linear focusing distances corresponding to exertion of either all or half of the available mean amplitude of accommodation as a function of age. (From Bennett AG, Rabbets RB. 1989. Clinical Visual Optics, 2nd ed, p 141. Boston: Butterworth-Heinemann.)

within the proximal edge of the depth of focus of their progressively receding near point.

4. Occasionally, especially in very early or incipient presbyopia, asthenopia related to attempts at excessive accommodative effort is reported. It may even lead to an accommodative spasm and pseudomyopia.
5. Transient diplopia and variable esophoria may be experienced as a result of the increased accommodative response/effort and the consequent synkinetically overdriven accommodative convergence that may be difficult to control consistently using compensatory negative fusional vergence.^{27,28}

Analysis of the Biological Components of Presbyopia

A variety of biomechanical, biochemical, and physiological factors contribute to the age-related loss of accommodation and the onset of clinical presbyopia^{12,15,163,195,257-264} (Figure 4-32). The following findings on the biological components of age-related changes in accommodation are summarized in Table

TABLE 4-5 Participation of the Various Lenticular and Extralenticular Components in the Age-Related Loss of Accommodation

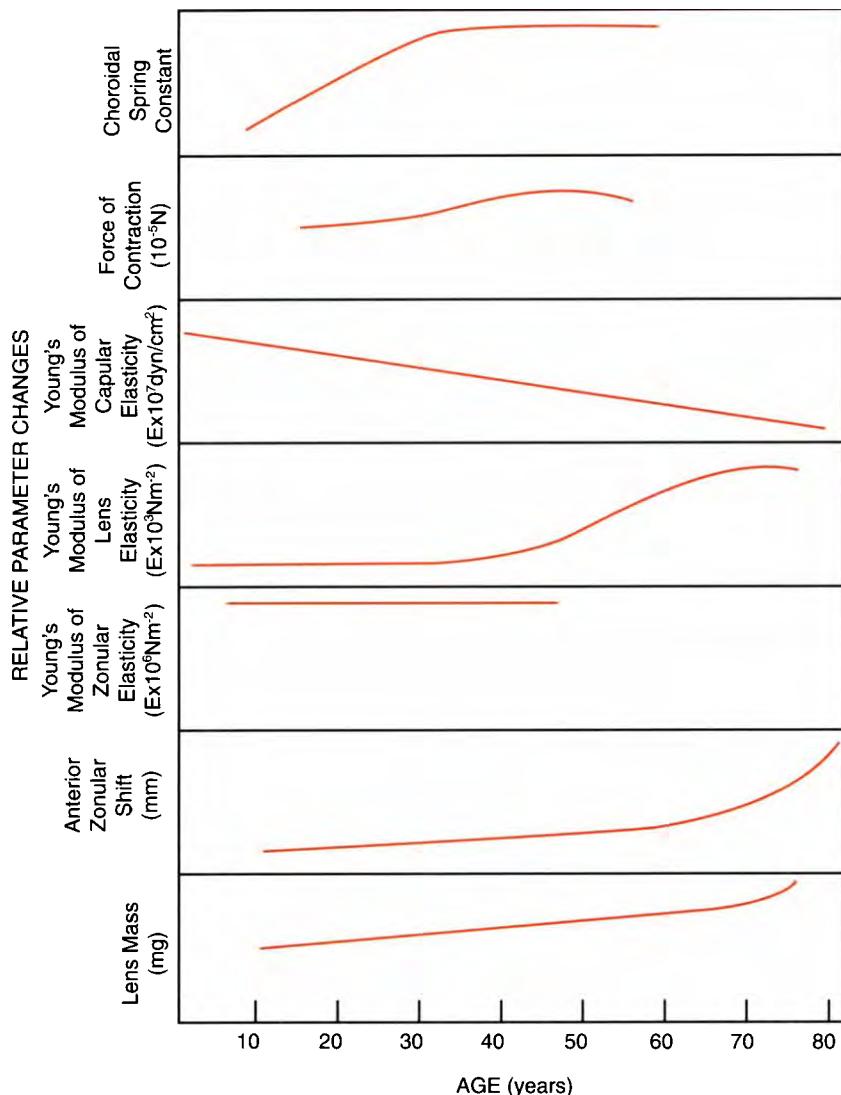
Component	Participation
Lenticular	
Lens capsule	Yes
Lens substance	Yes
Lens size/volume	Yes
Disulfide bridges	Yes
Extralenticular	
Zonular shift	Yes
Zonular fragmentation	Yes
Zonular elasticity	No
Ciliary muscle force	No
Ciliary muscle anatomy	Yes
Choroidal elasticity	Yes

4-5. Various aspects of the lens and capsule alone produce most of these changes.

The Three Primary Factors

Three factors have traditionally been considered the primary factors contributing to the age-related loss of accommodation.

1. The modulus of the elasticity (i.e., springiness) of the lens capsule decreases from youth to old age.²⁶⁵ It becomes progressively less stiff and more compliant, like an old, stretched-out rubber band. Thus, the energy it can store and then release to deform/mold the underlying lens substance (primarily the lens cortex) uniformly and thereby increase accommodation decreases with advancing age. The effective capsular energy is proportional to the loss of accommodation up to age 45 years.²⁶⁶
2. The modulus of the elasticity of the lens substance increases slightly from youth to age 40 years and more precipitously thereafter.²⁶⁷ The lens substance becomes stiffer, more plastic (like putty), and more sclerotic-like with advancing age. However, it should be noted that no true sclerosis in the physiological sense occurs, because the lens' overall water content remains constant with age.²⁶⁸ Thus, the energy required to deform the lens substance itself increases with age, with this energy being proportional to the modulus of elasticity. Fisher²⁶⁷ asserted that this factor contributed to 44% of the loss of accommodation.
3. The lens size/volume increases progressively with age.^{195,269} This makes the lens capsule function less

**Figure 4-32**

Changes in lens and ciliary muscle parameters as a function of age.

effectively. It is more difficult to deform a larger body than to deform a smaller body, especially with the concomitant change in shape. Fisher²⁶⁶ maintained that the increased lens size/volume and the decreased elasticity of the lens capsule contributed to 55% of the loss of accommodation.

Other Factors

Other factors may also contribute to the age-related loss of accommodative ability. An anterior shift of the equatorial fibers²⁷⁰ occurs because of the passive movement of the lens capsule with increased lens growth. Because the zonules are fixed within the lens capsule, as the lens increases in size and volume, it places force on the capsule that passively pulls the zonules forward. This change in zonule/capsule/lens geometry reduces the mechanical advantage of the zonular suspensory

system. A vector force applied perpendicularly to a load (e.g., the zonular equatorial fibers attached more or less perpendicularly to the young capsule/lens unit) is more effective than the same force applied tangentially (e.g., the now anteriorly shifted and tangential "equatorial" zonular fibers attached to the stretched, older capsule/lens unit).²⁷¹ However, this becomes a factor only in the mid-40s, when there is relatively little residual accommodation.

The equatorial zonular fibers decrease in number, become less dense, and appear to be more fragmented,^{270,272} which reduces their biomechanical advantage somewhat. This, too, occurs relatively late in life.

There is an increase in the number of disulfide bridges in the lens capsule and lens.²⁷³ These bridges stabilize the collagen molecules within the capsule and lens by the process of crosslinking. However, this

produces more resistance between the lens fibers themselves during accommodation, with the result that the lens becomes more rigid and difficult to deform. It also makes the capsule less elastic.

Recent work has shown that some changes in specific aspects of the ciliary muscle anatomy, including overall decreased length of the ciliary muscle, may contribute as much as 20% to 33% of the accommodative loss from age 30 to 50 years.^{274,275} However, other aspects remain constant (the width of the ciliary muscle) or even increase (the area of the circular portion of the ciliary muscle). Thus, it appears that some loss of accommodation with increased age may be attributed to gross anatomical and mechanical changes in the ciliary muscle. There is also some reduction of inward and forward movement of the entire ciliary muscle and ciliary muscle ring with age.²⁷⁴ Such a static shift in effect reduces the amount of accommodation that can occur, because it reduces the residual amount of ciliary body movement that is allowed to take place. In addition to normal lens growth, such a shift produces a more rounded and higher front lens radius of curvature during accommodation at far distances. This change in lens curvature may be expected to produce myopia at far. However, a concurrent decrease in the effective index of refraction compensates for any curvature-related power change (e.g., the "lens paradox"), making the distance refractive error with increased age relatively stable.²⁷⁶

Finally, choroidal elasticity progressively stiffens up to age 35 years, with a slower rate of increase in its modulus of elasticity thereafter.^{264,277} The choroid may be thought of as a spring that acts against the inward and forward pulling of the ciliary muscle during increased accommodation. Thus, the ciliary muscle needs to exert a slightly greater force with age to produce the same resultant dioptric change, which would be expected to produce a small increase in the response AC/A ratio with age as most recent studies demonstrate (see below). The choroid and the posterior zonules also act to restore passively the position of the ciliary body upon reduction of its contraction.

Components That Do Not Contribute

The three components that do not seem to contribute to the age-related loss of accommodation are described below.

The zonular elasticity remains constant.²⁷⁸ Thus, as the ciliary muscle contracts and moves both forward and inward with increased accommodation, releasing zonular tension, the forces per se transmitted from the zonules to the capsule/lens body remain the same throughout life. (However, recall that the biomechanical advantage of the equatorial zonular fibers is somewhat reduced later in life.)

The force/contractile power of the ciliary muscle increases (a maximum of 50%) from youth to age 45

years and exhibits a slight decline from age 45 to 60 years the period in which the accommodative loss is 100%.^{279,280} Presumably, the increased muscle force is necessary to overcome the increased choroidal antagonistic restoring force. Such an increase in force/innervation to the ciliary muscle should produce an increase in the response AC/A ratio with age in this time period. Indeed, the response AC/A ratio appears to increase by up to 50% from age 20 to age 45 years, after which it is too unreliable to measure because of the small or even absent changes in accommodation with increased age.²⁸¹⁻²⁸³ However, most of this occurs between the ages of 35 to 45 years.²⁸² It has been estimated that the ciliary muscle contractile force should not be zero until 120 years of age.²⁷⁴

Finally, the accommodative motoneuronal controller signal and related neural pathways/structures remain relatively constant over this time period.³⁶⁻³⁸

Analysis of the Model Components of Presbyopia

Age-related changes in accommodation can also be considered in terms of static and dynamic component contributions using bioengineering models and control systems concepts.^{72,90,112,284} These model components have, in a general sense, actual physiological analogues and therefore are homeomorphic.

The following analysis of the model components of the age-related changes in accommodation is summarized in Table 4-6. Not all components demonstrate age-related change. This underscores the value of component analysis, which allows "dry dissection" of the individual parts of the system, rather than requiring a global analysis that would combine affected and unaffected components, resulting in an overall diluted response measure.

Much of the experimental model-based findings contradict earlier results.^{285,286,287} However, earlier researchers used accommodative stimulus levels that exceeded the age-related linear response region of older subjects, and thus unwanted response saturation effects and other artifacts (e.g., slowed accommodative dynamics) contaminated their findings.

Static Components

The statuses of the seven static components^{36-38,72,90,284} (see Figure 4-8) with age are as follows.

Tonic Accommodation. Tonic accommodation decreases approximately 0.04 D/year, from around 1.80 D at age 20 years to 0.90 D at age 50 years, for a decrease of 50%.^{36,37} This is probably the result of subtle biomechanical limitations of the lens and lens capsule in response to the constant (i.e., age independent) neural innervation to the ciliary muscle. The aging biomechanics progressively restrict the amount of

TABLE 4-6 Participation of the Static and Dynamic Model Components in the Age-Related Loss of Accommodation

Component	Participation
Static Model	
Tonic accommodation	Yes
Depth of focus	
Objective	No
Subjective	Yes
Accommodative controller gain	No
Accommodative amplitude	Yes
Accommodative adaptation	Yes
Response AC/A ratio	Yes
Stimulus CA/C ratio	Yes
Dynamic Model	
Latency	Yes
Time constant	No
Peak velocity/amplitude relation	No
Microfluctuations	Yes

AC/A, Accommodative convergence/accommodation ratio; CA/C, convergence accommodation/convergence ratio.

"rounding up" of the lens in response to this constant tonic neural signal.

Depth of Focus. The mean objectively determined depth of focus (± 0.38 D) remains relatively constant over this same time period.^{36,37} This suggests that the blur-driven, reflexive accommodation described by Fincham¹⁹ does not become less sensitive with age. In contrast, subjectively determined depth of focus, which was always greater than its objective counterpart, as others have also demonstrated,¹⁸¹ progressively increases from ± 0.40 D to ± 0.90 D over this same time period.^{36,37} Concurrent age-related reduction in pupil size could only account for 30% of this effect.¹⁹⁵ This suggests that we become subjectively more tolerant of slight blur and accept it as the norm with increased age, perhaps as a blur-adaptive phenomenon.²⁸⁸

Gain. The accommodative controller gain (open-loop system gain)^{36,37} and slope of the accommodative stimulus-response function (closed-loop system gain)^{36,37,289} (see Figure 4-4) do not change from age 20 to 50 years, remaining approximately 8.00 and 0.85, respectively. This suggests constancy and stability of the accommodative motoneuronal controller and underlying neural pathways in response to the blur input, as well as an absence of any gross biomechanical limitations, within the age-related progressively smaller linear response region.^{1,290} The blur input is effectively multi-

plied by this neural gain term to derive the blur-driven response component to the overall accommodative response. A high gain would therefore result in a larger response than a low gain would. Although the notion of gain constancy has been challenged by some,^{54,291} their curve fitting assumptions were in error.

Accommodative Amplitude. As mentioned earlier, the accommodative amplitude decreases (around 0.30 D per year) over this time period.^{36,37,237,250} This is probably due to gross biomechanical limitations on the ability of the lens and lens capsule to deform. This non-responsive, naturally occurring, upper-end range non-linearity progressively encroaches and extends from the extreme near to the extreme far point of the eye with age. In effect, the near point approaches the far point and optical infinity.

Accommodative Adaptation. Accommodative adaptation decreases progressively with age (0.034 D per year), becoming zero by age 55 years.³⁸ This reflects the reduced drive of the adaptive loop from the direct, primary, blur-driven loop. This reduction may be due to decreased effort to accommodate (because even increased effort no longer deforms the lens sufficiently at near distances to provide clear vision), which may explain why the stimulus AC/A ratio has been found by some to decrease slightly with age.^{293,294} An alternative explanation is that it may be due to subtle biomechanical limitations of the lens and lens capsule, as mentioned above with respect to tonic accommodation.

AC/A Ratio. The stimulus AC/A ratio either remains relatively constant^{7,292} or decreases slightly^{293,294} with age. As mentioned, this decrease is probably due to reduced effort to accommodate, thus driving less accommodative convergence. In contrast, recent results show that the response AC/A ratio increases slightly over this same time period,^{281–283} changing from approximately 3:1 at age 20 years to 5:1 at age 45 (around 0.10^Δ/D/year). Beyond this age, this measurement develops a serious signal-to-noise problem and cannot be assessed reliably. This progressive and modest AC/A increase may reflect: (1) a true adaptive gain change (i.e., increase) in the crosslink gain from accommodation to vergence to compensate for the lens system's age-related reduced responsivity; (2) an age-related increase in ciliary muscle force to compensate for the increased stiffness of the choroid, which would increase the drive to the normal crosslink; and/or (3) slight intrusion of the measurements into the early nonlinear, partially saturating, upper range of accommodation, resulting in slightly more accommodation/effort to obtain a unit change in accommodation and hence more accommodative convergence (see later).

CA/C Ratio. Rosenfield et al.²⁸³ found that the stimulus CA/C ratio decreased progressively with age, from 0.90 D/MA at age 20 to zero at age 55 years. As mentioned earlier, this probably reflects lens saturation. This

finding confirmed and extended the earlier findings of Fincham and Walton.²⁴

Note that the *stimulus* CA/C ratio is specified here. Although the actual accommodative response is measured, the vergence response is assumed (correctly so) to approximate the vergence stimulus extremely closely because of the high gain of the disparity vergence system (100 to 250),⁷² with this difference being only a few minutes of arc (the magnitude of fixation disparity within Panum's fusional areas).^{295,296} Thus, comparison of the stimulus and response CA/C ratios reveals that they are essentially identical.

This is in contrast to comparison of the stimulus and response AC/A ratios. For the stimulus AC/A ratio, the accommodative vergence response is measured by assessing the phoria shift, whereas the change in accommodative response is assumed to equal the change in accommodative stimulus.⁷⁴ However, because of the lag of accommodation at near (which reflects the proportional controller property of static accommodation, with the accommodative error increasing in proportion to the accommodative stimulus⁶⁶), the actual accommodative response is less than the accommodative stimulus (see Figure 4-4). Therefore, in people with normal vision, the response AC/A ratio is approximately 10% larger than the stimulus AC/A ratio²⁹⁷ as it is typically measured clinically.

Dynamic Contributions

The four dynamic accommodative components^{36,38,298,299} with age are as follows.

Latency. The latencies (i.e., reaction times) for both increasing and decreasing accommodation increase slightly (around 2.5 msec/year) from 20 to 50 years of age, consistent with other age-related reaction time measures.¹⁷³ This reflects aging of the underlying neurology, probably related to slowing of the decision process itself or to the more basic nerve conduction velocity and efficiency.

Time Constant. The time constant (i.e., the time to reach 63% of the final exponential-like response amplitude) remains unchanged for both increasing and decreasing accommodation in the linear response region over this same time period. This suggests an absence of biomechanical limitations and changes in the newer (i.e., outermost cortex) and still normally responsive portions of the lens. In contrast, the innermost cortical and nuclear layers become progressively less responsive as they age and become compressed.¹⁵ However, if one attempts to change accommodation within the upper *nonlinear* range, that is, the last diopter or so before the amplitude (see Figure 4-4),²⁹⁰ the time constant is prolonged and the peak velocity decreases (see below) because of normal biomechanical limitations producing response saturation effects.¹⁰⁵ This upper end-point range effect occurs independent of age.

Peak Velocity. The peak velocity/amplitude relationship remains constant with age over the linear response region. That is, the peak velocity increases in proportion to the response amplitude. This suggests constancy and normalcy of the accommodative motoneuronal controller and underlying neural pathways.

Accommodative Microfluctuations. The accommodative microfluctuations progressively decrease in amplitude and frequency from age 20 to 50 years. This probably reflects subtle biomechanical limitations of the lens and lens capsule in response to the minute fluctuations in neural innervation to the ciliary muscle. Once again, the aging biomechanics progressively restrict lens responsivity to these small neural perturbations. This is consistent with a recent small-sample study.³⁰⁰

Theories of Presbyopia

There are two basic theories of presbyopia.* One is lens based, and the other is muscle based (Figure 4-33). In this section, both are discussed and illustrated with clinical examples.

The Helmholtz-Hess-Gullstrand theory attributes all of the loss in accommodation to biomechanical changes in the lens capsule and lens and none to the ciliary muscle. According to this account, the amount of ciliary muscle contraction (or effort/innervation) required to produce a unit change in accommodation remains constant with age. The residual response region is the manifest zone.³⁰⁶ Age brings no loss of power or contractive force of the ciliary muscle. Furthermore, because the lens responds progressively less with age, whereas the ciliary muscle does not, the amount of potential ciliary muscle force in reserve, or reflected in the nonresponsive latent zone,³⁰⁶ increases with age. The following example demonstrates this point. Suppose a 5-year-old child has a 15.00 D accommodative amplitude. The amount of ciliary muscle contraction necessary to produce the initial 1.00 D change would be 1/15th of its full amount. Each succeeding 1.00 D change would require another 1/15th of the total ciliary muscle force. At the 15.00 D accommodative amplitude, all of the ciliary muscle force would be exerted. When the child became middle-aged, he or she would have only 1.00 D of accommodation remaining. The ciliary muscle effort to exert that remaining 1.00 D would still equal 1/15th of its full amount, but there would now be 14/15ths of its contractive force in reserve and nonusable. If the lens and lens capsule could become fully/more responsive once again, for example with installation of eserine³⁰¹ or via an accommodating intraocular lens,³⁰⁷ the ciliary muscle would respond

*References 4,9,12,235,257,258,262,263,301–305.

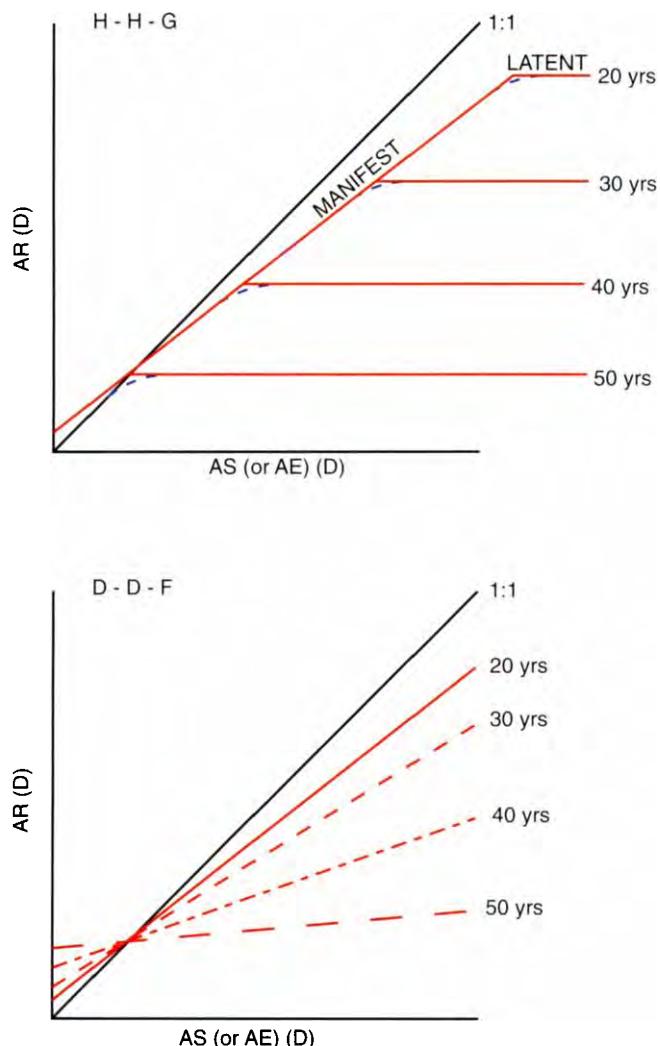


Figure 4-33

Comparison of accommodative response profiles for the Helmholtz-Hess-Gullstrand (H-H-G) and Donders-Duane-Fincham (D-D-F) theories of presbyopia. The diagonal line in each graph is the 1:1 line, and the dashed lines in the top graph represent Morgan's²⁹⁰ high-range end-point modification to unify the two theories. AR, accommodative response; AS, accommodative stimulus; AE, accommodative effort.

normally and expend this considerable reserve of force in a progressive manner nearly up to the accommodative amplitude. As a corollary, the response AC/A ratio over the linear, manifest region would not change with age.

Perhaps the most compelling physiological evidence in support of the Helmholtz-Hess-Gullstrand theory has been provided by Saladin and Stark³⁰⁶ using impedance cyclography. These investigators indirectly assessed the ciliary muscle contractile force by measuring correlated changes in ciliary body impedance using a large, scleral-type contact lens/electrode combination concur-

rent with direct, objective measurements of the lens response. As expected, they found ciliary muscle impedance to alter in proportion to changes in accommodation related to force changes in the ciliary muscle. More importantly, they also found that the impedance *continued to change even once the accommodative amplitude was exceeded*, and thus no further changes in accommodation occurred. These findings suggest that the ciliary muscle could be contracted even further and thus does have considerable force in reserve (i.e., latent), as predicted by the Helmholtz-Hess-Gullstrand theory. However, the capsule and lens could no longer respond to these normal additional forces.

In contrast, the Donders-Duane-Fincham theory attributes all of the age-related loss of accommodation to the ciliary muscle and none to the lens and lens capsule. According to this account, the amount of ciliary muscle contraction needed to produce a unit change in accommodation progressively *increases* with age. Thus, as one ages, the reduced amplitude is due to progressive weakening of the ciliary muscle itself. There is no loss in the ability of the lens and the lens capsule themselves. Furthermore, because the ciliary muscle responds less with age, whereas the lens does not, the amount of potential ciliary muscle force in reserve progressively decreases with age, in opposition to the Helmholtz-Hess-Gullstrand theory. The following example demonstrates this point. Suppose a 5-year-old child has a 15.00 D accommodative amplitude. At that age, the amount of ciliary muscle contraction necessary to produce the initial 1.00 D change in accommodation would again equal 1/15th of its full amount. Each succeeding 1.00 D change would likewise require another 1/15th of the total ciliary muscle force. At the 15.00 D accommodative amplitude, all of the ciliary muscle force would be exerted. When the child became middle-aged, he or she would have only 1.00 D of accommodation left. The ciliary muscle effort necessary to exert that remaining 1.00 D would equal 15/15ths of its full amount, leaving no contractile force in reserve. That is, regardless of the magnitude of the accommodative amplitude, the *total* amount of ciliary muscle contractile force would always be expended.

If the Donders-Duane-Fincham theory is correct, the response AC/A ratio should increase progressively with age and should be extremely high ($30^A/D$ or even greater) at and slightly beyond clinical presbyopic onset.⁶³ Some researchers have in fact found this to be the case.^{294,308,309} However, in two independent, comprehensive prospective population studies,²⁸¹⁻²⁸³ only a small ($0.10^A/D/year$) increase in response AC/A ratio was found. It increased from approximately 3:1 at age 20 to 5:1 at age 45 years. Even the more recent finding of Baker and Gilmartin³¹⁰ showing a moderate increase in response AC/A ratio in the near range of the incipient presbyope would better fit the Helmholtz-

Hess-Gullstrand versus the Donders-Duane-Fincham theory. These age-related changes are much smaller than those predicted by the Donders-Duane-Fincham theory and, although not in perfect agreement, are more in line with the Helmholtz-Hess-Gullstrand theory. Furthermore, they are consistent with Fisher's²⁷⁹ finding of increased contractile force of the ciliary muscle with age, as discussed earlier. Beyond 45 years of age, the AC/A ratio is too unreliable to measure, because of little or no residual accommodation.²⁸²

Morgan²⁹⁰ attempted to unify these two theories (see Figure 4-33). He asserted that the Helmholtz-Hess-Gullstrand theory held for most of the accommodative range where linearity was approximated but that within 1.00 D or so of the accommodative amplitude, the Donders-Duane-Fincham theory appeared to hold. At that high end, increased accommodative effort would be required to generate the final diopter, presumably because of nonlinear, biomechanical aspects related to the lens capsule and lens substance in this operating region. Such extreme lens range nonlinearities are age independent and reflect the basic biomechanical properties of the system.¹⁰⁵ A meta-analysis approach to the research in this area suggests that the Helmholtz-Hess-Gullstrand theory more accurately depicts the phenomenon and process of human presbyopia.

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5

Fusion and Binocular Vision

Kent M. Daum, Glen L. McCormack

This chapter describes the sensory and motor aspects of the normal binocular visual system. The binocular visual system enables a full appreciation of a variety of aspects of an object in the field of view. Over a wide field of view, the sensory system determines the direction, achieves a single image from the two eyes, and provides information useful for judging the distance of the object. The motor system closely coordinates the orientation of the eyes at all distances and angles for both stationary and quickly or slowly moving targets; it also coordinates with the accommodative system to maintain a clear image. An efficient sensory and motor binocular system provides single, clear, and comfortable imagery used for making a wide range of decisions about the nature of the object of regard.

An efficient and functioning binocular visual system requires proper function of the more fundamental portions of the visual system. For example, clear bifoveal images are needed for high-resolution stereopsis. Clear bifoveal images during near vision also require appropriate convergence and accommodation. Convergence and accommodation are controlled principally by binocular vision and retinal image focus, respectively. Because convergence and accommodation must be properly coordinated for clear vision and acute stereopsis, the systems are neurologically linked to each other. This neurological linkage allows binocular sensory and motor function to influence accommodation, and it also suggests that each potentially affects the measurement of the refractive status of the eye. Understanding binocular vision is essential to fully grasp accommodative behavior (Chapter 4).

The purposes of vision are to identify and localize objects, and the anatomy and physiology of the brain reflect this duality.¹ The purpose of binocular vision is to enhance vision over what it would be monocularly and particularly by way of stereopsis. Stereopsis, which means "seeing solidly," contributes to the judgment of depth and distance and participates in the recognition of some solid objects.

SIGNIFICANCE OF BINOCULAR VISION

Normal binocular vision with stereopsis (a type of depth perception) provides many visual advantages over poor stereopsis or monocular vision in a variety of everyday or specialized tasks. Good binocular vision and stereopsis critically enhance visual capability and provide more than just depth perception. Binocular visual enhancement is generally greater under conditions of reduced visibility and is also present with tasks that apparently do not involve critical depth perception.^{2,3} Good binocular vision and stereopsis provide both low-level (as in "camouflage breaking," or detecting figures against a background) and high-level (depth ordering) comparisons of information available to each of the eyes.⁴ As compared with monocular viewing, binocular vision and stereopsis also help provide better motor control (e.g., when reaching for a target or completing fine motor tasks); they also provide quicker and more accurate cognitive information (e.g., when estimating the time to collision).⁵ Jones and Lee⁶ have shown binocular vision to be superior to monocular vision in each of 10 widely varied tasks: (1) letter identification, (2) detecting camouflaged octopuses, (3) color discrimination, (4) bead threading using closed-circuit TV, (5) tracking a moving target using closed-circuit TV, (6) control of stance, (7) needle threading, (8) water pouring, (9) reaching with the hand invisible, and (10) reaching with the hand visible. Subjects performed each task more effectively under binocular than monocular conditions, although some of the tasks did not involve stereopsis. In addition, subjects tended to demonstrate a greater binocular advantage in dim as compared with bright illumination.

Likewise, Sheedy and colleagues⁷ performed similar experiments demonstrating improved binocular over monocular performance when using pointers and straws (30%), threading needles, shuffling file cards, orienting small pegs in a grooved pegboard, and

performing a reading task (3%). No improvement occurred with video display terminal (VDT) letter counting or during a beanbag toss. Good binocular vision and stereopsis provide information for optimal fine motor coordination involving reaching and grabbing.⁸ Figure 5-1 is a stereogram showing three rising limbs of a small tree. The reader may view the stereogram with and without crossing the eyes (see figure legend for instructions). If you were leaping to the nearest limb, which branch would you select? Binocular vision provides improved performance (speed and accuracy) over the lack of stereopsis present with an endoscope during paranasal sinus surgery⁹; other experiments also suggest superior binocular performance over monocular for surgical tasks.¹⁰ Finally, individuals estimate time to collision more accurately and quickly under binocular conditions than monocular conditions.⁵

The loss of stereopsis during constant strabismus (crossed eyes) has little obvious effect on visually guided behavior in most afflicted patients. The difference for most everyday tasks is likely small (perhaps only a few percentage points), and it is often not noticeable. In addition, humans are well adapted to learning compensatory behaviors to eliminate or even enhance the performance of a task with monocular or other cues. Pure binocular tasks are relatively rare in everyday life.

In summary, good binocular vision and stereopsis provide more information than just depth perception.^{2,3} Good binocular vision and stereopsis allow the viewer

to perceive objects more quickly and accurately than would otherwise be the case. This advantage is most important in situations in which visibility or contrast is low and in which critical decisions must be made accurately and quickly. Brief exposure duration may expose stereo anomalies.²

The binocular field of view also is larger than the monocular field of view. The normal monocular field of view is approximately 60° upward, 60° inward, 70° downward, and about 100° outward.¹¹ The binocular field extends about 200° laterally and about 130° vertically.¹¹ Laterally, this amounts to about 40° greater horizontally and about the same extent vertically. This provides greater ability to detect and react to peripheral stimuli.

Poor binocular vision produces fatigue, blur, headaches, and eye strain. Anomalies of binocular vision may reduce visual comfort and overall task performance.¹² Binocular visual anomalies are seen in individuals who possess two eyes but cannot use them together (e.g., strabismus) or efficiently (e.g., heterotropia/vergences dysfunction). Strabismus is commonly referred to as "cross-eyed" or "walleyed," and individuals with this condition may have their binocular capabilities (e.g., stereopsis) reduced or eliminated and/or their field of vision limited.¹² Strabismus may occur all the time (constant strabismus) or intermittently. Other individuals who retain bifoveal fixation (heterophoria or phoria) may be unable to maintain clear, comfortable vision as a result of stresses related to

Right Eye View



Left Eye View

**Figure 5-1**

Stereogram of a small tree. The relative depths of the three vertical branches are ambiguous in monocular vision, but they become obvious in binocular vision. The stereogram should be viewed by means of heterophoric (cross-eyed) fusion. Hold this book at a distance of 50 cm while fixating the tip of a pencil held in front of the bottom of the stereogram at a distance of approximately 25 cm from your nose. The picture will automatically come into focus after fusion and depth are appreciated. You may need to adjust the distance of the pencil to obtain fusion; the pencil may then be removed. Normal or abnormal limits to accommodation and vergence may prevent some readers from achieving fusion.

keeping their eyes fused (poor phoria/vergence relationship). Frequently these individuals report blur, asthenopia (eye strain), headaches, or diplopia (double vision) associated with the use of their eyes, and they may notice difficulties with stereopsis or other critical visual tasks.

BINOCULAR SENSORY FUNCTION

Binocular sensory function is essential for establishing the direction and location in depth of an object of regard and for combining the two images of the eyes. This section reviews basic principles of binocular vision and space perception. Comprehensive reviews of these topics are available.^{13,14}

Space Perception

Visual space perception is the subjective appreciation of the spatial extent and locations of objects in space. The visual system constructs a perceptual image of the world from the optical images on the retinas. One's surroundings (e.g., "the real world") are known as *object space*, and the perceived version of it is known as *visual space*. Both object space and visual space have geometries that define left versus right, up versus down, and near versus far. Because visually based judgments of object space are ultimately based on visual space, visual space must replicate object space as faithfully as possible. Understanding the general principles of space perception is necessary for appreciating these visual judgments.

The Cyclopean Eye

Like the Cyclops of Greek mythology (Figure 5-2), humans see the world as though from a single cyclopean eye situated between the two anatomical eyes¹⁵ and located at the egocenter. The sensation of the cyclopean eye is so compelling that preschool children asked to view objects through a tube reflexively bring the tube to the bridge of the nose.¹⁶ *Fusion* is the sensory process that unites the eyes' images into one. The visual system requires binocularity to achieve binocular visual perception (including stereopsis), and yet it achieves a single representation of a world viewed through two eyes.

Visual Localization and Visual Direction

To localize an object means to judge its location relative to some point of reference. When an object is localized with respect to the egocenter, that judgment is called *egocentric localization*. Egocentric localization provides the answer to the question, "Where is the object in three-dimensional space, relative to me?" Egocentric



Figure 5-2

The single eye of the mythological cyclops is a useful metaphor for human binocular vision. Not only do we obtain single vision with two eyes, we also behave as though we view the world from a point midway between the eyes.

localization is also called *absolute localization*, because it includes the ability to judge the locations of objects in units of measure, such as meters. A complete egocentric location judgment must include two components: egocentric direction and distance. Stereopsis is one of many contributors to the distance component of visual localization. Stereopsis provides a compelling sense of depth from binocular observation; it is not present while viewing with only one eye.

The egocentric direction of an object is its direction relative to the egocenter within the head. An egocentric direction may be conceptualized as an arrow of indefinite length arising from the egocenter. A special egocentric direction is called *straight ahead*, and it is analogous to a line originating at the egocenter and passing perpendicularly through the plane of the face. Other lines originating from the egocenter and passing through the face obliquely would represent leftward, rightward, upward, and downward.

The perceptual computation of the egocentric direction of an object begins with the determination of the object's image position on the retina. The sense of direction assigned to retinal images is an inherent property of the spatially ordered connections of the retina with the brain, which assigns a unique subjective direction to each retinal point relative to other retinal points. *Uniqueness* means that there are no two points on the retina that are associated with the same sense of

direction at any one instant of time. The unique direction associated with each retinal point is known as a *local sign*¹⁷ or *retinal locus*. Local signs are ordered so that neighboring images on the retina are perceived as neighboring objects, and widely separated retinal images are perceived as objects that are far apart. The local sign directional value of a retinal point, called *oculocentric direction*, is based on a set of coordinates that are conceptually analogous to those used to define eccentricity in the visual field. The fixation point serves as the origin of the oculocentric coordinate system, the vertical meridian of the visual field separates oculocentric left from right, and the horizontal meridian of the visual field separates oculocentric up from down. Therefore, oculocentric direction may be viewed as the quantification of the location of an object within the visual field; like visual field position, it is measured in units of angular subtense. Oculocentric direction is not a perceived direction as such but rather a factor used by the brain in the perceptual computation of egocentric direction. In other words, all judgments of visual direction are ultimately egocentric.

Local signs are not all equal. The ability to resolve differences of visual direction by comparison of local signs is related to visual acuity, and it declines with retinal eccentricity. The absolute threshold of oculocentric direction is quantified by tasks that require the observer to discriminate minimal differences of visual direction. An example of such a task is a Vernier acuity test, in which the observer judges the horizontal alignment of a pair of vertical lines (one of which is always higher than the other).

The neural mechanisms that calculate oculocentric direction are highly abnormal in amblyopia (a functional loss of vision in one eye usually caused by anisometropia or strabismus). Amblyopic patients are uncertain about the relative visual directions of foveal images in the amblyopic eye; in most cases, they see distorted foveal images.^{18,19}

A special local sign called the *principal visual direction* elicits the sensation of "looking at" a particular object. Therefore, the principal visual direction sense is associated with the fixation point and the center of the anatomical fovea in those with normal vision; it follows that the principal visual direction is the origin or center of oculocentric direction. When an off-foveal image becomes an eye-movement target, the comparison of the oculocentric direction of the off-foveal image to the principal visual direction establishes the metrics of an eye movement that would attain foveal fixation of that target. Therefore, the principal visual direction guides foveal fixation in individuals with normal vision. In patients with amblyopia caused by strabismus (strabismic amblyopia), the principal visual direction may reside at a peripheral retinal locus in the amblyopic eye, thereby creating the clinical anomaly *eccentric fixation*

(Chapter 31). When viewing with the amblyopic eye (i.e., the dominant eye is covered), these patients move the eye so as to place an image of interest on an eccentric retinal point rather than the fovea. This behavior accounts for a portion of the reduced visual acuity of strabismic amblyopes.²⁰

Oculocentric direction alone is not sufficient to reveal egocentric direction, because the eyes are constantly in motion. Egocentric direction is constructed from two pieces of information: (1) oculocentric direction and (2) the direction of gaze of the eye. The ability to sense the direction of gaze, called *registration* by Morgan²¹, may involve a contribution from eye muscle proprioception,^{22,23} but it is probably dominated by corollary discharge wherein brainstem oculomotor neurons send eye-position messages to those parts of the brain that compute visual direction.²⁴ The perceptual calculation of egocentric direction from oculocentric direction and registered gaze can be compared with an algebraic summation process in which visual field eccentricity represents oculocentric direction (rightward is positive) and ocular rotation from the primary position of gaze (see Eye Movements) represents registered gaze (right gaze is positive). For example, the egocentric direction of an object 2 degrees to the left of the point of fixation (oculocentric direction = -2 degrees) with the direction of gaze at 10 degrees to the right of straight ahead (registered gaze = +10 degrees) would be +8 degrees.*

Patients with paretic strabismus may experience egocentric direction judgment errors when monocularly fixating objects with an eye that has a paretic muscle. Paretic strabismus is the failure of one of the two eyes to align with its intended target because of partial failure of the extraocular muscles or the oculomotor nerves that innervate them (see Chapter 10). The monocular egocentric judgment error occurs because the eye moves to its intended fixation target only by the use of abnormally large amounts of innervation which, when registered in space perception, gives rise to an exaggerated egocentric direction for that target. This error is revealed by the past pointing test.²⁵ Patients with unilateral strabismus of nonparetic origin (i.e., patients who have a deviated eye despite functioning extraocular muscles and oculomotor nerves) also fail to accurately register the position of the nondominant (normally turned) eye, even when the dominant eye is covered.^{26,27} The cause of this behavior is unknown.

*The calculation of egocentric visual direction—a subjective entity—from the object space quantities visual field eccentricity and ocular rotation is not an exact process, because oculocentric direction and registered gaze may not precisely encode their object space counterparts. In anomalies such as strabismus and amblyopia, the oculocentric direction and registered gaze values derived from the defective eye can be highly inaccurate.

Depth and Distance Perception

The perception of three-dimensional space can be subdivided into two processes: distance perception and depth perception. *Distance perception* is the judgment of how far a given object is from the observer or from some other object, and it includes the ability to judge distances in absolute units of measurement (e.g., meters). Distance perception is not a relative assessment and is therefore also known as *absolute depth perception*. *Depth perception*, which is also known as *relative depth perception*, is the perception of the relative nearness of one object to another or the evaluation of the relative depth intervals between two or more points in space.

The following example illustrates the difference between depth and distance perception. An observer views a pencil on his desk located at a distance of 20 cm from himself, and he also sees a coffee cup on his desk located 30 cm from himself. Depth perception tells the observer that the coffee cup is 50% farther than the pencil, whereas distance perception reveals that the pencil and coffee cup are separated by 10 cm. Depth information alone usually cannot reveal the absolute distance between objects or the distance of objects from an observer. Therefore, depth information must be adjusted according to distance information to have value for visually guided behavior. In this example, if distance perception was able to show that the pencil is 20 cm distant from the observer, depth perception could be used to perceptually calculate that the coffee cup must be at a distance of 30 cm ($30 \text{ cm} = 20 \text{ cm} + 50\% \text{ of } 20 \text{ cm}$).

Depth perception is a function of many factors. Depending on the context, it usually includes more than stereopsis. Factors involved include binocular visual factors such as retinal disparity (provides stereopsis and is very precise) and vergence alignment (gross); monocular visual factors such as accommodation, looming, motion parallax, and the kinetic depth effect; and pictorial depth cues such as occlusion, perspective, texture gradients, relative size, height in visual field, shadow, luminance, and aerial perspective.²⁸ Stereopsis is a specific type of binocular depth perception that is the result of the horizontal separation of the two eyes and the subsequent ability to recognize retinal disparity.

Various attributes of images, known as *cues*, activate depth perception and distance perception. A *depth or distance cue* is an identifiable property of the optical images that is correlated with depth or distance. Because the salience of a depth or distance cue depends on the visual environment, the brain uses numerous distance and depth cues to optimize the reliability of space perception in any environment that might be encountered. Binocular vision activates several special cues to distance and depth perception, but most cues do not depend on binocular vision and are therefore known as *monocular*

cues. Figure 5-3 illustrates several monocular depth and distance cues. Some monocular cues are based on the fact that an object's distance from an observer is inversely proportional to its retinal image size. For instance, *linear perspective* is triggered by the convergence of lines to a vanishing point; a perfect example of this is the convergence of the rails in Figure 5-3; the separation of the rails is inversely proportional to viewing distance. The *texture density cue* provides depth information when the angular size of repeating patterns or textures (e.g., the gravel, railroad ties, and utility poles) diminish with distance. The *known size cue* allows the computation of depth and distance on the basis of retinal image size and knowledge about the true size of objects being viewed. For instance, the known size cue suggests that the cab of the near locomotive in Figure 5-3 might be approximately 4 m from the camera that created the photograph.

Luminance variations reveal depth in several ways. Shadows reveal three-dimensional relief in the presence of directional light sources. The stack of unused rails in the lower right corner of Figure 5-3 appears to stand up above the ground by virtue of the shadow it casts. *Aerial perspective* reveals depth over great distances by the reduced contrast of images viewed through atmospheric light scatter; this effect is clearly illustrated in Figure 5-3 by the faint contrast of the mountains in the background. The *overlay cue* reveals depth when the contours of near objects occlude the contours of far objects; this effect does not depend on angular size or luminance variables. The nearer of the two locomotives in Figure 5-3 appears nearer not only because of its larger angular size but also because it obstructs a part of the camera's view of the far locomotive.

Looming, motion parallax, and the kinetic depth effect are monocular cues that stimulate depth and distance perception when motion is present. *Looming* is the sense of movement in depth stimulated by a change in the size of a retinal image. *Motion parallax* is the sense of depth or distance stimulated by the differential motion of retinal images of objects that are farther or nearer than the point of fixation. Head motion induces the retinal image motions. Motion parallax can be appreciated with this simple demonstration: hold the index fingers of the right and left hands in the egocentric straight-ahead direction, one behind the other, and move the head laterally to and fro. When the far finger is fixated it is perceived to be stationary, whereas the near finger appears to move in a direction *opposite* to that of the head. When the near finger is fixated, the far finger appears to move *with* the head. The differential motion of the two fingers provides depth information. The *kinetic depth effect* provides another sense of depth based on the differential motion of portions of the retinal images. However, the retinal image motion in this case is caused by object motion rather than head motion.



Figure 5-3

This photograph of a rail yard illustrates six monocular depth and distance cues: (1) linear perspective, (2) overlay, (3) shadows, (4) aerial perspective, (5) texture density, and (6) known size. See the text for explanations of these cues. (*From Ball D. 1972. Portrait of the Rails, from Steam to Diesel, p. 54. Greenwich, CT: New York Graphic Society.*)

The geometry of the kinetic depth effect can be appreciated by holding the hand upright, with the palm of the hand parallel to straight ahead. Rotate the hand to and fro about the middle finger as a rotational axis. The index finger and little finger will be observed to be moving in opposite directions. The differential speeds and directions of motion of different parts of the hand are powerful cues about the interval of depth between the near and far limits of the hand. This cue is effective for any rotating three-dimensional object.

Clinicians use motion parallax to judge the relative position of objects when using a direct ophthalmoscope. For example, if a clinician is focused on the iris and the object in question moves against the direction of movement of the ophthalmoscope, the object must be anterior to the iris and is most likely on the cornea. An object moving with the ophthalmoscope would be behind the iris and may be in the lens or vitreous.

In a cue-rich environment, the visual system determines distance from a combination of cues. Landy and associates²⁹ propose a two-step process for the perceptual calculation of perceived distance from multiple cues. The first step, called *promotion*, converts the depth estimate from each depth cue into a perceived distance value; depth and distance cues may cooperate during the promotion process. The second step combines the promoted distance cues by a weighted average of those cues, as long as those cues are in reasonable agreement with each other. The weight assigned to each cue depends on the cue's importance in the visual environment. If two strongly weighted cues present conflicting distance information to the observer, those cues are not averaged but rather lead to the suppression of the relatively weaker cue by the stronger cue. The binocular cues to depth and distance, including stereopsis, are involved in the cue combination process described above and are discussed in Binocular Contribution to Depth and Distance Perception.

The Stimulus to Stereopsis

The lateral separation of the eyes provides each with slightly different views of the world. This differential perspective, known as *binocular parallax*, elicits convergence eye movements and is related to the stimulus to stereopsis. The magnitude of horizontal binocular parallax (P) for any given point in object space (x) (Figure 5-4, A) is a function of the lateral separation of the eyes divided by object distance, and it is quantified in angular units:

$$P = 2 \times \arctan(a/d) \times k$$

where a = one-half the ocular separation, d = the distance of the object from the line connecting the nodal points of the eyes, and k is a conversion factor that varies

depending on the angular units of P (e.g., degrees, prism diopters).

At least two points in object space must be visible for stereopsis to be appreciated. Geometric (or relative) disparity is the stimulus to stereopsis. *Geometric disparity* is the depth interval between two object points quantified in angular units of measurement.³⁰ Because the eyes are laterally separated, the range of geometric disparities encountered by stereopsis is much greater in the horizontal dimension than in the vertical dimension. Horizontal geometric disparity (D) is calculated as the difference of parallax angles (P_1 and P_2) subtended by two points (x and y) in object space (Figure 5-4, B):

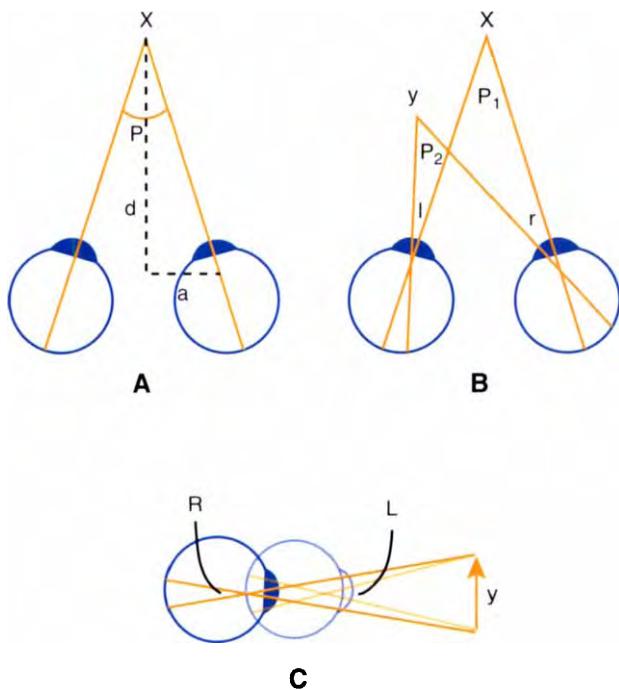
$$D = P_2 - P_1$$

Figure 5-4, B, shows a second way to calculate the geometric disparity between points x and y : the difference between the parallax angles P_2 and P_1 is equal to the difference between the longitudinal angles r and l . Therefore, $D = r - l$.

Vertical geometric disparity can be defined as the difference of a vertical angle subtended at the right and left eyes by a given object.³⁰ Vertical geometric disparity is zero for object points that are on the median plane but nonzero for objects to the left or right of the median plane. The *median plane* is a vertical plane that separates the head into equal right and left halves. Figure 5-4, C, shows an observer from his right side. The observer fixates an object Y in near vision, which is left of the median plane. Because the right eye (dark lines) is farther from object Y than the left eye (shaded lines), object Y subtends a larger visual angle in the left eye (angle L) than in the right eye (angle R). The difference of these two vertical angles is the vertical geometric disparity of object Y . Vertical geometric disparity can be appreciated by viewing the palm of the hand from a close distance (e.g., 25 cm) while the hand is held to the left of straight ahead (keep the head straight while the eyes look left). Look at the hand with one eye and then the other. Careful observation will show that the hand is slightly larger as seen by the left eye than by the right eye. When the hand is held to the right of straight ahead, it is seen to be slightly smaller by the left eye than by the right eye. These size differences induce vertical geometric disparity.

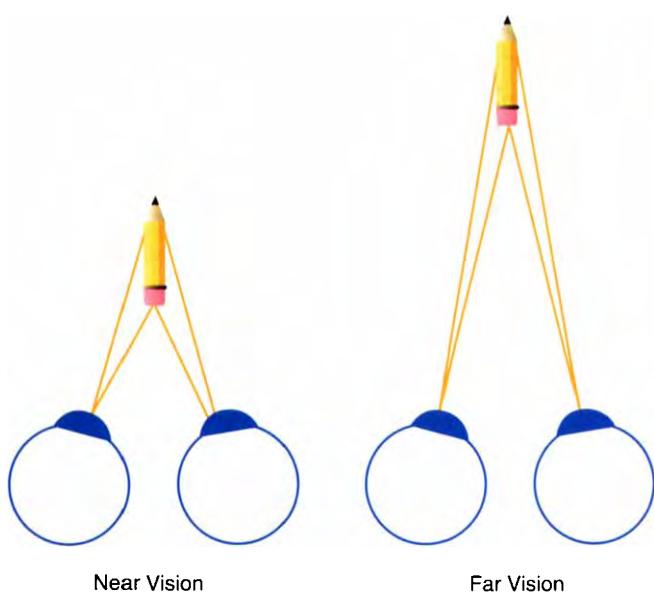
The Binocular Contribution to Depth and Distance Perception

Because of the lateral separation of the eyes, most of the geometric disparities that support stereopsis are horizontal disparities. Horizontal geometric disparity is an effective depth cue in near vision, because the greater size of parallax angles in near vision causes greater geometric disparities. However, horizontal geometric

**Figure 5-4**

The geometry of horizontal binocular parallax (P) and geometric disparity (D). Angular units of measurement are used to quantify binocular parallax and geometric disparity. A and B show the eyes from above the head; C shows the eyes from a side view. Horizontal geometric disparity (D) = $P_2 - P_1$ (or $r - l$) in B. Vertical geometric disparity (D) = $R - L$ in C. See the text for explanation.

disparity alone does not provide sufficient information for the brain to calculate perceived distance. This is illustrated in Figure 5-5: an observer views a pencil pointed away from himself in near vision and then in far vision. The true length of the pencil is the same in near and far vision, and a normal observer perceives that distance interval to be the same. However, it is apparent that, because the parallax angles (shaded lines) subtended by the end points of the pencil are larger in near vision, the geometric disparity between the end points must be larger in near vision. Accordingly, the horizontal geometric disparity cue would by itself suggest that the length of the pencil is greater in near vision. The fact that this percept does not occur indicates that perceived distance is not determined directly from horizontal geometric disparity. To use horizontal geometric disparity to judge this depth interval, it is necessary to know how far the pencil is from the observer. Stereopsis derived from horizontal geometric disparity is therefore a depth cue that, like some monocular depth cues, must be promoted by distance cues to attain perceptual significance for distance judgment.

**Figure 5-5**

Different geometric disparities are subtended by an object in far and near vision. The parallax angles subtended by the object are larger in near vision than in far vision, so the geometric disparity subtended by the pencil is greater. See the text for further explanation.

Theoretical arguments suggest that vertical geometric disparity should contribute to stereoscopic depth perception.^{31,32} Ogle³³ acquired systematic evidence for the stereoscopic effect of vertical geometric disparity, observing that the apparent slant of binocularly viewed surfaces is altered by vertical magnification of one ocular image. Ogle called this the *induced effect*, because it was thought that vertical disparities had no direct stereoscopic effect but rather induced changes of stereoscopic values associated with horizontal disparities. It has also been observed that vertical disparity contributes to the apparent curvature of binocularly viewed surfaces.³⁴ These observations, coupled with the theoretical arguments of Mayhew and Longuet-Higgins³² and of Gillam and Lawergren,³¹ strongly argue for the hypothesis that vertical disparity directly contributes to stereoscopic depth.

Mayhew and Longuet-Higgins³² and Gillam and Lawergren³¹ also showed that there is sufficient information in the combination of vertical and horizontal geometric disparities to allow the visual system to compute distance without using the known size distance cue. In other words, vertical disparities may be able to promote stereoscopic depth derived from horizontal geometric disparities. Rogers and Bradshaw^{34,35} have confirmed that textures sufficiently lateral from the median plane (e.g., 20 degrees) in near vision can promote horizontal geometric disparity information to distance information.

The brain can sense the angle of convergence, a process called *registered convergence*. Space perception can use registered convergence to judge both depth and distance³⁶ and to promote horizontal geometric disparities.³⁷ The stereoscopic depths of all objects in the vicinity of the fixation point can be promoted by the perceived distance of the fixation point sensed through registered convergence. The convergence cue is not redundant with the vertical disparity distance cue. The vertical disparity cue is most effective when the observer views binocular fusion fields extending beyond 20 degrees retinal eccentricity, whereas the registered convergence cue is relatively more effective at smaller retinal eccentricities.³⁷ The promotional effects of these cues are approximately additive.³⁷

The Spatial Limits of Stereopsis

For a hypothetical observer to appreciate the stereoscopic depth of every point in the three-dimensional binocular visual field without eye movements, the brain would have to compute retinal disparity by comparing each point on one retina with every point on the other retina. In practice, any given point on the retina of one eye can be shown to interact with a limited area of retinal points in the other eye. Consequently, stereopsis processes disparity for only a portion of object space at any one instant of time. This limited range of stereopsis may possibly be related to neuroanatomical and physiological economy. The brain can use vergence eye movements to adjust the limited range of stereopsis to view all of object space over time.

Stereopsis processes the portion of object space that is centered on the point of fixation. The range of stereopsis, therefore, moves in object space in association with eye movements. The *horopter* is at the center of the region of stereopsis (Figure 5-6); it is defined by all those points in object space that stimulate corresponding retinal points. Corresponding retinal points (or, simply, corresponding points) are the pair of points, one in each eye, that retain the same sense of visual direction.³⁸ By definition, any object that is on the horopter has the same visual direction as seen by each eye. Any object not on the horopter will appear in different directions to the two eyes. Any object that stimulates a pair of corresponding points also appears to the observer to be at the same stereoscopic distance as the point of fixation and to be single.³³ *The horopter is visually significant because it is the center of the range of single binocular vision and the region of highest relative stereoscopic acuity.*

Normally corresponding points have approximately the same anatomical locations on the two retinas relative to the foveas. For instance, the centers of the foveas are normally corresponding points, and so are points in the left hemiretinas that are equally distant from their

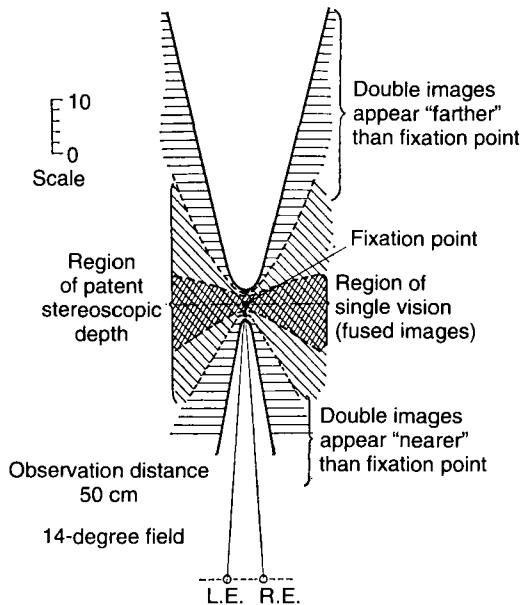


Figure 5-6

Relationship between qualitative binocular perception, single vision, and the horopter. Single vision with patent stereopsis is experienced near the horopter. Patent stereopsis extends greater distances from the horopter than does singleness, but it does not extend as far as qualitative stereopsis. The depth range of each type of binocular perception increases with retinal eccentricity. L.E., Left eye; R.E., right eye. (From Ogle KN. 1962. Part II: The optical space sense. In Davson H [Ed], The Eye, vol 4, p. 285. New York: Academic Press.)

respective foveas. Binasal or bitemporal pairs of points, although symmetric, do not provide the same visual directions and are not corresponding points. The anatomical relationships suggest that the physiological and anatomical starting point of binocular correspondence is the overlapped retinotopic mapping of the eyes in visual cortex. If corresponding points were distributed identically in the two eyes and the eyes had no optical distortions, the horizontal horopter would be a perfect circle called the *theoretical horopter* or the *Vieth-Müller circle*. The locus of the Vieth-Müller circle contains the fixation point and the nodal points of the eyes. Because of optical and perhaps neuroanatomical asymmetries, the empirical horopter (a horopter measured on an observer, usually simply called the horopter) is somewhat less curved than the theoretical horopter.

Retinal disparity describes the spatial relationship of retinal images with corresponding points. Images falling on corresponding points subtend zero retinal disparity; images falling on noncorresponding points subtend nonzero retinal disparity. An object closer to the observer would have its images fall temporally from corresponding points on the retinas.

An observer viewing the images alternately with the two eyes would perceive the right eye image to be left of the left eye image (and, of course, the left eye image to be right of the right eye image). The closer-than-the-horopter object subtends crossed retinal disparity, because each image is perceived to be on the opposite side of the body from the eye that perceives it. Crossed disparity arises from viewing objects that are closer than the horopter.

If an object were farther from the observer than the horopter, its images would fall nasalward from corresponding points, thereby causing the observer to see the right eye's image as rightward of the left eye's image (and the left eye's image to be left of the right eye's image). This object causes uncrossed retinal disparity and indicates that an object is being viewed that is more distant than the horopter.

Objects considerably away from the horopter result in large retinal disparities and cause diplopia. *Diplopia* is "double vision" and the percept of two different visual directions associated with one object. Uncrossed disparities elicit uncrossed diplopia (and an object closer than the horopter), and crossed disparities elicit crossed diplopia (and objects more distant than the horopter).

Retinal disparity (or absolute disparity) cannot be measured easily in a clinical setting while the patient is binocular. However, retinal disparity is quantified routinely during dissociated vision in the form of *heterophoria*. In the Maddox rod test (Chapters 10 and 21), the fixating eye views a small spot of light from a transilluminator or penlight (also a "muscle light"). The nonfixating eye views the light through a Maddox rod, which distorts the light into a vertical streak. The retinal image difference of the shapes renders them infusible (see Binocular Fusion for a discussion of factors that limit fusion). If the dissociated (bi-ocular) eyes maintained perfect alignment (zero heterophoria) with the Maddox rod in place, the streak would remain superimposed on the light, stimulating corresponding points in the centers of the foveas. Most individuals do not have a heterophoria of zero and therefore do not continue to point at the light when fusion is absent. In these cases, the eye viewing through the Maddox rod drifts to its phoric posture; it is not pointed at the light, and the streak image falls on peripheral retina. In an *exophore*, the streak falls on the temporal retina, stimulating a crossed retinal disparity (and crossed diplopia). In an *esophore*, the streak falls on the nasal retina, stimulating an uncrossed retinal disparity. When the appropriate magnitude of prism is placed before the eye wearing the Maddox rod, the streak again appears superimposed on the light; thus, the diplopia is being neutralized. The value of prism that neutralized the diplopic retinal disparity is assumed to be equal to the retinal disparity. If it is also assumed that the patient has normal binocu-

lar correspondence (see the next paragraph), this measured retinal disparity is numerically equal to the deviation of the visual axes; the patient's heterophoria has been measured. It follows that subjective heterophoria tests are direct tests of unfused retinal disparity, but they are indirect tests of ocular alignment.

The retinal disparity assessed by the Maddox rod test is used to assess the direction of the eyes when they are dissociated. The test effectively assesses the directions of the eyes, but the reader should note that the test does not indicate stimuli that are closer than the horopter or more distant than the horopter, because the stimulus is in a single location being fixated by the observer.

Retinal disparity should not be confused with fixation disparity. *Retinal disparity* is a measure of the relationship of the retinal images of an object with the corresponding retinal points. Retinal disparity can be measured for any pair of eyes and does not require fusion. *Fixation disparity* arises from the stimulation of corresponding points within Panum's area (see Binocular Fusion) and is a different concept that requires fusion and indicates a micro-misalignment of the eyes. Retinal disparity may be a stimulus to the motor system of the eyes to fixate an object closer or more distant than the previous fixation point. After the new stimulus is fused, fixation disparity guides the function of the motor system (see Binocular Motor Function).

Normal binocular correspondence changes little in most viewing environments.³⁹ However, Robertson and Schor⁴⁰ and Remole⁴¹ observed that binocular correspondence could change as much as 1^{Δ} in individuals with normal vision during fusional stress stimulated by prism vergence testing. In other words, retinal points normally disparate by as much as 1^{Δ} could become functionally corresponding points, whereas retinal points that usually correspond become disparate. This behavior is not abnormal, does not indicate binocular weakness, and does not significantly affect the interpretation of prism vergence tests.

In contrast with normal correspondence, anomalous retinal correspondence (ARC) occurs in some types of strabismus and associates an identical sense of visual direction with anatomically dissimilar retinal points. For instance, in a 10^{Δ} left esotrope with ARC, the fovea of the fixating eye may retain the same sense of direction as a nasal retinal point in the deviated eye 10^{Δ} from the fovea. ARC is less stable than normal correspondence, and the sense of direction of the deviated eye may easily and spontaneously change by many prism diopters. Testing for anomalous retinal correspondence in esotropic strabismus is important, because its presence lowers the prognosis for the cure of strabismus⁴² and alters the treatment methods (see Chapter 31).

There are both far (uncrossed) and near (crossed) retinal disparity limits to the range of stereoscopic depth perception (see Figure 5-6) that is centered on the

horopter. The depth range of stereopsis is smallest in foveal vision and increases with retinal eccentricity.³⁰ The disparity limits of the range of stereopsis can be defined by the quality of the stereopsis that is judged. Stereopsis near the horopter elicits depth percepts that are more compelling than stereopsis elsewhere. These depth percepts are directly proportional to the geometric retinal disparity and are associated with single binocular vision or small degrees of diplopia. Ogle³⁰ called this type of stereopsis *patent stereopsis*. Objects farther from the horopter elicit less-compelling depth percepts in which only the direction of depth is distinguished (i.e., nearer than fixation versus farther than fixation) and in which the images are seen as moderately diplopic.³⁰ The latter form of stereopsis is called *qualitative stereopsis*. Objects having retinal disparities larger than the limits of qualitative stereopsis cause only diplopia. The small depth range of foveal patent stereopsis requires accurate oculomotor alignment for stereopsis to function efficiently, so a strabismus patient having a small ocular misalignment has a significant loss of stereoscopic acuity (see Chapter 21).

The most remote distance at which stereopsis can resolve depth is limited by stereoscopic acuity and the lateral separation of the eyes. That distance can be calculated with the binocular parallax equation (see The Stimulus to Stereopsis), in which the stereoscopic threshold (in seconds of arc) is substituted for "P," "k" is set to 3600, "a" is measured in meters, and the equation is solved for "d." For instance, an observer with a 0.06-m interocular distance and a stereoscopic threshold of 20 seconds of arc could stereoscopically resolve a point as remote as 619 m as being nearer than infinity. More distant objects would not be stereoscopically discriminable from infinity. In practice, useful stereopsis is limited to much closer distances, because threshold stereopsis is not reliable.

Fine and Coarse Stereopsis

Physiological, psychophysical, and clinical findings suggest that stereoscopic perception results from the combined activity of two physiological subcomponents known as "coarse stereopsis" and "fine stereopsis."⁴³⁻⁴⁵ The physiological bases for coarse and fine stereopsis are probably the magnocellular and parvocellular subsystems of the visual pathways, respectively.⁴⁶ At any given retinal eccentricity, the fine stereopsis mechanism responds to higher spatial-frequency patterns, smaller retinal disparities (<30 minarc ['] at the foveas), and to stationary or slowly moving targets. Fine stereopsis dominates foveal vision, supports high stereoscopic acuity, and must have similarly shaped and sized images to function. Fine stereopsis probably accounts for the proportionality between stereoscopic depth and geometric disparity that is characteristic of patent stereop-

sis. It may also process the retinal disparities that control fine disparity vergence (see Disparity Vergence).

The coarse stereopsis mechanism responds more strongly to lower spatial-frequency patterns, large retinal disparities (30' to 10 degrees), and moving or flashed targets. It encompasses foveal and peripheral vision and can be activated by similarly or dissimilarly shaped targets. It may also process the retinal disparities that control coarse disparity vergence (see Disparity Vergence). The stereoscopic motion-in-depth mechanism⁴⁷ uses stereopsis to process changing retinal disparities of objects moving toward or away from the observer. (This is the same mechanism as the processing of disparities caused by stimuli from objects moving side to side or up and down.) Regan⁴⁷ suggests that the stereoscopic motion-in-depth mechanism is independent of those mechanisms that process static coarse or fine stereopsis. The independence is evident in persons who cannot see stereoscopic motion in depth in isolated portions of the binocular visual field but who can readily see static stereoscopic depth in those same portions of the binocular visual field.⁴⁷ Most persons with this stereo-motion blindness have otherwise normal binocular vision (i.e., straight eyes and normal stereoscopic acuity) and can extract depth motion percepts from the monocular looming cue.⁴⁷

Selective losses of coarse stereopsis function, called stereoblindness,⁴⁸ have been identified in some individuals. This disorder might be more properly called *static coarse stereoblindness*, because it is independent of the coarse motion-in-depth anomaly while fine stereopsis is maintained. Static coarse stereoblindness, like the coarse motion-in-depth anomaly, may appear in persons who have clinically normal binocular vision when standard examination procedures are used.⁴⁹ Some of these patients are incapable of perceiving stereoscopic depth for all coarse crossed disparities; others fail to detect coarse uncrossed retinal disparities. This asymmetry suggests that static coarse stereopsis in individuals with normal vision is further divisible into separate crossed and uncrossed mechanisms and that persons with static coarse stereoblindness do not have the use of one of those mechanisms. Coarse static stereoblind observers usually have an absence of coarse disparity vergence response for those disparities to which they are stereoblind.⁴⁹

Coarse stereopsis anomalies occur in persons who have normal stereoscopic acuity and ocular alignment⁴⁹ and in those who have no known problem related to binocular vision. Although it is conceivable that such persons experience difficulty in visual environments that emphasize coarse stereopsis function, this difficulty has not been reported. Clinically significant binocular vision anomalies such as constant early-onset strabismus often manifest as a loss of fine stereopsis (see Chapter 21).

Not only does coarse stereoblindness coexist with normal fine stereopsis, but fine stereopsis can also be lost in patients who retain functional coarse stereopsis. Rouse and colleagues⁵⁰ tested coarse static and motion-in-depth stereopsis in 11 patients with strabismus, amblyopia, or both who had little or no fine stereopsis as determined by conventional stereoscopic acuity tests. Half showed strong perceptual responses to both types of coarse stereopsis stimulus. Conventional stereoscopic acuity tests likely underestimate the stereoscopic ability of many strabismic and amblyopic patients.⁵⁰

Local and Global Stereopsis

Stereopsis also contributes to pattern recognition. Under certain viewing conditions, the distribution of geometric disparities in the binocular image can reveal the presence of forms that are all but invisible to object recognition and that do not require cues like color, contour orientation, or motion. A well-known example of stereoscopic object recognition is the random-dot

stereogram (Figure 5-7, A). When properly fused, the image of a square stands out in depth from the background. Here, stereopsis has revealed the presence of an object (the square) that is otherwise invisible.

To accomplish random-dot stereopsis, the visual system must perform extensive interocular image disparity computations across considerable extents of the binocular visual field in a process known as *global stereopsis*. By contrast, when visual cues like color and contrast reveal the presence of a form as being distinct from the background, disparity processing limited to the immediate vicinity of the form is sufficient to reveal its depth. The latter process, called *local stereopsis*, yields the depth seen in simple-line stereograms (Figure 5-7, B). Tyler⁴⁵ argues that both the fine and coarse subdivisions of stereopsis are engaged in local stereopsis but that only the fine component participates in global stereopsis. Clinical stereo tests based on global stereopsis require both fine and coarse stereopsis function and therefore are more sensitive to certain binocular anomalies than tests based on local stereopsis alone. Many

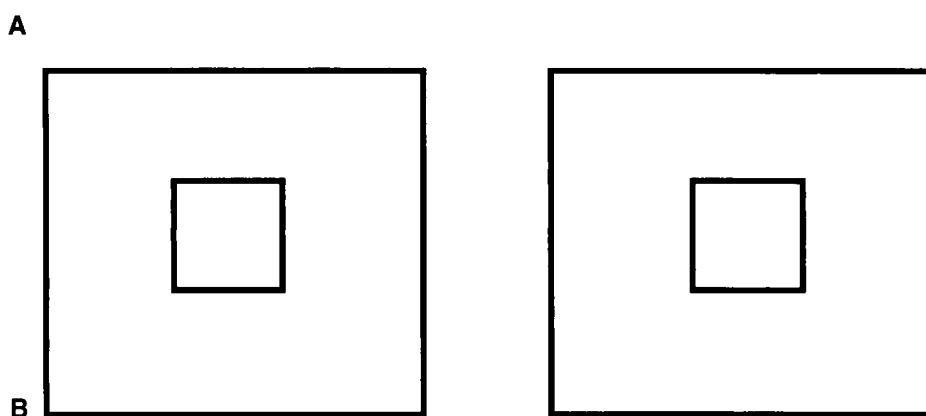


Figure 5-7

Comparison of stereoscopic depth in random-dot and line stereograms. The stereograms should each be chiastopically fused according to the instructions in the legend of Figure 5-1. A, The random-dot stereogram reveals a square standing out from the background. B, The square in the middle of the line stereogram also stands out from the background after the stereogram is properly fused. The line stereogram is the more easily fused of the two, because the local stereopsis computations required to appreciate the depth are less complex than the global stereopsis computations necessary for random-dot stereopsis.

patients with compromised binocular vision may present with slightly altered local stereopsis (e.g., those with intermittent exotropia). Other patients have significantly compromised binocular vision and essentially lack local stereopsis (e.g., those with constant early-onset strabismus). The use of random-dot stereograms (global stereopsis) and line stereograms (local stereopsis) for testing stereoscopic acuity is discussed in Chapter 21.

Stereoscopic Acuity

Stereoscopic acuity is the ability to discriminate very fine differences of depth as a result of geometric retinal disparity. Stereopsis is quantified as the minimum geometric disparity that elicits a sensation of depth. In nonstrabismic observers, the process of fine stereopsis determines stereoscopic acuity. The disparity threshold of coarse stereopsis is normally much higher than that of fine stereopsis. Foveal stereoscopic thresholds as low as 2 seconds of arc can be observed in the most accurate of normal observers.⁵¹ Disparities as low as 2 seconds of arc represent image displacements much smaller than the diameter of foveal cones. Consequently, stereopsis has earned the distinction of being called a *hyperacuity* (vernier acuity, another hyperacuity, was mentioned under Visual Localization and Visual Direction). The term *hyperacuity* implies that the observer's visual performance is better than that predicted on the basis of the diameter of foveal cones. Chapter 21 discusses the measurement of stereoscopic acuity and the factors that affect it. The effect of image defocus on stereoscopic acuity is also discussed under The Effect of Blur on Binocular Vision.

Binocular Fusion

In addition to stereopsis, normal binocular vision provides a single perceived image for most objects. The unification of the ocular images is called *fusion*. Fusion ensures that binocular visual space is faithful to object space and that the observer sees one thing when only one thing exists. The term *fusion* is sometimes used to represent two different processes: one being the construction of a single percept from two retinal images (also known as *sensory fusion*) and the other being vergence eye movement (also known as *motor fusion*). In this chapter, the term *fusion* refers to the sensory process.

The Spatial Limits of Fusion

Fusion is limited to the vicinity of the horopter in three-dimensional space in the same way that stereopsis is also limited. Fusion occurs when either corresponding points or points with small to moderate retinal disparities are stimulated. Large retinal disparities cause

double vision (diplopia), which is the opposite of fusion. Figure 5-6 shows the normal range of fusion in the visual plane as related to the ranges of patent and qualitative stereopsis.³⁰ The range of stereopsis exceeds the range of fusion; in other words, not all objects perceived stereoscopically are seen as single. The range of depth in object space that is fused without the aid of eye movement is known as *Panum's space*. The portion of retina that is optically conjugate to Panum's space is known as *Panum's area*. Panum's area is defined as an area of the retina of one eye, any point of which gives rise to a percept of singleness when stimulated simultaneously with a single point on the retina of the fellow eye. The spatial relationship of Panum's area to Panum's space is illustrated in Figure 5-8.

Images fused in Panum's areas on noncorresponding points raise a possible paradox, because a single object is formed that potentially has two different visual directions: that of the right eye and that of the left eye. The visual system resolves this paradox by averaging the right- and left-eye visual directions. As a result, the fused image has an egocentric direction that is intermediate between the directions of the right and left eyes. This directional averaging process is known as *allelotropia*.⁵² In individuals with normal vision, the ocular image directions are averaged symmetrically; in those with strong ocular dominance (e.g., in binocular anomalies),

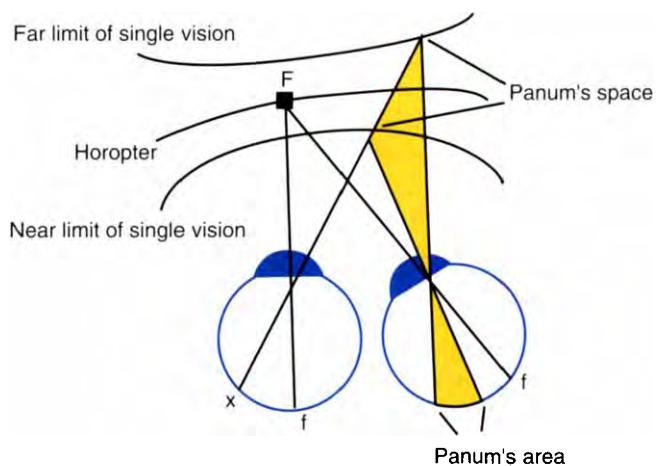


Figure 5-8

Visual plane drawing of the geometrical relationship of Panum's area (on the retina) to Panum's space (in object space). Point x in the left temporal hemiretina gives rise to a percept of singleness when stimulated at the same time that any one point in the Panum's area of the right eye is stimulated. Visual lines extrapolated from the limits of the Panum's area in the right eye reveal the far and near limits of Panum's space by their intersections with the visual line of point x of the left eye. The horopter is situated between the limits of Panum's space. f, Fovea; F, fixation point.

the averaging of direction is weighted in favor of the dominant eye.⁵³

Retinal eccentricity, the spatial frequency content, and spatial textures near the stimulus are variables that affect the size of Panum's areas. The retinal eccentricity and the spatial frequency variables bear directly on clinical practice, because they affect diplopia.

The size of a Panum's area, like the stereoscopic threshold, is a function of retinal eccentricity.³³ The ranges of fusion and stereopsis are at a minimum in foveal vision and expand in peripheral vision. Because of the smaller size of Panum's areas in foveal vision, patients' reports of diplopia usually result from foveal disparity rather than peripheral disparity. This pathological diplopia is foveal diplopia associated with anomalous binocular vision, usually strabismus. Occasionally, normally binocular patients may notice physiological diplopia. Physiological diplopia is associated with nonfixated peripheral objects that are remote from the horopter. Physiological diplopia can be distinguished from pathologic diplopia by careful questioning. Patients who have experienced physiological diplopia report that the diplopia was associated with objects that they were *not* fixating (i.e., in peripheral vision). Patients should be reassured that their physiological diplopia is normal. On the other hand, a patient who reports that directly viewed objects are seen as double may have pathological diplopia related to misalignment of the eyes and should receive a careful binocular vision examination.

A second factor used for determining Panum's area size is spatial frequency. *Spatial frequency* refers to the composition of spatial detail in a target's image. Higher spatial frequency makes up finer detail. The spatial frequency content of a fixation target affects the likelihood that the target will stimulate diplopia when it is imaged onto disparate retinal points. To test the effect of spatial frequency on Panum's area size, Schor and associates⁵⁴ used difference-of-Gaussian (DoG) targets* (Figure 5-9). Unlike a sharp-edged line target of a given width, a DoG target of the same width contains a small range of spatial frequencies. Schor and colleagues observed that the size of Panum's area changed inversely with DoG spatial frequency for spatial frequencies below 2.4 cycles/degree. In other words, diplopia is perceived more easily with intermediate and fine spatial structure than with coarse spatial structure. Curiously,

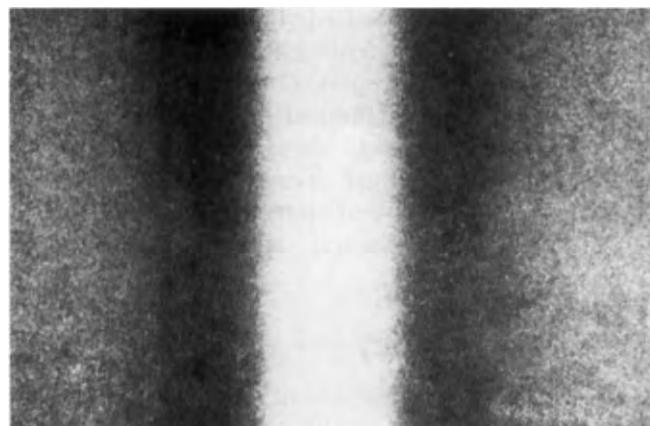


Figure 5-9

Vertical difference of Gaussian (DoG) bar target. DoG targets are useful for selectively stimulating isolated spatial frequency mechanisms during perceptual and oculomotor studies. See the text for explanation of DoG targets. (Courtesy of Dr. Michael Wesson.)

high-spatial-frequency targets yield the same size for Panum's area as do middle-spatial-frequency targets. This suggests that a diplopia threshold obtained from a wide, sharp-edged, bar target is determined principally by the edges of the target, which are composed of mid-range and high spatial frequencies. This finding suggests that clinical tests using a diplopia end point, such as the near-point-of-convergence test or a prism vergence test, should use targets containing middle and high spatial frequencies (i.e., fine detail) to obtain the highest sensitivity to the breakdown of binocularity.

The multiplicity of variables influencing Panum's area size may partly explain the wide range of values reported (2 min of arc to 2 degrees of arc) for the foveal size of Panum's areas in normal observers.¹⁴ In addition to features of the target itself, spatial textures near the fixation target can influence the threshold of diplopia.⁵⁵ Therefore, target elements used as diplopia probes should probably be isolated from neighboring fusion textures.

The Nonspatial Limits of Fusion

In general, the ocular images become more difficult to fuse as image *dissimilarity* increases. When ocular image differences are small and fusible, the visual system typically strikes a compromise between the image differences when constructing the binocular image. This compromise is essentially a perceptual averaging process. Luminance averaging (e.g., white + gray = light gray) occurs in the luminance domain, and color averaging (e.g., red + green = yellow) occurs in the color domain. Not surprisingly, the visual system averages the small luminance and color differences that commonly occur in a natural environment, such as when reading

*A DoG target is constructed from a broad, dark bar of moderate peak luminance and a narrower, bright bar of high peak luminance. Luminous intensity across each individual bar varies as a Gaussian function of position, which gives them the appearance of being "fuzzy." The luminance across the combined bars varies as a function of the difference of the Gaussian luminance distributions. DoG target spatial frequency is controlled by altering the width of the dark and light Gaussian bars.

a book illuminated by an oblique light source. Because of binocular parallax (as a result of the separation of the eyes in the head), one eye may receive more reflected light from an oblique source than does the other eye and, consequently, the book appears brighter to one eye than the other; with binocular vision, the book appears to have a luminance intermediate between the monocular extremes. Over time, differing retinal adaptation also causes a shift of color perception called the *Bezold-Brücke phenomenon*,⁵⁶ in which the dimmer image appears pinkish-white and the brighter image blueish-white. The resulting color difference is also fused to an intermediate value.

Occasionally retinal image differences occur that are so great that the brain cannot reconcile the differences. When this occurs, one of two physiological responses takes place: either (1) diplopia and binocular rivalry or (2) sustained suppression. When parts or all of the ocular images contain high luminance contrast and chromatic contrast but are spatially different, the patient experiences diplopia and binocular rivalry. *Binocular rivalry* is the alternating perception of two different objects in the same visual direction. When rivalry occurs, one eye's image dominates perception while the other eye's image is momentarily suppressed; however, during the next moment, the dominance of the eyes is reversed. Rivalry suppression is spatially limited to the region of rivalrous contours, but the size of rivalry suppression regions increases with retinal eccentricity.⁵⁷ These rivalry suppression regions are larger than Panum's areas in the same part of the visual field and are differently shaped (round) than Panum's typical elliptical areas.⁵⁸ Diplopia and rivalry may occur when multiple unfusible objects are visible in the visual field. Diplopia and binocular rivalry are necessary elements of normal binocular vision and allow the visual system to manage the images of objects situated outside of Panum's space. Because this diplopia and rivalry occur away from the point of fixation in those with normal vision, it is not usually noticed.

Large differences in color between the ocular images also induce binocular rivalry. When this occurs, the observer alternately perceives the different colors. A natural stimulus for color rivalry can occur when spatially separated objects of very different color, far from the horopter, happen to cast their images onto corresponding points. In clinical practice, color rivalry may be encountered by patients wearing red/green anaglyph glasses as in the Worth four-dot test or in certain stereograms. The colors in red/green anaglyph glasses are at the threshold of color rivalry for the average normal observer, so one normal observer may experience red/green rivalry, whereas another may experience color averaging when viewing the same red/green stereogram.

The process of fusion may vary over visual space or time. For instance, dissimilar ocular images may

produce transient fusion when initially falling on corresponding points; however, to sustain fusion, the images must be similar.⁵⁹ Also, fusion may occur for some aspects of stimuli and not others. For example, targets with similar contours and textures but dissimilar colors (e.g., red and green) may elicit fusion of the textures with stereopsis, even though the colors do not fuse.¹⁴ The differing colors appear alternately in perception in the form of color rivalry. The coexistence of contour fusion and stereopsis with color rivalry is what makes it possible for red/green anaglyph methods to be used in binocular vision testing and visual training. However, clinical devices employing red/blue anaglyph technology should be used with caution, because the large luminosity and focus differences between red and blue targets may induce suppression rather than alternate perception and prevent the desired fusion and stereopsis (J. Richman, personal communication, April 15, 1996). Fusion may differ over a visual field in that the fusion of similar images may occur in one portion while binocular rivalry occurs in a neighboring portion of the binocular visual field.¹⁴

Fusion also may vary as a result of luminance differences that occur in natural circumstances. *Luminance luster* occurs when an observer sees a large interocular luminance difference in one portion of the binocular visual field and little or no difference in the remainder of the binocular visual field. As a result of the rivalry, the difference region is perceived to shimmer. Luminance luster occurs in nature when viewing a small highly polished reflective surface such as a facet on a diamond. The luminance difference arises because the facet reflects a narrow beam of light to one eye but not to the other.

When one ocular image has higher contrast and edge sharpness than the other in a patient with normal binocular sensory function, those aspects of the weaker image that cannot be reconciled with the stronger image are continuously suppressed.^{60,61} A common cause of this behavior is uncorrected *anisometropia* (a difference in refractive errors between the eyes of 1 D or more). The degraded middle- and high-spatial-frequency features of the blurred image have low contrast and are suppressed, whereas the low-spatial-frequency features of the two images, which are minimally affected by blur, have high contrast and are fused. The effects of blur on binocular vision are addressed below under The Effect of Blur on Binocular Vision and also in Chapter 21.

Theories of Single Binocular Vision

The question of how the brain derives a single perceived image from two retinal images has long been debated. The question has been reviewed by Howard and Rogers,¹⁴ Ono,¹⁵ and von Noorden.²⁵ Howard and Rogers have condensed the debate into four hypotheses:

(1) the mental theory, (2) the suppression theory, (3) the two-channel theory, and (4) the dual-response theory. The mental theory, championed by Helmholtz, claims that we see double at birth, even when images are on the horopter, but we learn to ignore one image, just as adults ignore physiological diplopia to the side of fixation. The adherents of the suppression theory argue that we alternately suppress the ocular images but retain stereopsis by extracting disparity from the remembered image of the suppressed eye and the visible image of the nonsuppressed eye. Howard and Rogers present numerous pieces of evidence that weigh against the mental and suppression theories. The two-channel theory suggests that similar textures imaged onto corresponding points stimulate rivalry in one neural channel and simultaneously stimulate fusion with stereopsis in another neural channel. When a texture activates the fusion/stereopsis channel, that activity masks the perception of rivalry happening in the rivalry channel. The dual-response theory holds that similar images falling on corresponding points are fused with stereopsis (i.e., the images are combined under the rules of luminance averaging, color averaging, and allelotropia) and *without* rivalry. Rivalry only occurs when *dissimilar* images fall onto corresponding points. Hence, rivalry and fusion are viewed as mutually exclusive behaviors of a single neural process under the dual-response theory. Howard and Rogers conclude that the dual-response theory best explains the available evidence, but they acknowledge that the dual-response theory may only apply to achromatic pattern mechanisms in vision, whereas chromatic mechanisms may follow different rules. This conclusion would be in harmony with the fact that simultaneous fusion, stereopsis, and color rivalry are experienced by patients viewing clinical red/green anaglyph targets.

BINOCULAR MOTOR FUNCTION

Eye Movements

All human eye movements have one of two functions: (1) to support the high resolution of foveal vision or (2) to prevent neural blurring of images due to retinal image motion.⁶² These functions are accomplished by six types of movement, each type having discrete supranuclear neural control mechanisms and, to a degree, distinct clinical anomalies: (1) visual fixation, (2) vestibulo-ocular response (VOR), (3) optokinetic nystagmus, (4) saccades, (5) pursuits, and (6) vergences.⁶² Vergence eye movements in particular are prone to functional anomalies. This section briefly describes the various types of eye movements as a basis for understanding the role of vergence eye movements, and it then discusses vergence eye movements in greater detail. See Ciuffreda and Tannen⁶³ and Leigh and Zee⁶² for comprehensive discussions of eye movements.

Specification of the Direction of Gaze

Eye movements are quantified by rotation of the direction of gaze. The direction of gaze is most accurately represented by the *fixation axis*, a straight line that extends from the center of rotation of the eye to the point of fixation. When the fixation axis is perpendicular to the plane of the face, the eye is said to be in the primary position of gaze. All other directions of gaze are eccentric.

The center of rotation is usually considered to be about 13 mm behind the cornea, in the anterior vitreous.⁶⁴ Because the location of the center of rotation of the eye is difficult to determine outside of a laboratory environment, the line of sight is used to define the direction of gaze in clinical settings. The line of sight is a straight line that extends from the patient's point of fixation to the center of the entrance pupil of the patient's eye (the pupil as seen by the examiner).⁶⁵ Because the entrance pupil is closer to fixation targets than the center of rotation of the eye, the measurement of eye movement demand at the entrance pupil slightly overestimates the angular rotation of the eye. This overestimation is not significant for most clinical purposes.

In addition to rotation, the eye makes small translations (i.e., positional displacements) within the orbit that are associated with rotation. The impact of these translations on perception is minimal.

Eye Movements Supporting Foveal Vision

Several types of eye movement support foveal vision.⁶² Saccadic eye movements are the fastest of all eye movements, with velocities of up to 700 degrees per second.⁶⁶ Saccades move off-foveal images to the foveas by means of a sudden shift of the direction of gaze. Saccades are conjugate eye movements, and they rotate the eyes equally and in the same direction. They can be reflexive, responding to the sudden appearance of a target in the peripheral visual field, or they may be purely voluntary, produced at will by the observer, with or without a visible target.⁶²

Smooth-pursuit movements are also conjugate eye movements, and they keep the images of relatively slowly moving targets (up to about 40 degrees per second)⁶⁷ on the foveas. Smooth-pursuit eye movements are smooth rotations of the eyes rather than sudden shifts; they are stimulated when the observer looks at a moving target. Pursuit movements do not occur for imagined or remembered stimulus movements. After a moving target is chosen as a fixation target, the resulting smooth movement is controlled automatically as long as attention is held on the target. Therefore, smooth-pursuit eye movement is partly an attentive response and partly a reflexive response, and it is a form of behavior called the *psycho-optic reflex*. Psycho-optic reflex movements may be distinguished

from purely reflex movements (e.g., the pupil's response to light) that involve no conscious effort or control and from purely voluntary movements (e.g., saccades in total darkness) that require conscious effort both to initiate and to sustain the oculomotor innervation.

Vergence-step movements shift off-foveal images to the foveas by suddenly changing the distance of gaze, and they require convergence or divergence. Vergence steps are disjunctive movements, because the fixation axes move in different directions. Their neurological control somewhat resembles that of saccades,⁶⁸ but they have much lower peak velocities than saccades (70 degrees per second).⁶⁹ Vergence pursuit movements are also disjunctive movements that are analogous to smooth pursuits; they keep the images of objects slowly moving in depth on the foveas. Vergence eye movements are discussed in detail under Horizontal Vergence Eye Movements because of their intimate relationship with accommodation.

Eye Movements Supporting Stable Retinal Imagery

Vestibulo-ocular and optokinetic eye movements are conjugate eye movements that minimize retinal image motion. Vestibular eye movements are stimulated by the effects of head motion on the vestibular apparatus of the inner ear, and they are purely reflexive. The VOR quickly responds to brief and unintended rotational and translational head movements, such as those that occur when walking or running.⁶² In so doing, the VOR allows for the continuation of fixation on a point of interest that might otherwise be lost because of unintended head movement. The short latency of the VOR—16 msec⁷⁰—allows it to react more quickly to head position disturbances than visually elicited movements. Because the VOR serves to maintain fixation, it is suppressed during saccadic eye movements, which serve to change fixation.

The magnitude of eye rotation stimulated by a given head rotation is known as *VOR gain*. The VOR gain is calibrated by visual experience.⁷¹ Accordingly, it is increased for near viewing distances, because a given head perturbation displaces the retinal images of near objects more than it does the retinal images of far objects.⁷² A second factor that requires the adjustment of VOR gain is new spectacles.⁶² Rotating the head while viewing through spectacles requires more VOR-mediated ocular rotation for hyperopic spectacles and less VOR-mediated ocular rotation for myopic spectacles than for no spectacles as a result of the prismatic effects of the lenses. In the case of hyperopes, plus lenses magnify optical eye-movement demands. When the spectacle-corrected hyperope's head moves during the fixation of a static object, the magnified eye movement demand requires a larger VOR eye movement to maintain fixation than would the same head movement

without the spectacles. The minimization of eye-movement demand by minus lens spectacles explains the reduced VOR gain of spectacle-corrected myopes. Most persons rapidly adjust their VOR gains to maintain accurate VOR behavior through new spectacles.⁷³ However, patients experiencing large changes of prescription (e.g., a new bifocal prescription with a large reading segment) or who have slower-than-normal VOR adaptation may experience disorientation symptoms (e.g., slight dizziness, vertigo, nausea) because of conflicts between visual and vestibular cues to motion perception. The adjustment of VOR gain in these cases is usually completed over a several-day period.

When the body is rotated in space, the vestibular apparatus initiates *vestibular nystagmus*. This movement is composed of two segments: (1) a smooth and slow motion of the eyes in the direction opposite to body rotation and (2) a saccadic movement in the same direction as the body rotation. The smooth component maintains momentary stability of the retinal image as the head turns (like the VOR), whereas the saccadic movement resets the eyes to a new orbital position in preparation for another smooth motion.

If a stationary observer views persistent unidirectional movement of large objects (e.g., the passing scenery viewed from a train), *optokinetic nystagmus* occurs. The optokinetic movement is also composed of two segments: (1) a smooth and slow motion of the eyes in the direction of target motion and (2) a fast, saccadic-like movement in the direction opposite of target motion. These two components serve the same purpose as the analogous components of vestibular nystagmus: maintaining stable retinal imagery. Leigh and Zee⁶² observed that vestibular and optokinetic innervation serve complementary roles during whole-body rotation. Vestibular innervation controls the initial nystagmus but then decays as optokinetic innervation gains in magnitude. The sum of the two innervations closely follows target motion. Likewise, smooth pursuit and optokinetic innervations play complementary roles as the stationary observer views moving scenery, with smooth pursuit generating the first few seconds of nystagmoid movement and optokinetic generating the remainder. The transitions of these complementary innervations from the first to the second are not generally visible by direct observation of the eyes.

Vertical Eye Movements

In most respects, vertical conjugate eye movements behave like horizontal eye movements, but with slightly lower velocity, gain, and range of movement.⁶² With the appropriate stimuli, one can elicit vertical saccades, vertical smooth pursuits, vertical optokinetic nystagmus, vertical vestibulo-ocular reflex, and vertical vergence. At the cortical and collicular levels, a common visuospatial map determines vertical and horizontal movement;

however, at the supranuclear level, separate neural centers control the innervations for the vertical and horizontal conjugate gazes.⁶³

Torsional Eye Movements

Torsional eye movement is a rotation of the eye around the fixation axis. Torsional eye movements are purely reflexive under normal viewing circumstances. Two types of torsional eye movements exist: (1) cycloversion (conjugate) and (2) cyclovergence (disconjugate). Cycloversional movements are conjugate movements in which the vertical meridians of the retinas are rotated in the same direction and by the same amount. Cycloversions attempt to maintain the vertical meridians of the retinas on the objective vertical when the head tilts toward a shoulder. In humans, cycloversions fall far short of righting the vertical meridians of the retinas⁷⁴; the normal perception of verticality during head tilt must therefore arise from perceptual mechanisms. Although cycloversions play a minor role in normal ocular motility in humans, they serve as the basis of the Bielschowsky head tilt test, which determines the identity of the offending extraocular muscle in cyclo/vertical muscle paralysis.²⁵ In cyclovergence, the vertical meridians of the retinas rotate in opposite directions. Cyclovergence movements serve to compensate for cyclophorias.²⁵ Cyclophoria is a tendency of the vertical meridians of the retinas to deviate from parallelism in binocular vision, which becomes manifest in the absence of fusion.

Hering's Law of Equal Innervation

During the 19th century, Hering³⁸ proposed that the eyes are physiologically yoked together like a team of horses. Muscles having the roles of moving the eyes in the same direction (e.g., the right eye's lateral rectus and the left eye's medial rectus) were postulated to receive equal innervation. The law of equal innervation is the motor embodiment of cyclopean vision, because the eyes receive a single motor command that alters the cyclopean direction of gaze. The cyclopean direction of gaze is the egocentric direction of the point of fixation. The single motor command is subsequently split into equal right eye and left eye copies at a lower level in the brain. Hering also proposed that the yoking of muscles is different for convergence movements and that the medial recti are yoked together. When convergence occurs, the cyclopean eye receives a single command representing the intended distance of fixation, which is subsequently split into separate left-eye and right-eye components. Eye movements that require shifts in both the direction and the distance of gaze are a result of a combination of yoked conjugate innervation and yoked disjunctive innervation.

Major deviations of oculomotor behavior from Hering's law invariably reveal a motor anomaly. The

logic of Hering's law is applied clinically in tests such as the alternating cover test in different fields of gaze⁷⁵ (Chapter 10). For instance, Hering's law predicts that, if one eye is stimulated to make a purely lateral gaze shift while the other eye is occluded, the occluded eye should move in the same manner as the seeing eye. The failure of the occluded eye to make the same movement as the seeing eye reveals a damaged muscle or efferent nerve. The failure of the eyes to make a conjugate rotation in conjugate stimulus conditions is known as a *noncomitant* (or incomitant) movement. The alternating cover test is used to measure the magnitude of difference in the primary lines of sight. If Hering's law is sustained, an angle between the lines of sight measured in one direction should be the same as in any other direction. By convention, a change of 10° or more in the difference between the eyes may be regarded as an incomitant deviation.¹²

Horizontal Vergence Eye Movements

The purpose of vergence eye movements is to provide appropriate convergence and divergence for the eyes. In so doing, vergence eye movements put fixation targets on the horopter and keep them there. The vergence response of the eyes is determined by a composite of several underlying vergence innervations, most of which are evoked by "cues." These cues are identifiable features of the visual environment that usually correlate with target distance and are similar to those that evoke depth and distance perception.

Historically, two different views of the vergence system were proposed, one by Maddox⁷⁶ and another by Fincham and Walton.⁷⁷ Maddox proposed that vergence eye movements are driven by the sum of four innervations: (1) tonic, (2) proximal, (3) accommodative, and (4) fusional, with accommodation fundamental. On the other hand, Fincham and Walton emphasized that accommodation may be driven as a result of convergence movements of the eyes with vergence as the fundamental component. Clinicians use the accommodative convergence in prism diopters (Δ) per diopter (D) of accommodation (Δ/D, or ACA) ratio from Maddox's theory and, to a lesser extent, the convergence accommodation per convergence (D/Δ, or CA/C) ratio from Fincham and Walton. Maddox's theory was presented first and also has gained acceptance from clinical and research perspectives as a useful method for considering ocular vergence function.⁷⁸ The CA/C ratio is challenging to assess in a clinical setting and, thus, the model of Fincham and Walton is less used. Modern assessment of the ocular vergence system considers both accommodation and vergence as fundamental components of the vergence system⁷⁹ and uses a dual-interaction model combining the ideas of Maddox with those of Fincham and Walton.

Maddox Model of Vergence Movements

The function of the Maddox model components can be summarized as follows. *Tonic vergence* serves to provide a steady platform of innervation from which other vergence innervations can be efficiently launched.⁸⁰ *Proximal vergence* adds additional vergence innervation for near viewing when targets appear close to the observer.⁸¹ *Accommodative vergence* also adds additional vergence innervation for near viewing when accommodation responds to blur. *Fusional vergence* completes the convergence response by supplying any additional innervation required to attain single binocular vision. Maddox's model of vergence function served for many years as a conceptual basis for understanding vergence eye movements and for solving clinical problems. The Maddox model is still basically correct, but it is now clear that the generation and integration of vergence innervation is more complex than the Maddox model and involves more interaction with the vergence system than was once thought.

Tonic innervation causes the eyes to assume a position that differs from what would occur during death or deep anesthesia.⁸⁰ The divergent position of the eyes in death or anesthesia is a result of mechanical influences on the oculomotor system. Tonic innervation provides a neutral or starting position for the eyes and provides the physiological resting position of the eyes. The amount of tonic innervation to the eyes may vary, depending on the age of the patient, stress, the influence of drugs or alcohol, the nature of the visual environment (illumination level, retinal eccentricity of stimuli) and previous visual experience.⁸⁰ Tonic innervation biases the vergence system toward the resting position and is an important component in the determination of oculomotor position.

Proximal vergence stems from an awareness of nearness and is thought to provide convergence innervation to the eyes whenever a near object is being viewed.⁸¹ To the extent that the object is actually where it is perceived, proximal vergence therefore assists the oculomotor system by providing a portion of the necessary innervation to fuse the targets. In certain situations, proximal vergence may contribute up to about 50% of the necessary convergence for a near target.⁸¹ Proximal convergence is generally thought to be a result of distance estimation related to the apparent size or nearness from the convergence changes,^{82,83} and it is not necessarily related to the amount of accommodation. In certain situations, an observer may incorrectly perceive the nearness of an object. In these cases, proximal vergence may actually be counterproductive by providing an inappropriate response of the system.

Proximal vergence is frequently estimated by comparing the results of the far-near AC/A ratio to the gradient AC/A ratio (see Accommodative Vergence). The AC/A ratio is a fundamental aspect of an individual's

oculomotor system, and it is derived by measuring the change in vergence related to the change in accommodation when fixation is altered from one distance to another (i.e., the far-near AC/A). The gradient AC/A is also calculated by assessing the change in vergence related to the change in accommodation of the eyes, but the distance of the target is held constant, and lenses are interjected into the lines of sight of both eyes to alter the magnitude of accommodation. In the far-near AC/A, proximal vergence affects the near assessment of vergence, but it does not significantly affect the distance assessment. In the gradient AC/A, proximal vergence affects both assessments (i.e., before and after the insertion of the lens pairs) of vergence equally, because the distance is held constant. The AC/A values calculated by these two means is usually different, with the far-near AC/A ratio being substantially higher. The difference in AC/A ratios is usually attributed to the unbalanced effects of proximal vergence.

Accommodative vergence in the Maddox classification system is quantified by the AC/A ratio and, along with miosis of the pupil and accommodation, forms the *near triad*. (The convergence aspect of the near triad involves more than accommodative vergence, of course.) Accommodative vergence in Maddox's concept⁷⁶ adds to tonic and proximal vergence to further bring the eyes into alignment with a near stimulus. Accommodative vergence is most obvious when the vergence relationship of the eyes changes as a result of changes in accommodation when a patient views a fixed target through different sets of lenses, and it is a common factor in the clinical analysis of vergence function. Inappropriate levels of accommodative vergence are frequently encountered in cases of binocular visual dysfunction.

Fusional vergence is the final component of vergence in the Maddox classification. Fusional vergence is a flexible and powerful component that alters the vergence level of the eyes to achieve fusion. It adds convergence to the system when tonic, proximal, and accommodative vergence do not provide sufficient convergence of the eyes for the stimuli. On occasion, tonic, proximal, and accommodative vergence may provide excessive convergence of the eyes for a given stimulus, and fusional vergence may act to diverge the eyes sufficient to fuse the stimulus.

Although it is not a pure assessment, fusional vergence is usually considered to be measured with prisms, and it is closely related to prism adaptation. As prisms in the line of sight of one (bar prism) or both (Risley prisms) eyes are slowly increased (about 3° per second), the patient encounters a point at which blur occurs; the blur finding is generally considered to indicate the limits of fusional vergence. The additional change in vergence after the target is blurred indicates additional vergence as a result of changes in accommodation, and

it precedes the loss of fusion with diplopia. The point at which diplopia occurs—the *break finding*—indicates the limits of accommodative and fusional vergence. After diplopia occurs, a reduction of the prisms allows an assessment of the magnitude of prism before the eyes when recovery occurs. These vergence ranges (blur, break, and recovery findings) are used to assess positive (convergence) and/or negative (divergence) vergences at any distance.

Systems Analysis

Systems analysis is the application of cybernetic principles to the analysis of systems such as that which occurs in the motor response of the eyes during accommodation and vergence. *Cybernetics* is the science of communication, organic processes, or automated mechanical or electronic control systems, and it provides a useful way of conceptualizing accommodation and vergence function. Systems analysis also provides simplifying concepts such as *gain* and *feedback* (to be described later), which are useful for understanding both the normal and abnormal function of the vergence system of the eyes.

Complex models of vergence and accommodation function can be simplified by visualizing them in box diagrams called *system diagrams* (Figure 5-10). System diagrams represent physiological processes, and they do not necessarily represent anatomy, although they often have anatomical implications. The precise anatomy is often unknown, and the boxes represent physiological mechanisms that accomplish tasks. Examples of physiological processes are the visual system neurons that convert retinal blur into accommodative innervation or the extraocular muscles that move the eyes.

In a systems diagram, the lines between boxes represent communication paths between physiological mechanisms. Sometimes this communication is accomplished by a discrete neural pathway. For instance, the line connecting the disparity vergence (DV) mechanism to the extraocular muscle (EOM) mechanism carries a message of disparity vergence motor innervation to the muscles by way of the oculomotor nuclei (the nuclei are not shown). In other cases, the communication represented by a line is nonneural, such as the effect of vergence eye movement on the retinal disparity processed by the disparity vergence mechanism. Circles in the

diagram represent the combination of messages, such as the summation of motor innervation in the Maddox model. The combination of messages is not necessarily a linear summation.⁶³ The construction of systems models of accommodative and vergence function provides a conceptual presentation of the interactions between different portions of a system. As such, the model guides expectations about the relationship between the factors involved in the normal and abnormal workings of the oculomotor system for clinicians and research activities alike.

The following paragraphs present a model of vergence and accommodation function. Each step of the process will be represented by expanded system diagrams demonstrating the neurological linkage of vergence to accommodation. Although this chapter is primarily concerned with vergence activity, accommodative elements are necessary for understanding accommodation/vergence interactions. The model reflects vergence system function based on models in the research literature,^{81,84–86} and it presents an overview of binocular motor function to provide a logical basis for understanding normal binocular motor function as well as binocular anomalies. Nonquantitative systems analysis will be used to explain normal accommodation and vergence function, to interpret the physiological basis of phorometric tests, and to describe the pathophysiology of two common binocular anomalies using the model. System diagrams are also used to explain the behavior of accommodation and vergence during phorometry in Chapter 21, and they have been used as a tool for analyzing clinical problems.⁸⁸ Accommodation is discussed in detail in Chapter 4 and will only be addressed in this chapter as it relates to vergence function.

Disparity Vergence

Retinal disparity stimulates disparity vergence innervation (or, simply, disparity vergence). Crossed retinal disparity stimulates convergence, and uncrossed retinal disparity stimulates divergence. Because disparity vergence is the only form of vergence innervation that directly responds to retinal disparity, it is primarily responsible for maintaining binocularity by reducing retinal disparity to a minimum. All other forms of vergence innervation play a support role for disparity vergence.

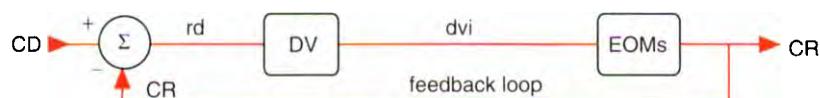


Figure 5-10

Simplified box diagram representing feedback control in disparity vergence. See the text for further explanation. CD, Convergence demand; Σ, summation; rd, retinal disparity; DV, disparity vergence; dvi, disparity vergence innervation; CR, convergence response.

The stimulus for disparity vergence is *retinal disparity*. Retinal disparity is a measure of the distance of an object from the horopter. Any object not on the horopter stimulates noncorresponding points. Retinal disparity describes the spatial relationship of retinal images to corresponding points. An object closer to the observer than the horopter subtends crossed retinal disparity, because each image is perceived to be on the opposite side of the body from the eye that perceives it. An object farther from the observer than the horopter causes uncrossed retinal disparity and indicates an object being viewed that is more distant than the horopter. Retinal disparity is not the same as fixation disparity. (Fixation disparity is a micromisalignment of the eyes during fusion as a result of Panum's fusional area.)

Disparity vergence is a psycho-optic reflex controlled by the magnitude and sign of retinal disparity associated with the intended fixation point. Attention is necessary only in the formation of the intent to fixate the stimulus. After the fixation point has been selected, the reflexive behavior of disparity vergence takes over and frees attention from the act of convergence; this allows attention to be concentrated on visual information processing.

A misconception regarding disparity vergence that dates back at least to Maddox⁷⁶ is that it is stimulated by double vision and that therefore its purpose is to restore fusion; this is why the term *fusional vergence* is sometimes used to refer to this type of vergence. However, binocularly driven vergence behaves as though it is driven by retinal disparity, regardless of whether there is associated diplopia.⁸⁹ For instance, disparity vergence can be activated by retinal disparities that are too small to cause diplopia. Stark's observation clarifies the purpose of disparity-vergence eye movements, which is to place targets of interest on the horopter so that maximum stereoscopic acuity can be achieved. This requires greater vergence accuracy than that required for fusion. Smaller magnitudes of diplopia may initiate disparity vergence under some circumstances, but the origin of vergence innervation in the face of a large magnitude of diplopia likely is voluntary rather than reflexive (see Voluntary Vergence below). Another common misconception regarding disparity vergence is that it is stimulated directly by stereoscopic perception. In fact, disparity vergence can be activated by a single luminous point situated off of the horopter (and visible to one eye), whereas stereoscopic perception requires a minimum of two points in space (i.e., geometric disparity) to be appreciated. Although stereopsis may not directly stimulate disparity-vergence eye movement, stereopsis may indirectly activate vergence by contributing to proximal vergence. Disparity vergence innervation and stereoscopic perception may both be derived from the activity of disparity-detecting binocular neurons in the visual cortex.⁹⁰

Disparity vergence is not a single physiological entity. Two antagonistic mechanisms—positive disparity vergence (i.e., convergence) and negative disparity vergence (i.e., divergence)—drive the eyes inward and outward, respectively.⁴⁹ Separate brainstem cellular groups called *convergence cells* and *divergence cells* innervate positive and negative disparity vergence, respectively.⁹¹ The number of divergence cells is significantly less than convergence cells, which may explain the lower amplitude and velocity of divergence movements. In addition, both convergence and divergence exhibit behaviors that suggest that each is further subdivided into components that are analogous to coarse and fine sensory function.⁹² Coarse disparity vergence is activated by large targets and large retinal disparities, and it responds to similar and dissimilar retinal images. Fine disparity vergence is optimally stimulated by small targets and small retinal disparities, and it requires similar images (like fine stereopsis). These two mechanisms share the responsibility for controlling vergence eye movement. Coarse-disparity vergence innervation initiates large-disparity vergence movements and then dissipates, earning it the alias *transient disparity vergence*.⁵⁹ Fine-disparity innervation completes vergence movements begun by coarse-disparity vergence, and it helps to maintain steady vergence, earning it the alias *sustained disparity vergence*.⁵⁹

The disparity-vergence mechanism may suffer partial or complete loss of function analogous to that of the stereoblindnesses. Persons who are stereoblind for coarse disparities fail to produce coarse disparity vergence movements from those disparities⁵¹; fine-disparity vergence behavior and stereoscopic acuity are usually preserved in these cases. Some patients with constant strabismus retain a limited capacity for a disparity-vergence response.⁹³ Because constant strabismus inevitably obliterates fine-disparity vergence, this residual disparity vergence is likely coarse-disparity vergence.

Feedback Control of Disparity Vergence and Fixation Disparity

The generation of fine disparity vergence innervation is modulated by a process called *negative feedback*. Negative feedback is a process in which a motor response reduces the stimulus that created it. For instance, crossed retinal disparity stimulates the production of positive disparity vergence innervation, which converges the eyes; increased convergence subsequently reduces crossed retinal disparity. If the magnitude of the initial convergence is not correct for precise ocular alignment, the retinal disparity/vergence response sequence is reiterated until retinal disparity is reduced to a minimal value. The process of negative feedback is described in Figure 5-10. A patient switching attention from a far point to a near point encounters a large positive

convergence demand (CD). The retinal disparity (rd) subtended at the observer's eyes by the near fixation target is the difference between the CD and the current convergence response (CR). The difference operation is represented by the subtraction of the CR from the CD at the circle labeled " Σ ." Because CR is initially zero, rd is large. The disparity vergence mechanism (DV) processes the retinal disparity and subsequently generates disparity vergence innervation (dvi), which is sent to the extraocular muscles (EOMs). The resulting CR reduces retinal disparity by way of the feedback loop; the feedback loop represents the ability of the convergence response to change retinal disparity. The minus sign at the summation circle indicates that positive vergence *reduces* retinal disparity. This feedback process is carried on continuously as fine-disparity vergence strives to reduce retinal disparity to a minimum. If the feedback loop functions so that the CR of the eyes reduces the retinal disparity, the feedback mechanism is described as *closed-loop*. If vergence were prevented from changing retinal disparity (e.g., by covering one eye), the feedback mechanism would be described as *open-loop*. An open-loop system does not have a method to adjust the system output to achieve a balance. Open-loop systems are less stable than closed-loop systems, because a counter for the stimulus does not function.

The negative feedback process poses a paradox: if disparity vergence is entirely successful in reducing retinal disparity, there is no longer a stimulus of disparity vergence, and fine-disparity vergence innervation dissipates. In practice, retinal disparity is not reduced precisely to zero by fine-disparity vergence. A slight deviation of the visual axes from perfect bifixation is maintained by the disparity vergence mechanism when disparity vergence innervation is demanded. This deviation, called *fixation disparity* (FD), causes a residual foveal retinal disparity (equal in size to the fixation disparity) that stimulates the continuous production of fine-disparity vergence innervation.⁹⁵ In individuals with normal vision, this fixation disparity is too small to affect fusion or stereopsis. The fixation disparity needed to drive fine disparity vergence is a function of the demand on disparity vergence (DVD) and of the gain (G) of the fine-disparity vergence mechanism:

$$FD = DVD \times \left(1 - \left(\frac{G}{1+G}\right)\right)$$

Gain is a quantitative description of how efficiently the fine disparity vergence mechanism converts retinal disparity into vergence innervation. Larger gains are associated with smaller fixation disparities. Normal disparity vergence gain is usually greater than 100,⁹⁶ which accounts for the high precision of normal disparity vergence. For instance, an observer who has a gain of 125 and must generate 5° of sustained disparity vergence

innervation to maintain alignment (DVD = 5) would be expected to have 0.04° (1.36 min of arc) of fixation disparity. By comparison, normal reflex accommodation, which is much less precise than disparity vergence, is characterized by gains of less than 10.⁹⁶ If a patient has a large fixation disparity, it must be caused by a large disparity vergence demand, a low disparity vergence gain, or some combination. Patients with nonstrabismic vergence dysfunction often have both low disparity-vergance gain⁹⁷ and larger-than-normal disparity-vergence demands, thereby causing large fixation disparities. Although fine-disparity vergence gain is not directly measurable in a clinical environment, it is inversely proportional to fixation disparity, which is clinically measurable.

The fine-disparity vergence mechanism cannot keep targets on the horopter when vergence demands are high, despite its high gain. This is because the retinal disparity (i.e., fixation disparity) that stimulates fine-disparity vergence must rise proportionally with the demands placed on the disparity-vergence mechanism. The purpose of some of the other vergence innervations discussed later (i.e., accommodative vergence, tonic vergence, and vergence adaptation) is to supply a portion of the vergence innervation needed for sustained ocular alignment, thereby reducing disparity-vergence demand; this assistance is called *bias*. In this case, bias is a good thing, because it reduces fixation disparity, thereby enabling a high level of stereopsis for most viewing distances. In individuals with normal vision, disparity vergence and its innervational biases are so efficient that fixation disparity may be undetectable when the observer views fixation-disparity devices that incorporate binocular foveal fixation locks in the targets; such a target is the associated phoria target on the Borish vectographic near-point card II (Stereo Optical Company).

Coarse-disparity vergence is not controlled by continuous negative feedback. The full magnitude of coarse-disparity vergence innervation is calculated before the vergence movement begins, and it is not recalculated until the next vergence eye movement takes place.^{59,68,98} Consequently, coarse disparity vergence is only calculated by the visual system intermittently (i.e., whenever a large disparity-vergence step is demanded). Fine-disparity vergence maintains control of vergence innervation during the periods between coarse-disparity vergence episodes.

Reflex Accommodation

The reflex-accommodation mechanism (RA in Figure 5-11) is stimulated by blur, and the accommodation it generates serves to reduce blur. Because this is negative feedback behavior, the model of reflex accommodation looks similar to that of disparity vergence. Accommodative function is discussed in detail in Chapter 4.

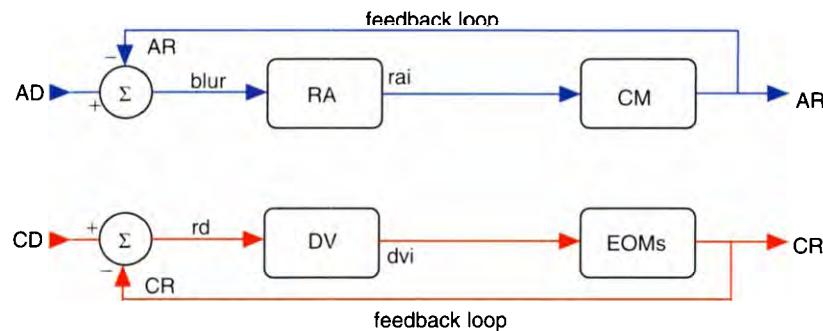


Figure 5-11

Box diagrams illustrating the similarity between the models of feedback control for reflex accommodation and disparity vergence. See the text for further explanation. AD, Accommodative demand; Σ , summation; RA, reflex accommodation mechanism; rai, reflex accommodation innervation; CM, ciliary muscle; AR, accommodative response; CD, convergence demand; rd, retinal disparity; DV, disparity vergence mechanism; dvi, disparity vergence innervation; EOMs, extraocular muscles; CR, convergence response.

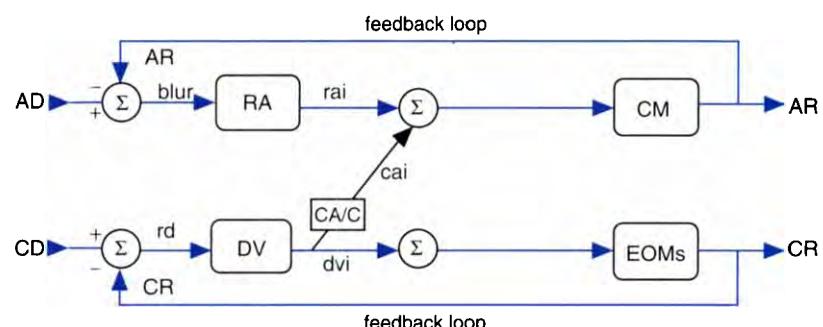


Figure 5-12

The interaction of disparity vergence innervation with accommodation is added to the diagrams shown in Figure 5-11. See the text for further explanation. AD, Accommodative demand; Σ , summation; RA, reflex accommodation mechanism; rai, reflex accommodation innervation; CM, ciliary muscle; AR, accommodative response; CD, convergence demand; rd, retinal disparity; DV, disparity vergence mechanism; dvi, disparity vergence innervation; EOMs, extraocular muscles; CR, convergence response; CA/C, convergence accommodation/convergence ratio; cai, convergence accommodation innervation.

Convergence Accommodation

The disparity-vergence mechanism generates not only disparity-vergence innervation but also synkinetic innervation called *convergence-accommodation innervation*. *Synkinesis* is an innervational mechanism whereby a single stimulus simultaneously generates multiple motor responses. Convergence accommodation helps to clear the fixation target as disparity vergence aligns the eyes to that target. Convergence accommodation serves as a proportional bias for reflex accommodation, which means that, as the accommodation and convergence demands increase, so does the assistance to reflex accommodation from disparity vergence. This action helps to minimize blur at all distances so that reflex accommodation can perform efficiently. Figure 5-12 represents the convergence accommodation synkinesis by a crosslink extending from the output of the disparity vergence mechanism to the reflex accommo-

dation pathway. The convergence accommodation innervation is summed with reflex accommodation innervation.

The relative strength of the convergence accommodation linkage is quantified by the CA/C ratio: the ratio of convergence accommodation (D) to convergence ($^{\Delta}$) stimulated by retinal disparity. The CA/C ratio is a proportional constant; therefore, convergence-accommodation innervation is the product of disparity vergence and the CA/C ratio. Because the CA/C ratio is constant, the greater the disparity vergence, the greater the convergence accommodation innervation. The CA/C ratio varies among individuals; in young adults, it averages 1 D/ 10^{Δ} at age 20 years.⁹⁹ For instance, if an observer with a 1/10 CA/C ratio exerts 5 $^{\Delta}$ of disparity vergence effort, he or she will generate 0.50 D of convergence accommodation innervation ($1/10 \times 5$). The CA/C ratio is highly correlated with the presbyopia process, which

declines linearly from the age of 20 years until presbyopia.^{77,99,100} At all ages, normal convergence accommodation generates less accommodative innervation than that needed for clarity. The balance of accommodative innervation comes from reflex accommodation and other accommodative biases.

For convergence accommodation to serve as an efficient proportional bias for reflex accommodation, the correct amount of convergence accommodation must be generated at each viewing distance. If the CA/C ratio were abnormally high or low, reflex accommodation would not receive appropriate convergence accommodation assistance from disparity vergence. The CA/C ratio may be abnormal in common nonstrabismic vergence anomalies (see Pathophysiology of Common Binocular Anomalies below and Chapter 21). Measurement of the CA/C ratio is discussed in Chapter 21.

Convergence accommodation is the main link between binocular vision and accommodation, and, therefore, with refraction. The effect of this link on refraction is reviewed under Binocularity and the Determination of Refractive State below.

Accommodative Vergence

Blur-driven reflex accommodation also produces synkinetic accommodative vergence innervation. The resulting accommodative vergence helps align the eyes as reflex accommodation works to clear blurred retinal images. Accommodative vergence, like convergence accommodation, is also a proportional bias. In this case, the bias serves to minimize disparity vergence usage at all viewing distances, thus restraining fixation disparity

and optimizing stereopsis. Figure 5-13 represents this synkinesis by a crosslink extending from the output of the reflex accommodation mechanism to the vergence system, where the accommodative vergence innervation is summed with disparity vergence innervation.

Accommodative vergence innervation is a product of reflex accommodation activity and the accommodative convergence/accommodation ratio (AC/A ratio). The AC/A ratio is a constant of proportionality, like the CA/C ratio. The AC/A ratio is not simply the inverse of the CA/C ratio but rather a separate neurological entity; its magnitude differs from the inverse of the CA/C ratio in most persons.¹⁰¹ The AC/A ratio describes the effect of accommodation on the vergence system. Higher AC/A ratios indicate a higher gain of the system and a corresponding larger effect of accommodation on the vergence system.

Studies of the AC/A ratio have used two methods to quantify it: (1) the stimulus AC/A ratio method and (2) the response AC/A ratio method. These are different than the aforementioned far-near and gradient AC/A ratios. In the response AC/A ratio method, measured convergence is divided by measured accommodation. In the stimulus method, measured convergence is divided by the accommodative stimulus value without regard for the actual accommodative response. In this case, the accommodative response is assumed to be equal to the accommodative stimulus. For the sake of expediency, the stimulus AC/A ratio is usually recorded in clinical settings, because it is easily measured and reasonably conveys most of the significant information required by the clinician. The response AC/A ratio can be deter-

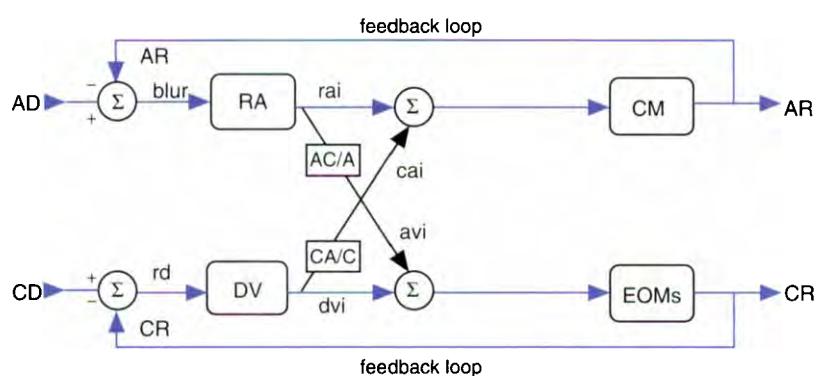


Figure 5-13

The interaction of reflex accommodation with convergence is added to the diagrams shown in Figure 5-12. The interconnection between accommodation and vergence is known as *dual interaction*. See the text for further explanation. AD, Accommodative demand; Σ, summation; RA, reflex accommodation mechanism; rai, reflex accommodation innervation; CM, ciliary muscle; AR, accommodative response; CD, convergence demand; rd, retinal disparity; DV, disparity vergence mechanism; dvi, disparity vergence innervation; EOMs, extraocular muscles; CR, convergence response; CA/C, convergence accommodation/convergence ratio; cai, convergence accommodation innervation; AC/A, accommodative convergence/accommodation ratio; avi, accommodative vergence innervation.

mined in a clinical setting, but it requires the measurement of accommodative response during AC/A ratio testing by a technique such as Nott retinoscopy (see Chapter 21). The response AC/A ratio is usually higher than the stimulus AC/A ratio, especially in adulthood.^{99,101} The difference between these two different methods of determining the AC/A ratios is due in part to the overestimation of accommodation in the stimulus AC/A ratio test because of depth of focus. The average stimulus AC/A ratio is approximately 3.5^{Δ} of convergence per diopter of accommodation ($3.5^{\Delta}/D$), but it varies greatly among normal persons, from $1^{\Delta}/D$ to $7^{\Delta}/D$.¹⁰² The response AC/A ratio changes modestly before age 40, and the stimulus AC/A ratio does not change at all.^{99,101,103} In addition to the age effect, small reductions of the response AC/A ratio have been observed after the correction of myopia.¹⁰⁴ Otherwise, the AC/A ratio is stable.¹⁰⁵

Determination of the AC/A ratio is an important aspect in the diagnosis of binocular motor anomalies and in the selection of appropriate therapy. The measurement of the AC/A ratio (discussed in detail in Chapter 21) requires the elimination of disparity vergence innervation so that the action of isolated accommodation vergence may be seen in the vergence response. Disparity vergence elimination is accomplished by blocking fusion by presenting a disparity that is too large to be fused (e.g., the von Graefe phoria method); by making the shapes of the ocular images unfusible (e.g., the Maddox rod test); or by complete occlusion of one eye (e.g., the cover test). Either of two methods can be used to alter the accommodative stimulus: the gradient method or the far/near method. The gradient method uses a lens-induced blur stimulus to accommodation, whereas the far/near method employs a change of target distance to create a blur stimulus. Because the far/near method uses a stimulus with a perceived distance that also changes (with one distance typically a proximal stimulus), it is not a pure AC/A ratio test, because it is contaminated by proximal effects. The patient's accommodative response to the far/near AC/A ratio stimulus is determined by a combination of blur and proximal cues. In emmetropes, the blur cue appears to dominate the accommodative response when both blur and proximal cues are available,^{106,107} so the AC/A ratio determined by the far/near method probably accurately represents the blur AC/A ratio. The proximal cue appears to control accommodative response in young myopes whose refractive error is progressing,¹⁰⁸ so the proximal cue probably dominates the AC/A ratio in progressing myopes when the far/near AC/A method is used. If the proximal cues do not stimulate accommodation and vergence in the same manner as the blur cue, the vergence/accommodation ratio determined by the far/near method would differ from the vergence/accommodation ratio measured by the

gradient method. Therefore, the gradient method is generally used to determine the AC/A ratio if that ratio will be used to prescribe spherical lenses for their effect on vergence (e.g., bifocals for convergence-excess heterophoria).

Dual Interaction

Reflex accommodation is directly responsible for clearing the ocular images, whereas disparity vergence is directly responsible for aligning the eyes. However, the two mechanisms assist each other by way of the accommodative vergence (AC/A) and convergence accommodation (CA/C) proportional biases. This mutual assistance of convergence by accommodation and accommodation by convergence is known as *dual interaction*. The dual interaction model appears to make the vergence and accommodative systems more efficient. Schor¹⁰⁹ suggested that a visual system with normal (i.e., average) AC/A and CA/C ratios would need to generate less sustained reflex accommodation and disparity vergence innervation to view a given target than would the same visual system with no interaction between accommodation and vergence. A second role for dual interaction may be to assist in the temporal coordination between accommodation and vergence. Schor also suggested that, if the AC/A and CA/C ratios deviate significantly from average, abnormally high innervational demands would be placed on both blur accommodation and disparity vergence. These high innervational demands likely would cause discomfort symptoms. Blur and diplopia would be perceived if image defocus and retinal disparity were large.

Tonic Vergence

The ciliary muscle and the extraocular muscles are innervated by tonic innervations. Tonic vergence innervation converges the eyes from their divergent anatomical position of rest—approximately 17^{Δ} horizontally in those with normal vision²⁵—to the tonic vergence resting state, which is approximately 3^{Δ} to 5^{Δ} convergent from straight ahead in those with normal vision.⁶³ The anatomical position of rest is the vergence angle the eyes would assume in the absence of any muscular innervation. The tonic vergence resting state is the vergence angle determined solely by tonic vergence innervation. Tonic vergence innervation can be viewed as a fixed innervational bias; therein lies its value to binocular vision. Because disparity vergence innervation is proportional to fixation disparity, disparity vergence could be overwhelmed if it were required to provide all of the innervation to converge the eyes. Without tonic innervation, disparity vergence would be required to muster the necessary convergence all the way from the anatomical position of rest to achieve and maintain the appropriate vergence for a given stimulus. To attain alignment using only disparity vergence would require a significant

magnitude of fixation disparity, even at remote viewing distances; stereopsis and binocular function would likely suffer. Because the visual system has no opportunity to support vergence angles more divergent than parallelism in a natural environment, the visual system can afford to establish a fixed amount of tonic innervation to bring the eyes approximately to parallelism without the use of disparity vergence. Therefore, little or no fixation disparity is experienced in normal far-point binocular vision because of tonic vergence.

Tonic vergence is incorporated into the systems model in Figure 5-14 as the box labeled "TV." TV is near the right end of the model, because there is no evidence that tonic vergence directly stimulates other innervational mechanisms. There is no input to TV, because tonic vergence innervation is not influenced by other mechanisms in the accommodation and vergence systems. Tonic accommodation maintains a steady 1 D level of accommodative innervation,¹⁰ and it is placed at a corresponding location in the accommodative system diagram, for similar reasons.

Vergence Adaptation

The oculomotor system has developed mechanisms to adjust to ongoing stimuli. These adjustments (or adaptations) reduce the stress on the system by allowing it to compensate for factors that initially cause stress. This adaptation reduces the overall requirements for ver-

gence similar to tonic innervation. Tonic innervation does not stem from visual stimuli to any significant extent. The adaptive processes function as a result of the response of the eyes to a visual stimulus. Adaptive vergence processes are important for adjusting to prisms that would otherwise overload the system and decrease its functionality.

The vergence adaptation mechanism generates a vergence innervation bias that serves to reduce fixation disparity by replacing disparity vergence innervation after the completion of a vergence response to a new demand. *Vergence adaptation is stimulated by accommodative vergence and disparity vergence motor innervations, not retinal stimuli.^{87,111}* Consequently, retinal disparity and blur stimulate vergence adaptation *only* if they activate disparity vergence innervation, reflex accommodation innervation, or both. For instance, a patient who accurately converges (fuses) through prisms for a sufficient period of time will adapt their vergence to that prism. If the same patient fails to fuse through the prism, vergence would not adapt to the prism, despite the continuous retinal disparity and diplopia experienced by the patient.

Although disparity vergence movement is often completed in less than a second, vergence adaptation usually requires minutes to fully adapt to a new magnitude of disparity vergence innervation. Moreover, once the adaptation mechanism has adapted vergence to a new

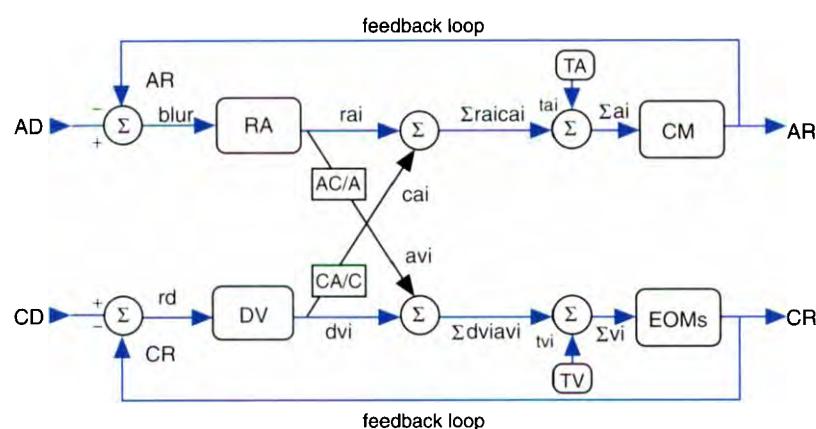


Figure 5-14

Tonic accommodation innervation (*tai*) and tonic vergence innervation (*tvi*) are added to the model shown in Figure 5-13. These innervations are summed with their respective fast stimulus-driven innervations. The sum of fast and tonic innervations yields the accommodative and vergent innervations that drive accommodation and vergence. See the text for further explanation. AD, Accommodative demand; Σ, summation; RA, reflex accommodation mechanism; *rai*, reflex accommodation innervation; CM, ciliary muscle; Σ_{raicai} , summed reflex and convergence accommodation innervations; TA, tonic accommodation mechanism; Σ_{ai} , summed accommodative innervations; AR, accommodative response; CD, convergence demand; *rd*, retinal disparity; DV, disparity vergence mechanism; *dvi*, disparity vergence innervation; Σ_{dviavi} , summed disparity and accommodative vergence innervations; TV, tonic vergence mechanism; Σ_{vi} , summed vergence innervations; EOMs, extraocular muscles; CR, convergence response; CA/C, convergence accommodation/convergence ratio; *cai*, convergence accommodation innervation; AC/A, accommodative convergence/accommodation ratio; *avi*, accommodative vergence innervation.

vergence demand, it learns to recognize that demand so that a subsequent reappearance of that demand can be met by a more rapid adaptation response.¹¹² This learning phase of the adaptation process can take much longer than the adaptation itself.

Vergence adaptation is not generated equally from fast vergence innervation arising from different sources, such as accommodative vergence, coarse-disparity vergence, and fine-disparity vergence.¹¹³ This behavior has clinical implications when one must decide whether a patient should receive a prism correction as opposed to a spherical lens correction for a binocular anomaly (e.g., esophoria). The patient's vergence adaptation mechanism may adapt very differently to the spherical lens than to the prism, even though the lens and prism might have equivalent short-term effects on vergence.

These behaviors justify the incorporation of vergence adaptation into the dual-interaction model of vergence and accommodation as shown in Figure 5-15. The box labeled "VA" represents vergence adaptation. An analogous construction is shown for accommodative adaptation (AA) because of physiological evidence that it serves a similar purpose in the accommodative system.⁸⁷ Vergence adaptation is stimulated by corollary inputs of accommodative vergence innervation (avi) and disparity vergence innervation (dvi). The innervation produced by the vergence adaptation mechanism (vai) is

then summed with the concurrent sum of the accommodative vergence and disparity vergence innervations (Σ dviavi) and with tonic vergence innervation (tvi). The sum of all vergence innervations (Σ vi) produces the vergence response that is visible to the examiner.

The vergence system modeled in Figure 5-15 does not generate excessive vergence innervation when vergence adaptation innervation is added to fast vergence innervation because of the negative feedback loop. Because the feedback property of disparity vergence is monitoring retinal disparity during vergence adaptation, any tendency for excess vergence innervation (Σ vi) is met immediately by a reduction of disparity vergence innervation. As a result, the sum of fast and adaptive innervations remains approximately constant while adaptation progresses. Because vergence adaptation is a slow process requiring several minutes for completion, it adds little to the initial vergence response to a near viewing distance. During the several minutes after a change to a near viewing distance, adaptive innervation accumulates and replaces the fast vergence innervation. Thereafter, adapted vergence innervation—as a percentage of total vergence innervation—may be larger than fast vergence innervation. When the eyes return to far vision, the sequence of events just described is reversed: the initial response to far vision is mediated by negative (divergent) disparity vergence innervation, which is

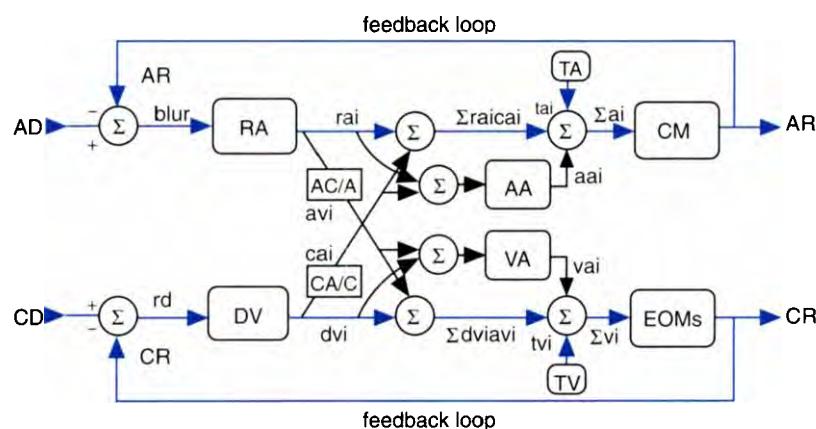


Figure 5-15

Vergence adaptation (VA) and accommodation adaptation (AA) mechanisms are added to the model shown in Figure 5-14. Accommodative adaptation innervation (aai) and vergence adaptation (vai) are stimulated by corollary discharges from both reflex accommodation and disparity vergence, and they are added to their respective fast and tonic accommodations. See the text for further explanation. AD, Accommodative demand; Σ , summation; RA, reflex accommodation mechanism; rai, reflex accommodation innervation; Σ raicai, summed reflex and convergence accommodation innervations; TA, tonic accommodation mechanism; tai, tonic accommodation innervation; Σ aai, summed accommodative innervations; CM, ciliary muscle; AR, accommodative response; CD, convergence demand; rd, retinal disparity; DV, disparity vergence mechanism; dvi, disparity vergence innervation; Σ dviavi, summed disparity and accommodative vergence innervations; TV, tonic vergence mechanism; tvi, tonic vergence innervation; Σ vi, summed vergence innervation; CA/C, convergence accommodation/convergence ratio; cai, convergence accommodation innervation; AC/A, accommodative convergence/accommodation ratio; avi, accommodative vergence innervation; EOMs, extraocular muscles; CR, convergence response.

later reduced as vergence adaptation adapts to far vision. The magnitude of vergence adaptation innervation in any given viewing situation depends on the dynamic behavior of the vergence system. Under rapidly changing viewing conditions, almost all changes of vergence are controlled by disparity vergence (assisted by accommodative and proximal vergence). Slowly changing or static vergence behaviors are likely dominated by adaptive vergence. This influence of dynamics on vergence adaptation also strongly influences the response of patients during phorometric tests; this is discussed under Accommodation and Vergence Physiology During Phorometry and in Chapter 21.

Accommodative adaptation interacts with reflex accommodation in much the same way that vergence adaptation interacts with disparity vergence.⁸⁷ When the eyes look from far to near, reflex accommodation initially clears the ocular images and is slowly replaced by positive adaptive accommodation innervation. Accordingly, accommodative vergence is reduced when accommodative adaptation replaces reflex accommodation. The restoration of far-point accommodation is initially controlled by sustained negative reflex accommodation, which must cancel residual positive adaptive accommodation innervation acquired from prior near vision. In time, negative reflex accommodation stimulates the adaptation of accommodation to a low level that is appropriate for far vision.

Patients with anomalous binocularly often have insufficient vergence adaptation.^{114,115} If vergence adaptation is totally absent, a patient may accept prisms prescribed to partially or completely compensate for the binocular stress and reduce the load successfully. However, some symptomatic patients have unidirectional defects of vergence adaptation that can null the potential benefit of a prism prescription. McCormack¹¹⁶ applied base-in (BI) prism to neutralize the exophoria of two symptomatic patients with simple convergence insufficiency. Their vergence systems adapted to the BI prism, thereby reinstating their high exophoria. North and Henson¹¹⁴ and Schor and Horner¹¹⁵ have shown that convergence adaptation is absent or inefficient in these patients. Prism prescription would fail in asymmetrical adaptation cases like these; therefore, prism as a treatment for binocular anomaly should be used with patients who do not adapt to it¹¹⁷ or with those who adapt only partially.

Tentative prism prescriptions are frequently checked to determine the extent of any adaptive response before the prisms are being prescribed in permanent form. Vergence adaptation stimulated by prism (i.e., prism adaptation) is assessed by comparing phorias between two measurements: (1) immediately after prism application (this is the "corrected" phoria measured through the tentative prism prescription) and (2) after the patient has worn the tentative prism prescription for a

minimum of 15 minutes (the "adapted" phoria measured through the prism prescription). If the prism prescription is to be successful, the adapted phoria should equal the corrected phoria.* A significant difference between the adapted and corrected phorias indicates that the patient has adapted his or her vergence to the prism, and the prism prescription will probably fail. Likewise, the systems model suggests that, if a patient was prescribed spherical lenses to reduce high phoria (e.g., minus lenses for exophoria), the lenses would only have a lasting effect on vergence if accommodation does not adapt to the lenses. The accommodative adaptation effect on vergence may be evaluated by comparing heterophoria immediately after the application of a tentative spherical lens prescription to the phoria after a minimum of 15 minutes of wearing the tentative prescription. A significant difference indicates accommodative adaptation and suggests that the lens prescription may fail to correct a vergence imbalance. Lens or prism additions intended to treat vergence should be considered for their potential effect on vergence and accommodation before being prescribed.

Proximal Vergence

Proximal vergence and proximal accommodation are triggered by any cue that elicits depth and distance perception. In other words, the stimulus to proximal innervation is perceived nearness. The proximal mechanism serves as a source of proportional bias innervation for both reflex accommodation and disparity vergence, helping to minimize blur and fixation disparity as targets move closer. Maddox's concept⁷⁶ of the oculomotor system primarily considered proximal vergence effects and largely ignored possible proximal accommodative effects.

Proximal vergence initiates large vergence step movements, as when looking from a remote distance to a reading distance,^{107,119} and it assists vergence when tracking objects moving smoothly in depth.^{120,121} The vergence initiation role of proximal vergence is an important component of vergence eye movements, because large shifts of viewing distance pose accommodation and vergence demands that are beyond the range of reflex accommodation and disparity vergence.^{106,107} Proximal vergence likely contributes only a few percentage points of the static near response when disparity and blur cues are available.^{106,107}

Proximal vergence, like disparity vergence, is a psycho-optical reflex. When an observer fixates a near

*Vergence adaptation does convert horizontal heterophoria to orthophoria. The small lateral physiological heterophorias measured in most individuals with normal vision are correlated with stable binocularly and minimum fixation disparity and, accordingly, are maintained by vergence adaptation.^{116,118} Vergence adaptation to vertical disparity tends to reinstate vertical orthophoria.

object, perceptual mechanisms automatically compute its distance and produce proportionate amounts of accommodation and vergence innervation. The reflexive nature of proximal vergence is evident in the phenomenon called *instrument convergence*.⁸¹ Instrument convergence is a tendency of the eyes to cross when looking into instruments such as biomicroscopes, stereoscopes, and other devices that optically simulate far binocular vision. The user's perception of the actual nearness of the viewed target causes excess convergence innervation; this can overwhelm information from disparity vergence, and it usually cannot be voluntarily overcome. When double vision ensues, the novice observer can do little to ameliorate the double vision other than to look away from the instrument. A reduced binocular visual field, which is common to many optical infinity devices, is probably a major contributor to instrument convergence. Kertesz¹²² notes that disparity vergence gain is sharply reduced when the diameter of the binocular fusion target is reduced below 10 degrees. The temporary application of a base-out (BO) prism can sometimes restore single vision to persons struggling with instrument convergence.

Proximity cues also generate iris sphincter innervation (miosis) concurrently with proximal vergence and proximal accommodation innervation. These three innervations compose a near triad synkinesis. The amount of proximal vergence that is associated with a unit of proximal accommodation is expressed as the proximal convergence/accommodation ratio ($^A/D$, PC/A ratio). The PC/A ratio must be measured in viewing conditions that do not stimulate reflex accommodation or disparity vergence, because these would otherwise dominate the accommodation and vergence responses. Measuring the PC/A ratio can be accomplished by occluding one eye, presenting a nonaccommodative target such as a broad DoG target to the fellow eye, and then measuring the change in vergence and accommodation that is induced by moving the DoG target closer to the observer. The average stimulus PC/A ratio (vergence response to target distance) is approximately $2.5^A/D$ when viewing static targets,¹²⁰ and it varies among individuals.

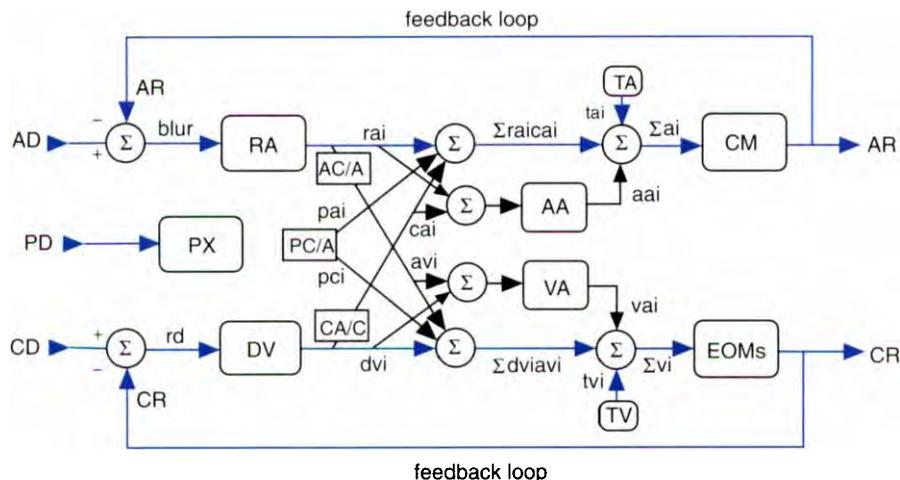
Current systems models suggest that proximal innervation arises independently of reflex accommodation innervation and disparity vergence innervation.^{86,106,107} Moreover, proximal innervation does not appear to alter accommodative adaptation.¹²³ These findings suggest that proximal innervation should be represented in the systems model as shown in Figure 5-16. The independence of proximal innervation from other sources of accommodation and vergence innervation indicates that the PC/A ratio is likely to differ from the AC/A and CA/C ratios in many persons. This underscores the importance of carefully considering the type of stimulus to be used for testing the AC/A ratio, as discussed earlier.

Voluntary Vergence

Voluntary innervation is another near triad synkinesis: it generates accommodation (voluntary accommodation), convergence (voluntary vergence), and pupil constriction.¹²⁴ As its name implies, the voluntary synkinesis generates accommodation and convergence through conscious attention to the acts of accommodation and vergence. Voluntary effort differs from proximal innervation in at least two respects. First, voluntary control requires conscious attention not only to select a target but also to maintain the accommodation and vergence innervation for that target. In this respect, voluntary accommodation and vergence are like skeletal muscle control. Second, voluntary innervation results from the intent to move the eyes, whether or not there is a sensory stimulus. Accordingly, patients may produce voluntary vergence for any reason they desire, which explains how trained persons can visibly cross their eyes in the absence of a fixation target. The purpose of voluntary innervation may be to supplement the psycho-optic reflex innervations when their neural controllers become fatigued.

When voluntary vergence is called into play, the ratio of voluntary vergence to voluntary accommodation is highly correlated with the AC/A ratio.^{125,126} Although this seems to suggest that voluntary vergence might be a form of accommodative vergence, voluntary vergence does not behave like accommodative vergence in all respects. Voluntary vergence is associated with pupil constriction,¹²⁴ whereas accommodative vergence activated by blur alone does *not* cause pupil constriction.¹²⁷ Moreover, voluntary vergence innervation achieved in darkness or in the light with diplopia has no significant impact on vergence adaptation.¹²⁴ Accommodative vergence associated with reflex accommodation activates vergence adaptation.⁸⁷ The lack of vergence adaptation by voluntary vergence activity has a parallel in conjugate eye movement. Deubel¹²⁸ observed that saccadic gain adaptation for purely voluntary saccades is independent of saccadic gain adaptation for reflexively elicited saccades. The inability of voluntary vergence to stimulate vergence adaptation prevents voluntary vergence from generating strong vergence adaptation aftereffects when voluntary effort is relaxed.

Voluntary innervation probably contributes to many optometric findings. Instructions typically given to patients encourage them to maintain clear and single vision and to concentrate their attention on the motor acts of accommodation and vergence. Voluntary vergence is useful during the early stages of visual training, because it allows the patient to control ocular alignment even when disparity vergence gain is low. Voluntary innervation is inappropriate for prolonged control of accommodation and vergence, because the act of attending to motor behavior draws attention away from visual information processing. Moreover, when voluntary

**Figure 5-16**

Proximal innervation is added to the model shown in Figure 5-15. The apparent nearness of a visual target, called proximal demand (*PD*), generates proximal motor innervation by way of the proximal mechanism (*PX*). That innervation is sent to the accommodation and vergence systems in accordance with the value of the proximal convergence/accommodation ratio (*PC/A*), and it is summed with the other fast accommodation and vergence innervations. See the text for further explanation. *AD*, Accommodative demand; Σ , summation; *RA*, reflex accommodation mechanism; *rai*, reflex accommodation innervation; Σ_{raicai} , summed reflex and convergence accommodation innervations; *TA*, tonic accommodation mechanism; *tai*, tonic accommodation innervation; *Sai*, summed accommodative innervation; *CM*, ciliary muscle; *AR*, accommodative response; *CD*, convergence demand; *rd*, retinal disparity; *DV*, disparity vergence mechanism; *dvi*, disparity vergence innervation; Σ_{dviavi} , summed disparity and accommodative vergence innervations; *TV*, tonic vergence mechanism; *tvi*, tonic vergence innervation; Σ_{vi} , summed vergence innervation; *EOMs*, extraocular muscles; *CR*, convergence response; *CA/C*, convergence accommodation/convergence ratio; *cai*, convergence accommodation innervation; *AC/A*, accommodative convergence/accommodation ratio; *avi*, accommodative convergence innervation; *pai*, proximal accommodation innervation; *pci*, proximal convergence innervation.

vergence dominates near vision, there is no vergence adaptation to support vergence stability and accuracy. Thus, patients habitually resorting to voluntary innervation to gain clarity and singleness (e.g., convergence-insufficient patients) can be expected to experience reading problems, discomfort symptoms, or both.⁸⁸

The Near Response of Accommodation and Vergence

When accommodation and vergence are switched from far vision to near vision, most of the elements described earlier are activated. The sequence of innervational events may be followed in Figure 5-16. Because tonic vergence and tonic accommodation innervations by themselves would place vergence and accommodation at an intermediate distance of gaze, far-point vision is maintained by the use of small amounts of negative reflex accommodation and negative disparity vergence innervation that cancel the tonic innervations. The response to a near target (e.g., a 40-cm near-point test card) is initiated by proximal innervation, which brings the near target within range of coarse-disparity vergence. Coarse-disparity vergence then adds additional tran-

sient innervations that bring the near target within range of positive reflex accommodation and positive fine-disparity vergence, which complete and sustain the oculomotor responses. All of this activity, including dual interaction between disparity vergence and reflex accommodation, is normally completed within 1 second. The transition to near vision drives reflex accommodation and fine-disparity vergence innervations from their small negative magnitudes at a far point to the modest positive magnitudes required to hold accommodation and vergence at 40 cm. The demands on reflex accommodation and fine disparity vergence at 40 cm will be less than the nominal 40-cm stimulus values of 2.5 D and 15°, because tonic accommodation and tonic vergence provide some of the positive motor innervation needed for near vision. Beginning with the onset of the near response and continuing for the next several minutes, the vergence adaptation and accommodative adaptation mechanisms gradually build their innervational responses to replace some of the positive reflex accommodation and sustained disparity vergence innervations that initially provide clear and single vision at 40 cm.

Regaining far vision after a period of sustained near fixation initially demands the generation of negative accommodation and vergence innervations to overcome the positive adaptive innervations accumulated at a near point. This process begins with transient negative proximal vergence followed by negative coarse-disparity vergence, negative sustained-disparity vergence, and negative reflex accommodation. After several minutes of far fixation, these negative innervations stimulate the adaptation of accommodation and vergence to the lower values of adaptive innervation typical of far vision, thereby reducing the need for large magnitudes of negative sustained disparity vergence and negative reflex accommodation innervation.

Vertical Vergence Eye Movements

The vertical vergence system is composed of three basic components: (1) disparity vergence, (2) tonic vergence, and (3) vergence adaptation. Vertical disparity vergence is stimulated by the presence of global vertical retinal disparity (a constant disparity extending over the binocular visual field), and it is able to elevate (supraduction) or depress (infraduction) one eye relative to the other by a few prism diopters. The vertical vergence range is an order of magnitude smaller than the range of horizontal vergence (i.e., instead of a 20^{Δ} or so to break in horizontal vergences, the vertical vergence break finding is often $3\text{--}5^{\Delta}$). Large vertical disparity vergence amplitudes are not required in individuals with normal vision, because the switching of gaze between far and near vision does not alter global vertical retinal disparity. Vertical disparity vergence is controlled by negative feedback, so vertical fixation disparity is expected when vertical disparity vergence is active.

Vertical vergence exhibits adaptive behavior when challenged with a prolonged and fusible vertical vergence demand.^{129,130,131} Vertical vergence adaptation, like its horizontal counterpart, is probably stimulated by vertical disparity vergence innervation, and it sums with vertical disparity vergence innervation to produce the vertical vergence response. Vertical vergence adaptation attempts to reinstate the habitual vertical phoria, which is usually—but not always—zero. Vertical heterophoria is often accompanied by defective vertical vergence adaptation. Prismatic prescriptions for vertical heterophoria are usually worn successfully by the patient who does not adapt to vertical prism.^{130,132} Testing for vertical prism adaptation should be performed before the prescription of vertical prism to identify those vertical heterophores who will fully adapt to vertical prism. The method for testing vertical vergence adaptation is much the same as for horizontal, but it should be extended to 30 minutes of prism-wearing time, because vertical prism adaptation is slower than horizontal prism adaptation.¹³⁰ Patients who do not adapt signifi-

cantly within 30 minutes will usually wear vertical prism successfully.¹³⁰

Vertical vergence has no known direct neurological link with horizontal vergence or accommodation and therefore does not significantly influence the determination of refractive error. However, a sufficiently large global vertical disparity reduces stereopsis.¹³³ Also, horizontal vergence may be impaired by the presence of a large global vertical disparity.¹³⁴ These effects probably result from a disturbance in the processing of horizontal retinal disparity when the retinal images are vertically misaligned. Of course, a sufficiently large vertical disparity between the images results in a complete loss of fusion. The interaction of vertical imbalance with horizontal vergence indicates that the clinical analysis of and prescription for horizontal vergence require the appropriate consideration of vertical vergence.

PATHOPHYSIOLOGY OF COMMON BINOCULAR ANOMALIES

Systems analysis has generated a clearer understanding of the causes of convergence insufficiency and convergence excess heterophoria. These anomalies are discussed in detail, because they are the most common binocular anomalies encountered in clinical practice. Figure 5-16 may be helpful for following the discussion of these anomalies. The complete pathophysiology of many other nonstrabismic anomalies, such as fusional insufficiency and basic esophoria, is poorly understood.

Simple Convergence Insufficiency

Convergence insufficiency is generally regarded as a syndrome that includes an exodeviation of the eyes at the nearpoint, relatively little or no deviation of the eyes with distance fixation (generally a small exophoria), a relative deficit of positive relative convergence, and a receded nearpoint of convergence.¹³⁴ The syndrome produces a variety of symptoms such as ocular fatigue, headaches, and asthenopia.¹²

North and Henson¹¹⁴ observed that patients exhibiting the clinical characteristics of simple convergence insufficiency (high near exophoria, low AC/A ratio, low positive relative convergence, and excess accommodation in near vision) had poor or nonexistent convergent adaptation. Schor and Horner¹¹⁵ confirmed North and Henson's observations, and they added the observation that simple convergence-insufficient patients had unusually rapid adaptation of accommodation as well. Schor and Horner proposed that this adaptation asymmetry might be a major cause of simple convergence insufficiency. The logic behind their proposal is as follows. Rapid accommodative adaptation would replace reflex accommodation nearly as fast as the latter

is generated, thereby reducing accommodative vergence to abnormally low levels. This would reduce overall convergence; in addition, it partially explains the large near exophoria observed in these patients, and it is responsible for the low AC/A ratios. The loss of proportional vergence bias from accommodative vergence is compounded by highly inefficient vergence adaptation, which fails to adapt to disparity vergence and accommodative vergence innervations. Therefore, inefficient vergence adaptation also contributes to these patients' near exophoria and low measured AC/A ratios.

Frequently, these patients have a rather low lag of accommodation in near vision. This near accommodative excess associated with simple convergence insufficiency may be explained by abnormally high convergence accommodation activity. The high convergence accommodation activity is the result of high disparity vergence activity necessitated by insufficient accommodative vergence and vergence adaptation. Negative reflex accommodation offsets the excess convergence accommodation within its capacity to do so, but often negative reflex accommodation is overwhelmed by excess convergence accommodation, thereby leading to manifest overaccommodation in near vision.

The adaptational anomalies are probably the causes of simple convergence insufficiency, but they are not likely these patients' only physiological problems. Such patients also usually have low gains in both reflex accommodation¹³⁶ and disparity vergence.⁹⁷ Moreover, some of these patients seem unable to use voluntary innervation to assist the weak reflex controllers.⁸⁸

The Schor-and-Horner hypothesis for simple convergence insufficiency explains the success of vision training for these cases. The magnitude and rate of convergence adaptation can be increased significantly by vision training.^{137,138} Improved positive vergence adaptation reduces the demand on disparity vergence and concomitantly reduces excess convergence accommodation. Moreover, accommodative facility training increases the gain of reflex accommodation,¹³⁶ and vergence facility training increases the gain of disparity vergence.⁹⁷

Convergence Excess

The chief clinical characteristics of convergence excess heterophoria are moderate to high esophoria in near vision, a high AC/A ratio, and a significant lag of accommodation in near vision.¹² Convergence excess heterophoria is caused by excessive sustained accommodative vergence innervation. Schor and Horner¹¹⁵ observed that the excessive accommodative vergence innervation is associated with insufficient adaptation of tonic accommodation and unusually rapid adaptation of tonic vergence. They proposed that these tonic adaptation asymmetries are major factors in the pathophysiology

of convergence excess. The logic for their proposal is as follows. The accommodative adaptation mechanism does not adapt to near vision, so reflex accommodation must carry a larger percentage of the near sustained accommodative load. The higher-than-normal reflex accommodation effort causes excess accommodative vergence by way of the AC/A synkinesis. The effect of the excess accommodative vergence innervation on binocular alignment is compounded by the unusually strong and rapid adaptation of tonic vergence to near vision. The excessive accommodative vergence and vergence adaptation responses are offset by *negative* disparity vergence innervation to maintain ocular alignment. By contrast, individuals with normal vision use positive disparity vergence in near vision. The negative disparity vergence innervation causes negative convergence accommodation innervation by way of the CA/C synkinesis, which increases the lag of accommodation already made large by insufficient accommodative adaptation. This increases the demand for positive accommodative innervation from the struggling reflex accommodation mechanism. The additional reflex accommodation response to the effects of negative convergence accommodation then triggers the generation of additional accommodative vergence through the AC/A synkinesis, thereby further destabilizing the vergence system. The ability of convergence-excess patients to maintain clear and single binocular vision ultimately depends on the gains of the reflex controllers and the degree of abnormality of the adaptive mechanisms.

The pathophysiology of convergence excess suggests that visual training aimed at boosting accommodative adaptation should lower accommodative vergence activity and therefore solve the problem. Studies of the trainability of accommodative adaptation have not been performed. However, it is well established that training-induced changes of the AC/A ratio are small and transient.^{139,140} Because the AC/A ratio is determined in part by accommodative adaptation, the lack of success in significantly changing the AC/A ratio by visual training suggests that accommodative adaptation may not be alterable by visual training. Convergence-excess heterophoria is most successfully managed by plus lenses for near vision, which reduce reflex accommodation and therefore excess accommodative vergence.¹⁴¹

BINOCULARITY AND OPHTHALMIC TESTS

The Effect of Blur on Binocular Vision

Stereoscopic acuity is degraded by reduced contrast and image blur, and unilateral blur is more devastating on stereoscopic acuity than bilateral blur.^{142,143} The physiological basis of the additional stereoscopic loss caused

by unilateral blur is probably foveal suppression of the blurred image.^{61,144} Blur-induced suppression apparently reduces contrast sensitivity in the suppressed eye beyond the loss due directly to blur, and contrast is correlated with stereoscopic acuity.¹⁴⁵ Suppression behavior makes it clear that a balanced refractive correction is necessary for good stereopsis. Intentional differences of refraction left in place by the practitioner (e.g., monovision contact lens prescriptions for presbyopes) exact a penalty of poor fine stereopsis.⁶¹

Dwyer and Wick¹⁴⁶ observed that the correction of even modest refractive errors can improve the binocularly of patients who have both uncorrected refractive error and nonstrabismic binocular anomaly. This observation correlates with Simpson's¹⁴⁴ finding that anisometropias greater than 0.50 D initiate blur-induced suppression. Most of the improvements observed by Dwyer and Wick were apparent within 1 to 2 years after the dispensing of the refractive corrections. This result is significant for showing that longstanding uncorrected refractive error can have deleterious effects on binocularly for many months after the refractive errors are corrected. The authors speculate that longstanding blur impairs the neurology of disparity vergence control mechanisms and that the recovery of this neurology takes time.

Blur-induced unilateral foveal suppression can be used to advantage in patients with refraction. By modestly blurring one eye, one can prevent that eye from contributing to high-spatial-frequency vision, even though fusion and oculomotor alignment are maintained by middle- and low-spatial-frequency vision. Hence, the visual acuity and refined refractive status of the unblurred eye can be assessed in isolation, even though both eyes are open and aligned to the refraction target; this is the basis of the Humphriss fog refraction technique (see Chapter 20). The benefits of refracting the patient while he or she is binocular are discussed later under Binocularity and the Determination of Refractive State.

Spectacle Corrections and Binocular Vision

Although clear ocular images are necessary for good stereopsis and vergence eye movements, the provision of clear ocular images with spectacle lenses may create other visual effects that distort the horopter and stereoscopic perception, unbalance binocular motility, and perturb visual comfort. Two types of spectacle lens prescription probably account for most spectacle-induced binocular vision problems: (1) prismatic and (2) anisotropic. Prismatic corrections may cause prismatic distortion of stereopsis, whereas anisotropic corrections may cause anisophoria and aniseikonia. These optical effects pose challenges to binocular motor and binocular sensory function.

Prismatic Distortion of Stereopsis

Ophthalmic prisms are commonly prescribed BI or BO to assist the oculomotor system. When prescribed prisms induce distortions of the retinal images, they cause a curvature distortion of stereoscopic visual space.³³ For instance, the distortion induced by BO wedge prisms cause an objectively flat wall to appear concave toward the observer. The accompanying prism distortion of stereopsis is the result of this asymmetrical image magnification along the base-to-apex line of prisms. The distortion can easily be appreciated by viewing a regular geometric target (e.g., a brick wall) through a high-power wedge prism held away from the eye. This distortion is minimal in meniscus-design ophthalmic lenses with front curves of approximately +8.00 D (e.g., in moderate hyperopia corrections), but it is relatively strong in lenses with nearly flat front curves (e.g., in high myopia corrections).³³ Prism distortion is one reason that the power of prism prescriptions should be kept to the minimum value required for a good oculomotor effect. This is achieved by splitting any prism between the eyes when prisms are prescribed.

Anisophoria

Anisophoria is a significant change of heterophoria associated with a change of gaze direction, and it is commonly caused by the differential prismatic effects of anisotropic spectacles. Prismatic effects caused by plus lenses increase the ocular rotation required to fixate the limits of an object, whereas minus lenses decrease the required ocular rotation. When this occurs, the anisometrope's right and left eyes must rotate differently when looking left, right, up, and down. This differential rotational stimulus (or prismatic effect) is proportional to the eccentricity of gaze from the optical axes of the lenses, so increasing retinal disparities are encountered by the patient with increasingly eccentric gaze through the anisotropic spectacle prescription. These retinal disparities become disparity vergence eye movement stimuli, because they require different angular rotations of the eyes to bifixate targets in different directions of gaze. Therefore, patients wearing new anisotropic spectacles must use a combination of versional and disparity vergence oculomotor innervation to align the eyes to targets viewed off the optical axes. For instance, an anisometrope with a hyperopic right eye and myopic left eye must converge in left gaze and diverge in right gaze. Most persons with normal vision adapt vertical saccades,¹⁴⁷ smooth pursuits,¹⁴⁸ the VOR,¹⁴⁹ and adaptive vergence¹⁵⁰⁻¹⁵² to the differential prismatic demands of anisotropic spectacles so that accurate fixation takes place in any direction of gaze with little or no use of disparity vergence. This adaptive process is called *gaze-specific adaptation*. Because some forms of gaze-specific adaptation create nonconjugate ocular rotation

by way of innervational mechanisms that are normally conjugate (e.g., saccades), gaze-specific adaptation violates Hering's law of equal innervation.

Although gaze-specific adaptation of versional eye movement innervation is not easily observed in a clinical setting, the vergence aspect can be observed clinically. Sethi and Henson¹⁵³ and Schor and associates¹⁵¹ proposed that gaze-specific adaptation of vergence is accomplished by the creation of vergence adaptation innervation that varies with the field of gaze. Therefore, one can monitor gaze-specific adaptation by testing phorias in different fields of gaze, as in the prism adaptation test. When a patient completes gaze-specific adaptation, the oculomotor system will have created a heterophoria that is invariant with field of gaze when the phoria is measured through the patient's anisometropic spectacle prescription. Henson and Dharamshi¹⁵⁰ observed that gaze-specific adaptation is most rapid in those fields of gaze in which more visual activity occurs. Their observation suggests that gaze-specific adaptation is probably responsible for the V pattern of normal horizontal heterophoria wherein lateral phorias are more "eso" in down gaze than in the primary position of gaze. In other words, down-gaze "eso" is probably created by gaze-specific adaptation stimulated by the frequent use of the eyes for close viewing when in down gaze.

Anisophoria occurs when gaze-specific adaptation is not able to neutralize disjunctive eye-movement demand. Patients whose oculomotor systems cannot achieve gaze-specific adaptation in response to new anisometropic corrections have noncomitant heterophoria. Symptomatic vertical noncomitant phoria is relatively common in the lower field of gaze of wearers of new anisometropic spectacles. If gaze-specific adaptation is unable to neutralize a vertical imbalance, vertical phoria in down gaze will be evident. The magnitude of the heterophoria is predictable from the anisometropia and the eccentricity of down gaze.¹⁵⁴ Chapter 31 discusses a method for testing gaze-specific adaptation in down gaze. Patients with insufficient gaze-specific adaptation may require a slab-off prism to wear anisometropic spectacles comfortably.¹⁵⁴

Aniseikonia

Aniseikonia is a difference in the size of the ocular images, and it commonly occurs with visual distortion, headaches, and discomfort. *Iseikonia* is the condition of equal ocular images. Ocular image size is the size of an image as *perceived* by the observer. (Retinal image size is the geometrical size of the image on the retina and does not involve perception, only optics.) Ocular image size is determined by the number of local signs spanned by a retinal image. Therefore, the ocular images are perceived as equal if the retinal images cover the same number of local signs in each eye, irrespective of what

the physical size of the eye might be or what the sizes of the retinal images might be.

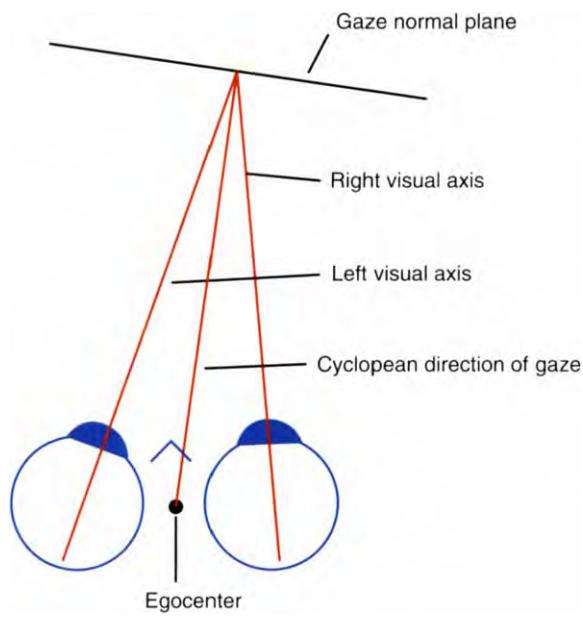
Anisometropic spectacles may cause aniseikonia. For instance, the more myopic eye of most anisometropic patients is larger than the fellow eye¹⁵⁵; this condition is called *axial anisometropia*. Regardless, these patients have equal ocular image sizes (*iseikonia*) when their vision has been corrected with contact lenses.^{156*} Iseikonia persists because the retina of the relatively larger myopic eye usually stretches during ocular growth to match its larger optical image.¹⁵⁷ As a result, a given target activates the same number of local signs in each eye. Axial anisometropes usually have aniseikonia when their refractive error is corrected by spectacle lenses.

For those anisometropes who have aniseikonia, the aniseikonia is associated with retinal disparities that increase with visual field eccentricity. The variation in disparity is not reduced globally by vergence eye movement. If the rate of increase of these disparities exceeds the rate of increase of Panum's areas with eccentricity, peripheral diplopia and rivalry occur. The magnitude of aniseikonia that exceeds the allowable increase of peripheral retinal disparity is approximately 7%.¹⁵⁸ Because binocularly in the presence of large aniseikonia is unstable, vergence alignment is usually lost, and foveal diplopia ensues.

Aniseikonia caused by spectacle lenses can be classified as overall or meridional. Overall aniseikonia is a uniform enlargement of one eye's ocular image relative to the other, and it is typically caused by spherical anisometropia. Meridional aniseikonia is an enlargement of one eye's ocular image (as compared with the other) in a specific meridian, and it is typically caused by astigmatic anisometropia. These forms of aniseikonia may occur in combination, and they may have differing effects on stereoscopic space perception. The pathophysiology of the aniseikonic distortion of stereopsis, which is described later, explains the visual experience of the aniseikonic patient, and it is the basis of the testing of aniseikonia by the Space Eikonometer (discussed in Chapter 32).

The stereoscopic consequences of aniseikonic retinal disparities can be appreciated by analyzing object planes. An *object plane* is a flat surface that is pierced at its center by the cyclopean direction of gaze. A particularly important object plane—the gaze-normal plane—is perpendicular to the cyclopean direction of gaze (Figure 5-17). The perceived orientation of an object plane is influenced by the combined effects of horizontal and vertical disparities subtended by textures on the surface of the plane (see The Binocular Contribution to

*Because contact lens corrections are close to the principal planes of the eyes, they usually have little effect on retinal image sizes (see Chapter 26).



The gaze-normal plane—an object plane that is perpendicular to the cyclopean direction of gaze—is useful for analyzing stereoscopic distortion. The gaze-normal plane is perceived to be perpendicular to the direction of gaze by observers with normal vision.

Depth and Distance Perception). Aniseikonic ocular images contain distributions of retinal disparities that are similar to those encountered by individuals with normal vision viewing object planes under different viewing circumstances.

Ogle³³ analyzed aniseikonic stereopsis by subdividing it into three subcomponents: (1) the geometric effect, (2) the induced effect, and (3) the oblique effect. The geometric effect is a stereoscopic distortion caused by horizontal aniseikonia. The distortion can be explained by a geometric drawing of optical magnification in the visual plane (Figure 5-18). Points P and Q define the lateral limits of a gaze-normal plane that is fixated at F. An observer's right-eye ocular image is magnified by a $\times 90$ meridional magnifier,* thereby creating horizontal aniseikonia. The short-dash lines radiating from the right eye represent the right-eye optical ray paths and apparent visual directions of object points P, F, and Q *prior* to the application of the magnifying lens. P, F, and Q are imaged at locations p, f, and q on the right retina. The solid lines radiating from the right eye

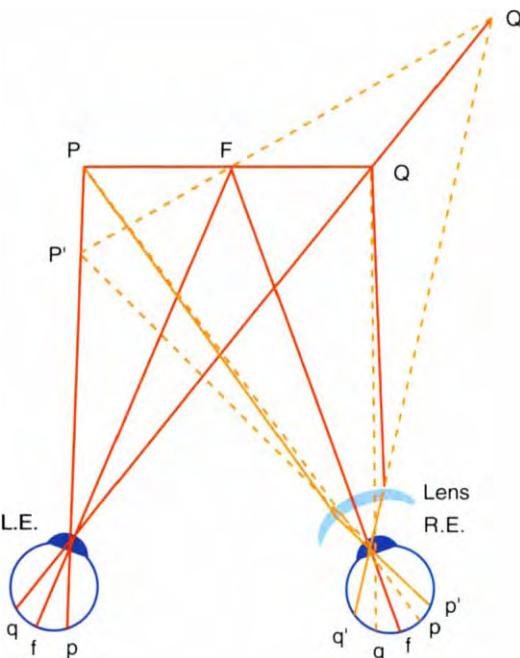


Figure 5-18

Visual plane drawing of the geometric effect distortion experienced by observers whose right ocular image is horizontally larger than their left ocular image. The gaze-normal plane is perceptually skewed, farther on the right and closer on the left. See the text for a detailed explanation. Object space points P, F, and Q are imaged on retinal locations p, f, and q in each eye before a lens is placed in front of the right eye. The points are imaged at retinal locations p', f', and q' of the right eye after application of the lens. P, F, and Q are then perceived to be at P', F, and Q'. L.E., Left eye; R.E., right eye. (From Ogle KN. 1950. Researches in Binocular Vision, p 143. New York: Hafner. By permission of Mayo Foundation.)

represent the magnified optical ray paths from points P, F, and Q in object space to retinal points p', f', and q' on the right retina *after* magnifier application. The long-dash lines drawn to the right eye represent the *apparent* directions of P and Q arising from retinal image points p' and q' in the right eye *after* magnifier application. The solid lines radiating from the left eye represent the optical ray paths from points P, F, and Q in object space to points p, f, and q on the left retina, as well as the *apparent directions* of points P, F, and Q. Retinal points p and p' in the left and right eyes, respectively, give rise to a crossed retinal disparity, thereby causing object point P to appear closer than F at location P'. Likewise, points q and q' in the left and right eyes, respectively, give rise to an uncrossed disparity, thus causing object point Q to appear farther than F at location Q'. Hence, the gaze-normal plane appears to be rotated about a vertical axis, closer on the left and farther on the right. A similar construction confirms that a $\times 90$ magnifier placed before

*A magnifier lens, like a Galilean telescope, magnifies an optical image without altering its focus. Magnifier lenses are also known as size lenses. However, ophthalmic magnifier lenses typically magnify the retinal images by only a few percentage points. A meridional magnifier has its maximum magnification effect perpendicular to its axis meridian and no magnification in its axis meridian.

the left eye instead of the right eye causes the opposite stereoscopic distortion.

This geometric effect produces ocular images similar to those experienced by an observer with normal vision who views, during symmetrical convergence, an objectively rotated object plane that is closer on the left and farther on the right. The horizontal aniseikonic disparities would be indistinguishable in these two situations, and the average vertical disparities would be zero in both cases. The normal view differs from the aniseikonic view in that the normal observer experiences a gradient of vertical disparities across the horizontal dimension of the rotated object plane, with the vertical disparities being larger at the near end of the plane and smaller at the far end of the plane. The horizontally aniseikonic observer does not experience the vertical disparity gradient. Vertical disparity gradients—or the lack of them—become perceptually significant when very large binocular targets are viewed in near vision.³⁷

The *induced effect* is a stereoscopic distortion caused by vertical disparities associated with vertical aniseikonia. When a $\times 180$ meridional magnifier is placed before the right eye during symmetrical convergence, the gaze-normal plane appears to be rotated about a vertical axis, with its right side closer than its left side. Hence, the induced effect is qualitatively the *opposite* of the geometric effect. The induced effect creates approximately the same amount of slant as the geometric effect for small and moderate magnitudes of aniseikonia (<4%), but it generates relatively less slant than the geometric effect for larger aniseikonias.³³ When a vertically larger right eye image causes the induced effect, the ocular images are similar to those experienced by an observer with normal vision viewing an object plane in right gaze that is farther from the cyclopean eye on its left side and closer on its right side, paralleling the Vieth-Müller circle and the longitudinal horopter (Figure 5-19). The associated ocular images are horizontally equal, but the right-eye image is taller. The induced effect images differ from those caused by a slanted object plane in eccentric gaze, because the latter presents a gradient of vertical disparities to the observer in addition to the average vertical size difference.

The ocular image of one eye is uniformly larger than that of the other eye in overall aniseikonia. Small or moderate overall aniseikonia does not distort the apparent gaze-normal plane, because the opposing geometric and induced effects arising from horizontal and vertical aniseikonia, respectively, cancel each other out. Larger overall aniseikonia generates a geometric effect distortion, because the induced effect fails to respond proportionally to larger magnifications. Geometric effects associated with large overall aniseikonia may not appear practically significant, because such large aniseikonia usually causes a failure of stereopsis and sometimes of fusion. Individuals with normal vision experience

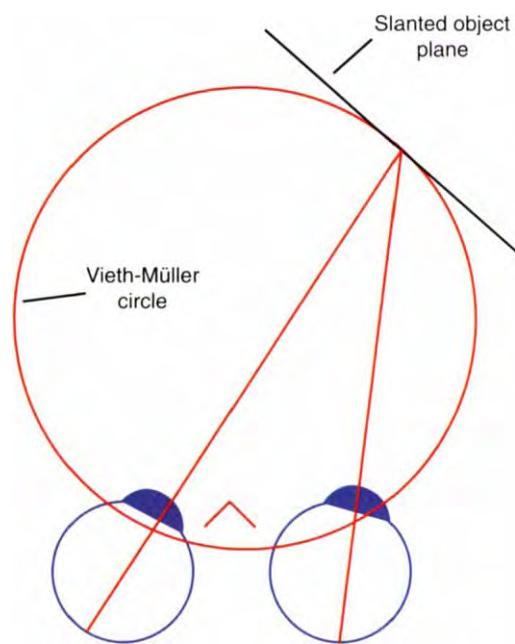


Figure 5-19

When an observer with normal vision views, in right eccentric gaze, an object plane that is tangent to the Vieth-Müller circle, the retinal images are horizontally equal, but the right-eye image is taller than the left-eye image. The observer with normal vision correctly judges this object plane as closer on the right and farther on the left. These retinal images are similar to those seen by an observer with normal vision who is viewing a gaze-normal plane in symmetrical convergence through a $\times 180$ meridional magnifier placed before the right eye or by an aniseikonic observer with a right vertical aniseikonia. See the text for details.

overall image size differences when viewing a gaze-normal plane in eccentric gaze.

The oblique effect stereoscopic distortion is observed when the axis of meridional aniseikonia is in an oblique meridian. Figure 5-20 shows a simulation of the left and right retinal images when magnified in the 45- and 135-degree meridians, respectively, by oblique-axis aniseikonia. When the targets in Figure 5-20 are fused (see the legend of Figure 5-1 for the method), the gaze-normal plane appears to be slanted away from the observer at the top of the plane and closer to the observer at the bottom of the plane, as shown in Figure 5-21. The oblique stretching of the retinal images tilts the horizontal and vertical meridians of the images toward the elongated meridians of the images. The differential rotation of the vertical meridians, called *declination** can explain the

*Cylinder lenses cause meridional magnification (as well as blur). Declination can be observed by viewing a textured surface through a minus cylinder lens held away from the eye. Rotating the cylinder lens about its optical axis declines the vertical and horizontal meridians of the surface toward the axis of the cylinder lens.

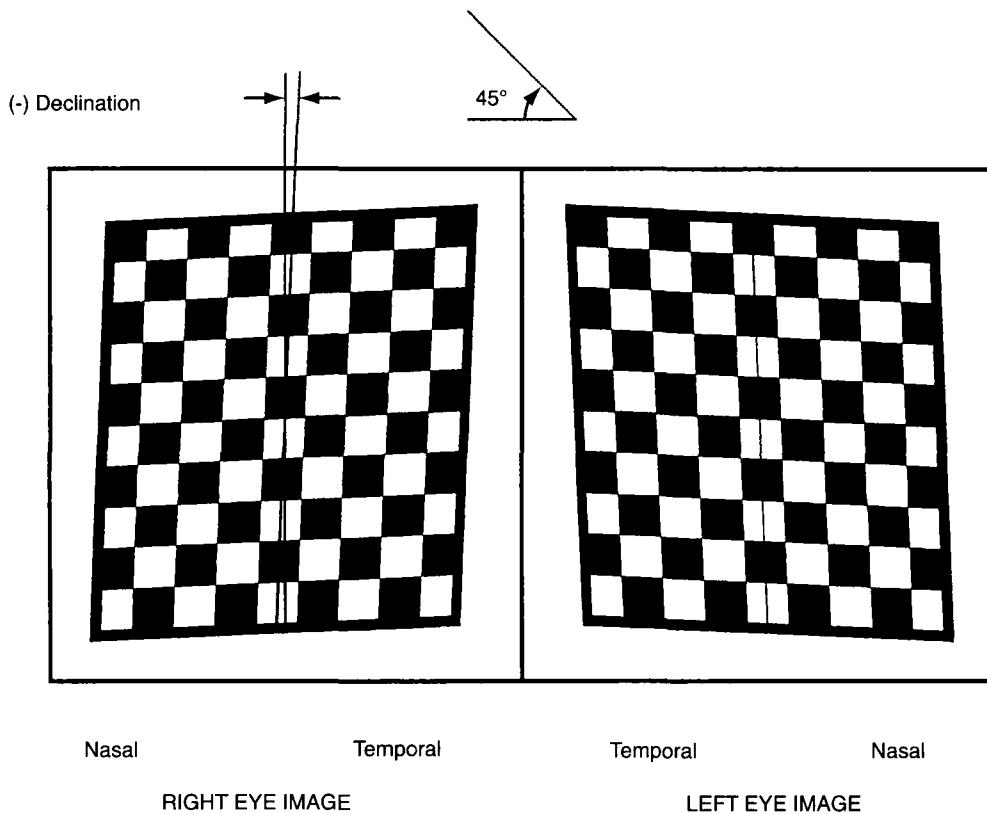
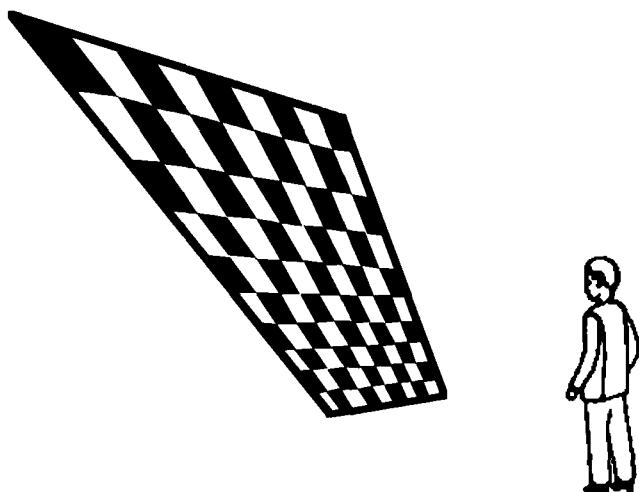


Figure 5-20

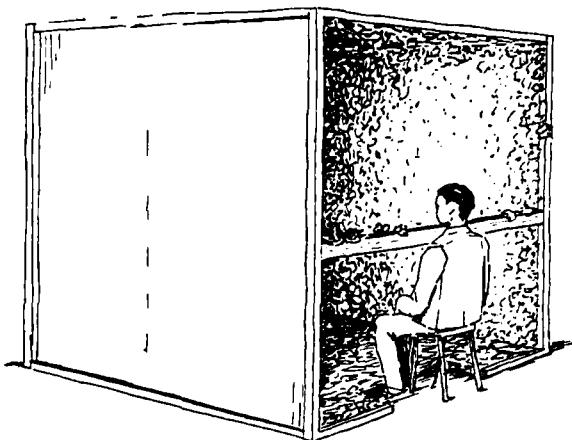
These distorted checkerboards simulate the effect of oblique aniseikonia on the retinal images or the effect of meridional magnifiers whose axes are placed at 135° and 45° before the left and right eyes, respectively. *Declination* is the tilt of the vertical meridians of the images. The tilt of the top of the vertical meridians away from the nose (toward the temporal visual field) is referred to as a *negative declination*. When the targets are chiastoptically fused according to the instructions given in the legend of Figure 5-1, the fused target will appear to be rotated about a horizontal axis as if it were farther from the observer at the top and closer to the observer at the bottom. The tilt of the distorted targets may be compared with the vertical orientation of the frame containing retinal images that are not distorted. The distortions of the checkerboard targets, which have been enlarged to better illustrate their shapes, cause large retinal disparities when their images are fused at 40 cm. Fusion may occur more easily if this figure is viewed from a greater distance.

oblique effect stereoscopic distortion. When the observer foveally fixates the center of the fused object plane, the vertical meridian lines above the fixation point are displaced to the temporal visual fields (nasal retinas), subtending increasing uncrossed disparities with vertical eccentricity. Accordingly, the gaze-normal plane appears to tilt away from the observer at the top. Similar reasoning accounts for the apparent tilt of the lower portion of the object plane toward the observer. This oblique effect cannot be explained by invoking the geometric and induced effects, because the horizontal and vertical sizes of the retinal images are equal. Oblique-effect stereoscopic distortion is additive with the geometric and induced-effect distortions. The retinal images caused by oblique magnification are similar to the images seen by an observer with normal vision viewing an object plane rotated about a horizontal axis.

Although object planes are useful for analyzing stereoscopic distortion, they are *not* useful as a clinical test, because monocular depth cues contained in them may interfere with aniseikonic stereopsis. Gillam¹⁵⁹ observed that, when the quantity of linear perspective depth cues in a binocular target was increased, stereoscopic distortion induced by size lenses was reduced. The conflict between stereoscopic slant and perspective slant apparently caused the suppression of stereoscopic slant. Longstanding suppression of aniseikonic stereopsis (stimulated by conflict with veridical monocular depth and distance cues) likely causes the reduced stereoscopic acuity observed in some of those aniseikonic patients who retain fusion. Aniseikonic distortions caused by stereopsis are clearly visible in a device known as the "leaf room," a small room that has interior walls that are covered with leaves³³ (Figure 5-22). The room is visually

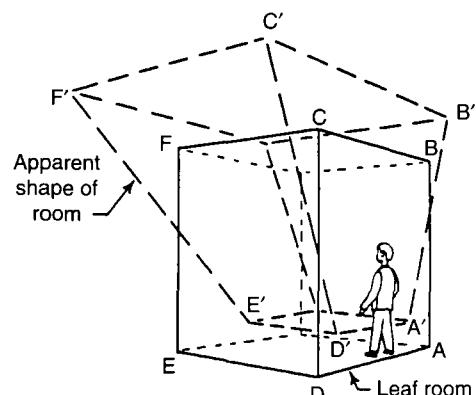
**Figure 5-21**

Perspective drawing of the appearance of the gaze-normal plane when viewed through magnifying lenses that stretch the retinal images in the 45- and 135-degree meridians of the left and right eyes, respectively. When the targets in Figure 5-20 are fused, the observer should see a slant of the checkerboard that is qualitatively similar to this perspective drawing.

**Figure 5-22**

The leaf room. This room which accentuates the stereoscopic perception of visual space, thereby revealing stereoscopic distortions. (From Ogle KN. 1950. Researches in Binocular Vision, p 144. New York: Hafner. By permission of Mayo Foundation.)

analogous to a random-dot stereogram, but it covers the entire binocular visual field. The highly textured walls of the leaf room place a much greater weight on stereopsis than monocular depth cues, and so they reveal stereoscopic distortions that may be suppressed by monocular depth cues in other visual environments. An example of the appearance of the leaf room in aniseikonia is depicted in the perspective drawing of Figure 5-23. The observer has an oblique effect distortion induced by

**Figure 5-23**

Perspective drawing of the apparent shape of the leaf room as viewed by an observer wearing magnifying lenses that stretch the retinal images in the 45- and 135-degree meridians of the left and right eyes, respectively. The wall of the leaf room facing the observer is stereoscopically equivalent to the gaze-normal plane depicted in Figure 5-21. Objective leaf room corners A, B, C, D, E, and F are perceived to be at locations A', B', C', D', E', and F' after magnifying lenses are applied. (From Ogle KN. 1950. Researches in Binocular Vision, p 147. New York: Hafner. By permission of Mayo Foundation.)

magnifying lenses that stretch the retinal images in the 45- and 135-degree meridians of the left and right eyes, respectively. The Space Eikonometer is a practical clinical alternative to the leaf room for viewing stereoscopic distortion, because it presents a stereoscopic environment with minimal monocular cue contamination (see Chapter 32).

Moderate aniseikonic disparities fall within the range of normal stereopsis and are similar to disparities encountered by individuals with normal vision, but they still may cause symptoms in some patients.* When aniseikonic stereoscopic distortion is visible to the patient, it alters the apparent orientation of the ground and of large vertical objects such as trees.[†] This illusory perception causes symptoms of spatial disorientation, including dizziness, nausea, and vertigo. More often than not, aniseikonic patients do not experience spatial distortion during casual viewing circumstances, even if they have meridional aniseikonia.¹⁶⁰ Suppression of aniseikonic stereopsis by more veridical monocular depth cues probably explains this lack of perceived distortion. Nonetheless, many aniseikonic patients experience headache and eyestrain as a result of their condition,

*The prismatic effects of anisometropic spectacles, discussed previously, can also cause symptoms. The discussion that follows pertains to the effects of aniseikonia (not prismatic effects).

[†]Stereoscopic distortion may emerge because the aniseikonic patient enters a visual environment rich in disparity cues and weak in monocular cues, such as a forest or a rolling grassy field.

even if they do not experience visual distortion.¹⁶⁰ The physiological basis of these subjective discomfort symptoms is unknown, but it may be the anisophoric effects of anisometropic spectacles.¹⁶¹

When aniseikonia is induced by magnifying lenses in individuals with normal vision, those observers adjust the relationship between disparity and stereoscopic depth to reduce aniseikonic distortion.^{162–165} This adjustment is probably the result of an alteration of the depth cue averaging process (see Depth and Distance Perception), in which the weighting factor for stereoscopic depth is diminished and the weighting factors for monocular cues are increased. The reduced stereoscopic weighting factor presumably persists even in the absence of monocular depth cues, thereby causing reduced distortion in clinical stereopsis-based tests of aniseikonia. Whether patients with naturally occurring aniseikonia or with postsurgical pseudophakic aniseikonia are able to make similar adjustments of the disparity/depth relationship has not been determined.

Binocularity and the Determination of Refractive State

Binocularity During Subjective Refraction

Binocular vision can have a powerful stabilizing effect over accommodation and therefore can prevent over-minus refractions in many patients when binocular refraction methods are used. The reason for the stabilization is that, when an accommodative response is triggered by overminus, it activates excess accommodative convergence innervation. Compensatory negative disparity vergence innervation is recruited to offset the excess accommodative vergence innervation to maintain alignment of the eyes. Negative convergence accommodation (synkinetic with the negative disparity vergence) then cancels some or all of the initial positive reflex accommodation response. As a result, the eye does not accommodate to the tentative overminus lens, so blur ensues, thus leading the patient to reject the overminus. In essence, binocularity partially inhibits manifest accommodation during the binocular subjective refraction at distance.

During the *monocular* subjective refraction, with one eye occluded, the accommodative vergence stimulated by overminus turns the occluded fellow eye inward without disparity vergence compensation. Therefore, the reflex accommodation response to overminus is completed, thereby allowing clear vision through the overminus. Thus, overminus occurs relatively easily in the monocular subjective refraction, because the patient does not have to maintain single binocular vision by exerting fusional divergence. In addition, *binocular* refractions of patients with large far-point exophorias should be completed with caution, because the visual system may encourage overminus to achieve single

binocular vision via the associated accommodative convergence.

The binocular refraction testing environment also supports supplemental far-point testing of stereopsis, sensory fusion, and fixation disparity. There are few efficient alternatives for testing those functions outside of binocular refraction. See Chapter 20 for a complete discussion of the binocular subjective refraction.

Exophoria and Refraction

Large far-point exophoria may induce excess accommodation during far binocular vision, thereby lowering binocular visual acuity relative to monocular acuity. Borish¹⁶⁶ suggested that these patients might be using voluntary accommodative vergence to supplement their disparity vergence and therefore create voluntary synkinetic accommodation, which results in blur. An alternative explanation is based on the hypothesis that these patients have defective convergence adaptation. The high far exophoria of these patients suggests that either they have no convergence adaptation or that the convergence adaptation capacity they possess has been depleted by the effort to converge the eyes from a highly divergent tonic posture to their exophoric posture. In either case, the result would be the same: disparity vergence innervation would be used to converge the eyes from their vergence resting state to parallelism, thereby generating significant amounts of synkinetic convergence accommodation innervation and blur. This behavior complicates the refinement of spherical lens power, which is usually performed at the conclusion of the refraction, while the patient is binocular. One solution to this problem, for the sake of refraction, is to apply BI prism before the binocular refinement of spherical lens power¹⁶⁶ (see Chapter 20); this would reduce the positive disparity vergence and convergence accommodation. The amount of BI prism could be selected on the basis of the far-point associated phoria (i.e., the minimum prism that eliminates fixation disparity; see Chapter 21).

The ultimate solution to the problem of far-point blur caused by exophoria is slow convergence visual training, which is known to improve the rate of vergence adaptation.^{137,138} Rapid vergence adaptation would reduce sustained convergence accommodation in far vision. Alternatively, BI prism has been proposed as a permanent solution for far-point exophoric blur under the presumption that it reduces exophoria.¹⁶⁶ However, the prism would have to be evaluated by the prism-adaptation test to ensure that inefficient (slow) divergence adaptation does not reinstate exophoria over time.¹¹⁶

Refraction with Base-In Prism

Various noncycloplegic procedures are available to relax accommodation during refraction for cases in which

cycloplegia is contraindicated (see Chapter 20). Base-in prism is sometimes used to help relax accommodation during the delayed plus refraction procedure.¹⁶⁷ This type of prism relaxes accommodation by activating divergence, and, with it, synkinetic negative convergence accommodation. The relaxation effect of BI prism adds to any relaxation accomplished by negative reflex accommodation induced with plus lenses. The benefit of using BI prism with prolonged fogging procedures is compromised by prism adaptation, which reduces negative disparity vergence and therefore negative convergence accommodation over minutes of time.

The BI refraction procedure described below is based on systems-analysis principles and may be useful for those patients whose reflex accommodation does not respond well to myopic blur. Ong and Ciuffreda¹⁶⁸ reported that patients with transient far-point blur after near work may have low negative reflex accommodation gain and that patients who are developing late-onset myopia (after age 15 years) have low negative reflex accommodation gain. Saladin⁸⁸ observed that low accommodative gain is often found in patients with binocular anomalies such as convergence insufficiency. The low gain of negative reflex accommodation in these patients may impede refraction by preventing fogging procedures from relaxing accommodation. Negative convergence accommodation can relax a limited amount of accommodation in those patients, despite their poor response to blur. Low reflex accommodation gain for myopic blur can be identified by a poor response to plus lenses during the accommodative facility test (see Chapters 10 and 21).*

Here are the steps used in performing a BI refraction:

1. The procedure should be performed at far point. A near target is not recommended, because it elicits an unknown amount of proximal accommodation that is not directly affected by plus sphere or BI prism. Proximal accommodation opposes accommodative relaxation stimulated by sphere and prism.
2. Snellen letters containing middle spatial frequencies (e.g., the 20/100 or 6/30 line) should be used as an accommodative target to optimally stimulate reflex accommodation. Reflex accommodation responds more efficiently to middle spatial frequencies than to high spatial frequencies.^{169,170} The success of accommodative relaxation procedures ultimately depends on the ability of reflex accommodation to hold far-point clarity after it is attained by the procedure. The patient should be instructed to view the clarity of the *edges* of the letters when the practitioner wishes to accurately assess the patient's accommodative response.
3. The refracting room should be well lit. This aids the perception of the remoteness of the letter chart, further discouraging proximal accommodation.
4. The tentative far-point refraction should be placed in the phoropter and the Risley prisms inserted with an initial value of 0^{Δ} . The initial binocular sphere should be adjusted to the minimum plus that induces sustained far blur.
5. Base-in prism is increased at a *rate* of 2^{Δ} per second until the edges of the 20/100 Snellen letters are cleared. The letters become clear because of the relaxation of accommodative response induced by negative convergence accommodation. If no magnitude of fusible BI prism affects the clarity of the letters, either of two possibilities exist: (1) the patient has no latent hyperopia, or (2) the CA/C ratio is very low. If other indications still suggest the presence of latent hyperopia, the low CA/C ratio hypothesis may be true. If so, BI refraction should be abandoned in favor of techniques using blur to relax accommodation.
6. Plus sphere is added in 0.25 D increments until the sustained blur is reestablished.
7. Steps 5 and 6 are repeated until BI prism no longer restores letter clarity. The plus is then reduced to restore sustained far-point clarity while the patient continues to view the 20/100 letters through the BI prism. It is important not to exceed the BI-to-break prism value during the procedure, because a break of fusion indicates a loss of disparity vergence innervation and consequently the loss of negative convergence accommodation. As a result, the patient will slip back into an accommodative excess posture. Moreover, steps 5 and 6 should be accomplished as quickly as possible. If the patient is allowed to fuse through BI prism for too long, vergence adaptation will replace negative disparity vergence and its associated negative convergence accommodation.
8. When the maximum plus has been extracted from this procedure, the BI prism should be *very slowly reduced* to zero. A rate of BI prism reduction that induces blur is too fast. It may be necessary to allow several minutes of time to eliminate the BI prism. There are two reasons for slow prism reduction. First, time is provided for accommodative adaptation to adjust to the lower levels of fast accommodation innervation induced by prism; this helps to maintain relaxed accommodation. Second, if BI prism is suddenly

*Accommodative facility training can restore the gain of negative reflex accommodation in these patients,¹³⁶ which should help in maintaining far-point clarity.

removed after vergence has partially adapted to it, a sudden and large increment of positive disparity vergence will be required to negate divergent adaptation and maintain fusion; this causes synkinetic positive convergence accommodation and therefore reinstates far-point blur. Slow reduction of BI prism allows the vergence system to readjust to the far-point vergence demand without inducing significant positive convergence accommodation. The return of adaptive vergence to its habitual far-point value can be verified by determining that the far-point phoria has returned to its habitual value or, better yet, by demonstrating that far-point fixation disparity has returned to its habitual value.

9. After step 8 has been completed, the entire process can be reiterated to ascertain more plus, if necessary.

There are several limits to the use of BI prism to relax accommodation. The first of these limits is the CA/C ratio, which varies among patients with normal vision. Patients with low CA/C ratios experience little change of clarity from divergence, regardless of how much BI prism they accept. If the practitioner chooses to verify that a low CA/C ratio is compromising a BI refraction, a CA/C ratio measurement method like that described in Chapter 21 can be used, but BI prism would be applied rather than BO prism. A second limit of BI refraction is the limit of divergence. Because negative convergence accommodation innervation is a product of the CA/C ratio times the magnitude of negative disparity vergence, patients with limited divergence ability may not experience much change of accommodation, regardless of the CA/C ratio. A 20-year-old latent hyperopia patient with a typical 1:10 CA/C ratio and a 5^{Δ} BI vergence break limit can relax a half diopter of accommodation by the initial attempt at BI refraction. The third limit of BI refraction is vergence adaptation, which was mentioned earlier. Finally, this procedure is not compatible with fogging methods, because significant fog masks the small incremental changes of accommodation brought about by vergence.

Accommodation and Vergence Physiology During Phorometry

Phorometry consists of those tests of heterophoria, relative convergence, and relative accommodation that are accomplished with the aid of a phoropter or trial frame, Risley prisms, and Maddox rod. For many decades, these tests have served as standards for the testing of accommodation and vergence, whereas the Maddox model of accommodation and vergence has served as a framework for explaining patients' responses to these tests. For instance, the Maddox model postulates that the difference between a heterophoric vergence angle and the

binocular vergence angle is a measure of the amount of fusional (disparity) vergence innervation active during binocular vision and that a test of relative convergence measures the amplitude of fusional (disparity) vergence. The following control-systems explanation of accommodation and vergence behavior during phorometry tests shows that these tests are more complex than the Maddox model explanations would suggest. The reader may wish to consult the first section of Chapter 21 regarding phometric test methods before reading this section, and it may be helpful to refer to Figure 5-16 while studying the explanations.

Heterophoria

Heterophoria, or "phoria," is the misalignment of an eye that occurs when binocular sensory fusion is blocked. In far-point vision, normal heterophoria is nearly zero. The far phoria is determined by the tonic vergence resting state and negative accommodative vergence. The tonic vergence resting state is the vergence angle dictated by tonic vergence innervation alone. It is measured in the absence of visual stimulation and averages 4.8^{Δ} eso.¹⁷¹ The difference between the far phoria and the tonic vergence resting state has been explained as follows.¹⁷¹ First, the tonic accommodation resting state focuses the eye at approximately 66 cm (1.5 D)¹⁷²; this state is the accommodative response in the absence of visual stimulation. Second, for an emmetropic (or corrected ametropic) patient to attain far-point clarity, he or she must cancel the tonic accommodation innervation with negative reflex accommodation innervation. Third, the negative reflex accommodation innervation generates negative accommodative vergence innervation by synkinesis. Fourth, the negative accommodative vergence innervation diverges the eyes from their tonic vergence resting state; the resulting vergence angle is the far-point heterophoria.

The vergence angle observed during a near phoria test also involves multiple innervational factors. Blocking binocular fusion eliminates disparity vergence innervation, but the magnitude of the heterophoria that is revealed is not just a measurement of the disparity vergence innervation that was eliminated. Because of dual interaction, the loss of disparity vergence innervation initiates simultaneous changes of accommodative innervation, which may mask the decay of disparity vergence innervation. For the sake of discussion, these changes may be analyzed from the time of onset of cover test occlusion (a normal exophoric observer is assumed). Occlusion of one eye causes that eye to turn outward from the direction of the target. The decay of disparity vergence innervation brings about a synkinetic decay of convergence accommodation innervation, thereby necessitating an increase of reflex accommodation to maintain clarity. Simultaneously, proximal vergence innervation is slightly reduced by occlusion

because of the loss of binocular input to distance perception.¹¹⁹ The synkinetic loss of proximal accommodation innervation would require additional increases of reflex accommodation innervation to maintain clarity. Increases of reflex accommodation innervation that were stimulated by the loss of convergence and proximal accommodation innervations would activate additional accommodative convergence innervation by synkinesis, thereby offsetting some of the decay of disparity and proximal vergence innervation brought on by occlusion. Hence, the movement of the occluded eye to its heterophoric posture is determined by the simultaneous and oppositely directed changes of proximal and disparity vergence innervation on the one hand and accommodative vergence innervation on the other. The net magnitude and direction of movement (and, therefore, heterophoria) is determined by patient-dependent AC/A and CA/C ratios and proximal processing. Accommodation and vergence probably do not change their adaptive states during the brief period used to measure heterophoria (up to 30 seconds).

Relative Convergence

Historically, the term *relative convergence* was thought to refer to a measure of the limit of convergence while holding accommodation constant. The limit is revealed when blur or diplopia ensues. Accommodation is not truly constant during such tests, even when the patient experiences subjective clarity. Therefore, the term is used to refer to the ability to change the angle of convergence while maintaining subjective clarity.

For the sake of discussion, positive relative convergence is analyzed, and it can be assumed that similar arguments apply to negative relative convergence. It is also assumed that convergence is stimulated by smoothly incrementing variable-power prisms at a rate of change of 2^{Δ} per second, which is optimal for disparity vergence.¹⁷³ Base-out prism displaces the retinal images temporally, thereby producing a crossed disparity that stimulates positive disparity vergence innervation via feedback control. Positive disparity vergence generates synkinetic convergence accommodation innervation via the convergence accommodation synkinesis. This additional accommodative innervation, if unopposed, would blur the ocular images. Negative reflex accommodation innervation is recruited to offset the excessive convergence accommodation and maintain clarity. This negative reflex accommodation then reduces accommodative vergence innervation via the accommodative vergence synkinesis. This reduction of accommodative vergence is usually less than the initial disparity vergence stimulated by the prisms. However, the reduction of accommodative vergence, if unopposed, would cause ocular misalignment and diplopia. Consequently, additional positive disparity vergence innervation is recruited to maintain ocular alignment, and it synkinetically generates more convergence

accommodation. It might appear that a never-ending loop of accommodation/vergence interactions might cause accommodation and vergence to assume extreme values for even a small value of prism. However, equations published by Schor¹⁰⁹ suggest that normal reflex accommodation and disparity vergence innervations stabilize at values that allow clarity and singleness for modest degrees of prism. The speed of this dual interactive process in individuals with normal vision is virtually as fast as the rate of prism application. Sustained perceived blur occurs when convergence accommodation innervation exceeds the offsetting capacity of negative reflex accommodation.¹⁷⁴ Diplopia occurs when the limits of both positive disparity vergence and voluntary vergence have been exceeded. In patients with abnormally high or low synkinesis ratios, the innervation required to maintain alignment and clarity during a relative convergence test may be many times larger than the value of the prism itself and therefore causes abnormally low findings.

Vergence adaptation contributes to the vergence response during the relative convergence test. Vergence adaptation is stimulated by the increase of fast vergence innervation as the prism magnitude is incremented. The percent contribution of adapted vergence innervation to total vergence innervation is strongly influenced by the rate of prism application. If prism power is incremented at the disparity vergence speed limit (i.e., fusion is maintained), little vergence adaptation occurs because of the inherently slow rate of vergence adaptation. If a slow rate of prism application is used, vergence adaptation innervation may contribute a significant percentage of the total vergence response. Vergence adaptation during the relative convergence test has two immediate consequences. First, because adaptive vergence innervation does *not* innervate accommodation via synkinesis, it does not upset the accommodative response. Hence, clarity is more easily achieved if adapted vergence innervation supplies the required vergence innervation. Accordingly, the range of clear vision in the relative convergence test is increased by vergence adaptation. Second, when adapted vergence innervation assumes a higher percentage of the vergence innervation load, more disparity vergence innervation is relieved of its load. This untapped disparity vergence reserve is then available for response to additional prism, and it therefore increases the range of single vision.¹⁷⁵ Because the prism rate alters the outcome of relative convergence tests, it is important to use a consistent rate (e.g., 2^{Δ} per second) to obtain results that are comparable with published standards.

The contribution of vergence adaptation to a relative convergence test may be observed in the phoria measured immediately after the relative convergence test. After positive relative convergence, the phoria may be several prism diopters more eso than the phoria measured just before the relative convergence test. After a

negative relative convergence test, the phoria will be more exo. Although adaptive effects usually indicate strong vergence function, they nonetheless may bias the results of subsequent phoria or vergence tests, especially if a positive relative convergence test is performed first.¹⁷⁶

Relative Accommodation

Relative accommodation tests (e.g., the positive relative accommodation and negative relative accommodation tests) stimulate not only accommodation but also vergence by way of dual interaction. For the positive relative accommodation test (minus-lens-to-blur), concave (minus) lenses stimulate positive reflex accommodation innervation via feedback control. Positive reflex accommodation generates additional accommodative vergence innervation via the accommodative convergence synkinesis. This additional vergence innervation, if unopposed, would cause diplopia. Negative disparity vergence innervation (i.e., divergence innervation) is recruited to offset the excessive accommodative vergence innervation and maintain alignment. Negative disparity vergence then reduces convergence accommodation innervation via the convergence accommodation synkinesis. The reduction of convergence accommodation, if unopposed, would cause a lag-induced blur; consequently, additional positive reflex accommodation is recruited to maintain clarity. As is seen in the relative convergence test situation, the innervational interplay stabilizes in a short time.

Accommodative adaptation can play a significant role in relative accommodation tests. Accommodative adaptation contributes to the relative accommodation response in two ways. First, as accommodative adaptation assumes more of the accommodative innervational load, reflex accommodation is released, thereby becoming available for additional response to minus lenses. Second, accommodative adaptation reduces accommodative vergence and subsequent dual interaction effects, thereby blocking the growth of negative convergence accommodation. These contributions depend on the speed of the relative accommodation test. Accommodative adaptation is minimal when the patient is rapidly responding to the minus lenses, but it is more important when positive reflex accommodation nears its innervational limit. Near that limit, the patient is observed to require more time to clear minus lens increments. The slow response is caused by the patient waiting for accommodative adaptation to develop, which eventually clears the target. Hence, the range of relative accommodation is increased if time is provided for accommodative adaptation to occur. If accommodative adaptation is strong and the positive relative accommodation test is performed slowly, some patients can clear minus lenses up to the minus lens amplitude of accommodation limit. Patients with simple convergence insufficiency—who are known to have exception-

ally robust adaptation of tonic accommodation¹¹⁵—behave in this manner. Hence, a consistent rate of lens application is desirable for the relative accommodation test. A rate of 0.25 D per second is useful, because normal reflex accommodation can follow the lens changes, whereas accommodative adaptation cannot.

The relative accommodation tests usually show blur before diplopia, and usually diplopia is not observed at all. This behavior again points to the relative weakness of reflex accommodation when accommodation and vergence mutually interact during phorometry. In other words, the capacity of reflex accommodation to respond to the combination of minus lenses and negative convergence accommodation is less than the capacity of disparity vergence to offset accommodative vergence. Those capacities are directly related to the gains of reflex accommodation and disparity vergence.

Summary of Phorometry

The heterophoric vergence angle is governed by varying amounts of tonic, accommodative, proximal, and adaptive vergence innervations. The disruption of binocular vergence that reveals heterophoria eliminates disparity vergence innervation, but it also alters accommodative vergence innervation by way of dual interaction. Hence, the magnitude and direction of heterophoria are determined by both the vergence and the accommodation systems.

Tests of relative convergence and relative accommodation place opposing demands on accommodation and vergence. BI prism and minus sphere demand increases of negative disparity vergence innervation and positive reflex accommodation innervation, whereas BO prism and plus sphere demand increases of positive disparity vergence innervation and negative reflex accommodation innervation. All of these tests are affected by adaptational behavior and therefore are time sensitive.

The innervational mechanisms that control the outcomes of phorometry tests are so complex that it is perhaps impractical to attempt to analyze their individual actions in a clinical setting. A more useful approach may be to view these tests as nonspecific probes of accommodation and vergence plasticity, correlating their limits to published standards of normality such as Morgan's "expecteds"^{177,178} or graphical analysis (see Chapters 21 and 22).

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6

The Ophthalmic Case Historian

Charles L. Haine

Most clinicians know that providing patient care is a combination of art and science. During the eye examination—and particularly during clinical refraction—there appears to be more science than art. Most of the clinical data that the clinician collects can be quantified, and the clinical procedures used to gain the information are grounded in visual science. Case history is one example in which art is more at play. During the taking of the case history, the clinician must listen to the patient and attempt to understand exactly what the patient is trying to convey. The taking of a case history from a patient is very much a problem-solving exercise; in this exercise, the patient is the one who knows what is bothering him or her, and the physician must first elicit the complaint and then follow that with more questions to determine the nature of and reason for the complaint. The physician must ask the correct questions to obtain the crucial details about that complaint. It is this social interchange between patient and physician that makes or breaks a clinician in his or her quest to understand the patient's problems.

With experience, the clinician learns to do what is known as "pattern matching."¹ During this process, a patient presents with a set of complaints, and the clinician listens to and records them. The experienced practitioner then compares the signs and symptoms to similar sets of complaints from past patient encounters. As the clinician sees more and more patients, his or her accuracy of recognition of clinical entities improves to the point at which valid provisional diagnoses are made in the vast majority of case histories that he or she takes.

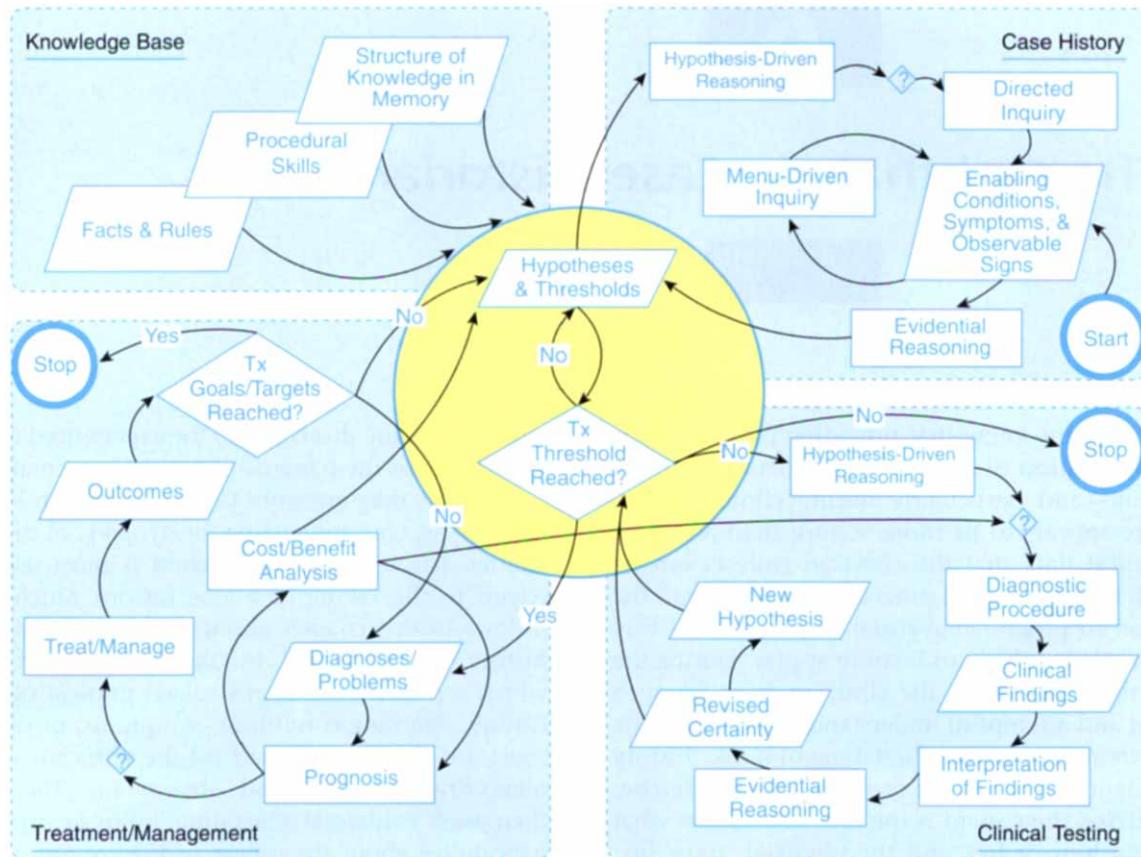
For student clinicians, the process is slightly different, because they have not yet built a base of clinical patterns; the student must collect information from the patient and then form conditional hypotheses that can be tested. These hypotheses can be tested by gathering additional information from the patient about the complaint and by beginning to collect objective data during the commencement of the clinical examination. There is a difference in the type and amount of hypotheses (differential diagnoses) formed by the two groups. The experienced practitioner forms fewer hypotheses, which usually are more specific than those of the student

clinician. Again, this is due to the experienced clinician's knowledge of the patterns of patient presentations.

The flow diagram from Corliss² shown in Figure 6-1 is useful for conceptualizing the dynamic of clinical reasoning. The top half of the chart is most useful with regard to the taking of a case history. Much of what follows in this chapter about the structure of the case history is represented by the menu-driven inquiry, which represents the standardized portion of the case history. Enabling conditions, symptoms, or observable signs are the facts elicited by the clinician using the menu-driven inquiry and observation. The clinician then uses evidential reasoning to make preliminary hypotheses about the causes of the conditions, symptoms, and observable signs. Lastly, the clinician reviews the hypothesis and further refines the line of questioning (hypothesis-driven reasoning), which the practitioner then uses to narrow the number of possible diagnoses. This process becomes second nature to the experienced clinician, who rarely thinks of evidential reasoning or menu-driven inquiry. However, to the new clinician, who has not been raised in a problem-solving environment, this is a new way of thinking about people and their problems.

This chapter focuses on providing the clinician with the tools needed to use the upper-left-hand portions of Figure 6-1. Although this chapter cannot provide all of the facts and rules that are needed to be an excellent clinician, many are provided as examples; it is hoped that this will be a good basis on which to build. What can be addressed are the procedural skills and the structure of the knowledge base in memory. The procedural skills include instruction about how to conduct an interview, and the structure of the knowledge base forms the outline of the following sections of this chapter.

It should be noted here that taking a case history is not limited to the early portion of the examination process; rather, it is a dynamic interaction that continues throughout the entire examination. (Note the arrows in Figure 6-1 that go to the Case History area from the lower portions and those that originate in the Case History area and point to Clinical Testing or Treatment/Management.) Tradition has dictated that the case

**Figure 6-1**

Comprehensive model of clinical decision making. See the text for a detailed explanation of the model. *Tx*, Therapeutic.

history be recorded at the top of the clinical record. However, much of the history can be—and, in fact, is—obtained during the examination process. It would be an interesting experiment to record case history on the examination record form at the point at which it is actually obtained during the examination process. In other words, if a clinical finding stimulates the clinician to ask a question, the patient's response would be recorded immediately following the results of the clinical finding that provoked that line of questioning. This would produce a case history that was continuous, which began at the onset of the patient encounter and ended after the last clinical finding was determined. As an academic exercise alone, it is likely that a clinician would gain significant new insights into the clinical decision-making process that he or she uses.

It is the patient's complaints and supporting information that provide the road map for the clinical examination. Although it is difficult to imagine examining a patient without ascertaining his or her clinical history, without such information, the clinical examination would drift aimlessly to a faulty conclusion, or the clinician would have to perform every examination

procedure known so that nothing was missed. Case history is the key to an efficient, comprehensive, problem-oriented examination of the patient and resolution of the patient's problems.

Since 1971, the problem-oriented medical record of Weed³ has been the standard for clinical record keeping. It is based upon the SOAP format, which stands for subjective, objective, assessment, and plan. The subjective of SOAP is the case history or the information imparted by the patient about the reason that he or she is seeking care. The point of mentioning the problem-oriented record is that, when it is time for the practitioner to assess and plan for the management of the patient, the clinician is obliged to link the assessment and plan back to the complaints of the patient.⁴ This closes the loop: the clinician is expected to deal with the reasons that the patient presented for examination (e.g., the chief complaints and any secondary complaints that the patients presents during history taking).

It matters little what type of record keeping is performed, because it will yield few valid results if the case history is ignored. During the recent past, new practitioners entering the eye care professions have been

shocked by the brevity of ophthalmic record keeping when they visit an established ophthalmic practitioner's office. It mattered not whether it was an optometrist or ophthalmologist they were visiting; the record of the examination was not as robust nor was it as well documented as they were taught it should be during their professional education. In particular, the case history has been an area in which practitioners have recorded little beyond the details surrounding the patient's visual problem. However, this situation can no longer be tolerated, because medicolegal considerations have changed the practitioner's responsibility to document. "If it is not recorded, it was not done" is a dictum that a clinician should carry into the examination room.

Furthermore, as the move to managed care increases, eye care practitioners are going to be practicing in health-maintenance organizations, hospitals, and government medical programs; credentialing and privileging will become paramount issues for these practitioners. In these arenas, there is both internal and external review of medical records for quality assurance purposes. The internal review is one of the means of documentation of care rendered⁵ that a hospital executive committee will use to establish credentials and privileges.⁶ The Joint Commission on Hospital Accreditation Organization (JCAHO) makes periodic external reviews of most civilian and government health-care facilities. As part of that review, record perusal for completeness and relevance is a standard procedure. Here again, the eye care practitioners must have high-quality records,⁷ including case history, about the patients that they have treated to help the hospital gain JCAHO accreditation status.

Finally, ophthalmic practice patterns are changing, and, with that change, clinicians need to alter the information that they elicit during the case history. As recently as 1972, by law, only ophthalmologists (in the eye care field) could use pharmaceutical agents in their practice; today nearly all optometrists have the same or similar opportunities to treat ocular disease. With this shift in the scope of practice, it is no longer acceptable to query the patient only about the visual system and to then begin examining the patient. It is necessary to know something about the patient's general health, both past and present, because that may alter the formation of the diagnostic hypothesis. The concept of "standard of care" has placed further emphasis on the importance of case history in the delivery of quality health care by vision-care providers.

CONTENT

Demographic Data

The case history is opened with some introductory information about the patient.

Age

The age of the patient is the first piece of information that is recorded. It is common knowledge that certain diseases and conditions are more prevalent in certain patients of a given age range. For instance, chickenpox is a common childhood disease, and shingles is a common disease in adulthood; both are caused by the same virus, but they have different manifestations that depend on the age of the patient. Presbyopia is not a concern with an adolescent patient, but it is important for the 55-year-old adult. The age of onset of a disease is critical to the diagnosis and prognosis assigned to the patient.

Gender

From a vision-care professional's point of view, the most obvious sex-linked hereditary problem is color-vision deficiencies. There are many more differences between males and females in the distribution of disease. In addition to the obvious anatomical and physiological differences, men and women tend to lead different types of lives. Men tend to abuse their bodies more than women (substance abuse being much more common in males). Until the last 40 years, women did not smoke cigarettes at the same population rate as men. Now the health consequences from smoking for the two sexes appear to be converging to a point at which women have the same risk as men.

The Chief Complaint

The chief complaint is the reason that the patient has come to the physician for this particular examination. (No patient thinks of history taking in these terms, but all clinicians should.) The chief complaint is usually limited to one to two sentences. When recording the chief complaint, it is best to record it in the patient's own words, without any interpretation by the examiner. The chief complaint is best elicited by asking patients what is bothering them or why they made the appointment. Open-ended questions work best in all parts of the case history, but this type of questioning is crucial when detailing a complaint. Try not to use directed questions—for example, "Is your vision blurry?"—because the patient may try to help the physician too much. These questions may elicit inappropriate responses merely because the patient thinks that the physician wants a given answer. Also, it is much easier for the patient to fabricate a response to a direct question. Again, the patient is not usually trying to deceive the physician, but rather he or she is trying to be helpful. Not only does direct questioning lead to invalid responses on the part of the patient, but it is also limiting. There are only two responses to the question, "Is your vision blurry?": yes or no. In this case, the patient may have volunteered other information had the

question been asked in an open-ended manner. Lastly, the terminology used should come as close as possible to that of the patient. If a patient is asked if he or she has ever had iritis, he or she will likely give a different response than if he or she had been asked about a previous episode of sensitivity to light and a red eye.

History of Present Complaint

The history of the present complaint is that point at which the clinician details or characterizes the chief complaint of the patient. It is here that the philosophical difference in the clinician's approach between vision problems and disease is noted. With ocular disease, the standard medical model works with ease, but vision problems are not quite the same; the differences are examined below. With the medical model, there are several areas of pursuit that work for most signs and symptoms. The complaint is specified using the following categories.

Location. Where is the sign or symptom manifested? If the complaint is pain, can the patient localize it? Sometimes it is good to have the patient point to the location. For internal symptoms, the patient can still localize the area of the symptom by pointing with his or her fingers in two planes of reference. With a blurred vision complaint, the location is logically a point in space at which the images are blurred.

Severity. How extreme is the symptom? This is usually thought to be limited to pain, but it can apply to most signs and symptoms. It can even apply to blurred vision, although it is hard to get patients to characterize blur in terms of severity. When the symptom is pain, the adjectives used to describe it would be sharp, dull, lancing, piercing, radiating, and excruciating. Pain severity is best measured on a 1-to-10 scale by asking the patient, "How severe is the pain that you experienced on a scale of 1 to 10, with 1 being minimal discomfort and 10 being unbearable pain?" The response is then recorded using this scale.

Character of the Sign or Symptom. What type of pain is it? Is it boring, sharp, or dull? Does it radiate? If blur when reading is the complaint, is the blur constant at a fixed distance, or does it come on after reading for a while? Is the blur from the letters splitting into two or just a constant sustained blur at the reading distance? Does it occur when going from the end of one line to the beginning of the next? All of these characteristics have differing etiologies and require differing treatment strategies.

Nature of Onset. Did the sign or symptoms come on suddenly, or was the onset so slow as to leave the patient with an unclear knowledge of the onset? Sudden painless loss of vision in one eye might have a vascular etiology, whereas gradual loss of vision might be the result of a space-occupying lesion in the cranium. A

slow onset of blur at a distance may be associated with an increase in myopia.

Duration. How long has the complaint been present? A patient with a sudden loss of vision of 1-hour's duration has a much better prognosis than does the same patient after 10 days with the same complaint. The duration can be critical to a diagnosis. For instance, the visual aura of migraine is almost always described as lasting approximately 20 minutes and disappearing before the headache commences. If the aura lasts longer than 20 to 30 minutes or extends into the headache phase, the clinician should investigate other causes for the visual symptoms besides migraine.

Frequency. If the complaint is not constant, what is its periodicity of recurrence? This is the area in which to ascertain the nature of the frequency; in other words, is the periodicity of the symptom increasing, decreasing, or stable? A patient with a pattern of increasing frequency of transient ischemic attacks (TIAs) is of more concern than a patient with only rare TIAs.

Exacerbation and Remission. It is not uncommon for a condition to have periods in which the patient has no signs or symptoms; however, the condition can still be present. At other times, the patient has signs and symptoms of the disease. It is necessary to characterize the nature and extent of periods of exacerbation and remission. A classic example of a disease of this type is multiple sclerosis, in which the symptoms wax and wane over a period of weeks to years. The signs and symptoms get progressively worse, and the patient never does return to normalcy during periods of remission. However, the disease does not produce a steady downhill progression.

A more pertinent example is recurrent herpes keratitis due to the herpes simplex virus. Here the exacerbations are followed by varying periods of remission. Each exacerbation may be marked by its own corneal opacification, which is the only sign that the patient has anything wrong with the cornea during the periods of remission. It is not known what makes the virus become active, but, when it does, it produces signs and symptoms in the nerve endings of the same nerve that was involved previously.

Relationship to Bodily Activity or Functions. The clinician must be vigilant to ascertain if there is any relationship between the complaint and bodily functions or activity. For example, does the blur increase when the patient is reading? Is there claudication on mastication? Does the headache get worse when reclining, or does the headache come on daily at the end of the work period? All of these are important clues to the etiology of the complaint, and they can be helpful to the practitioner.

Accompanying Signs or Symptoms. When discussing the patient's complaint, the clinician should be aware of any relationship to other signs or symptoms.

For example, diabetic patients may note that their distance vision is blurred on some days and on not others. If asked, they may be able to further explain that their blood sugars are usually elevated during the same day that they noticed the blurred vision.

Migraine is diagnosed only after determining that the appropriate antecedent components are present and that they are followed by an appropriate type of headache. In other words, a headache without accompanying signs and symptoms may be a migraine, but the diagnosis is much more certain with the requisite company.

Most of the preceding could apply to a vision problem, but the language does not readily adapt. When there is a vision-related complaint, the patients often do not know at what point their vision became blurred. Alternatively, the problem may not wax and wane. What does frequency mean when applied to blurred vision? Furthermore, the visual system is measurable without invasive procedures, and the patients are unable to quantify their complaints in precise terms.

This discussion brings to mind a 55-year-old over-the-road truck driver who noticed blur at near during his hospitalization for a myocardial infarction. He was positive that the blurred vision was caused by his myocardial infarction. In reality, his lifestyle changed radically when he was hospitalized, and it is probable that changes in the way he used his visual system were the cause of the apparent sudden change in vision (rather than the heart attack). In this case, the onset would be reported as sudden when, in fact, the onset was gradual. It is best to be wary of the history of patients without being disbelieving.

Secondary Complaints

The clinician wants to characterize these complaints much as he or she did the chief complaint. Hence, most of the preceding information will be included for these complaints as well. Often these complaints are part of the same problem that is causing the chief complaint, but not always. In fact, it is in this area of the history that the clinician should always try to invoke the "law of parsimony." This law, simply stated, holds that, if one diagnosis fits a group of signs and symptoms, it is the best choice of a working diagnosis or hypothesis. Let us examine this in more detail. If Condition 1 has *a* and *b* for symptoms, Condition 2 has *a*, *c*, and *d* for symptoms, Condition 3 has *a*, *b*, *c*, and *d*, for symptoms and the patient has symptoms *a*, *c*, and *d*, it is more parsimonious to make the diagnosis of Condition 2 for this patient. Condition 2 has all three symptoms that the patient manifests and is the most likely choice, but Condition 3 cannot be ruled out, because symptom *b* may be silent in this patient. The clinician needs to investi-

gate more symptoms or signs before discerning between Conditions 2 and 3.

Ocular History

One should elicit information in the following categories about the patient's ocular history.

History of Spectacle Wear

The clinician should gather information about the first time glasses were prescribed to the patient, how the patient wore those glasses, and when or if subsequent glasses were prescribed. The wearing pattern and time of first prescription may yield significant information about the type and magnitude of the visual correction of the patient. It is common to obtain a history of someone being given reading glasses during his or her school years only to find that he or she has not worn glasses for years (these patients are usually hyperopic).

Last Eye Examination

Date of Examination. The date of the last ocular examination seems to be limited in informational content, but it can yield much inferential information about the importance that the patient places on visual symptoms and their impact on the patient's lifestyle. In some clinical settings, it is not unusual to have a patient who has never undergone an eye examination.

The clinician must also be careful that the patient is not confusing an eye screening with an eye examination. A frequent response to the question about the date of the last eye examination is, "I had my eyes examined about 1 year ago." Upon further questioning, the patient reveals that the eye test was part of the driver's licensing examination or a physical examination by his or her primary care physician.

Results. The results of the last eye examination may provide useful information about conditions that are not apparent upon casual observation of the patient, and these may direct the examination of the patient in a way that was not expected. The physician might ask, "What did the physician tell you about your eyes at the completion of your last examination?"

Suggested Treatments

In this area, is it easy to ask a question that is too limiting, such as "Did your last physician recommend any change in the type of spectacles or the way in which they would be used?" A better question in this area might be, "Did the physician recommend any new treatments or a change in the type of treatment for your eyes?" The first question ignores every type of treatment besides spectacle wear. Clearly, a more open-ended question yields better information.

History of Any Ocular Surgery

This history may be significant for the present complaint, even though the patient may not connect the present complaint with the previous surgery. For example, when one is trying to ascertain the etiology of diplopia, knowledge of childhood squint surgery or refractive surgery as an adult can be beneficial. It would certainly complicate the physician's diagnostic evaluation if he or she were unaware of such a history.

History of Any Ocular Disease or Trauma

If for no other reason than the fact that history is bound to repeat itself, a complete history of ocular disease and trauma is necessary. This would include not only the type of episode but also the type of treatment rendered and any sequelae.

Often the patient is not aware of the significance of prior trauma on the current condition for which they have sought care. A history of blunt trauma to the eye is crucial when a patient has monocular glaucoma or is about to undergo cataract extraction with intraocular lens implantation; the patient would not be aware that such a history has any significance.

Medical History

Here the clinician wants to describe significant past illnesses or injuries and any sequelae from those episodes. In addition, previous surgical procedures could be helpful. A young adult male patient with chronic back pain and recurrent red eye would lead a clinician to consider ankylosing spondylitis. The same patient without the back pain complaint would pose a more vexing diagnostic problem.

Drugs and Medications

Prescription medications should include the name of the drug, the reason that it is being taken, the dosage, and the duration that it has been used. The relationship between systemic conditions and ocular problems is legendary. Medications taken by the patient can indicate the nature, course, and ocular complications of certain conditions. Diabetes is one such condition. The type of diabetes can be surmised from the type of medication used to treat it. From the type of diabetes, the course, prognosis, and ocular complications can be estimated.

Over-the-counter medications should include the drug name, why it is being taken, the dosage, and the duration of use. These drugs are often ignored during history taking, but they are important to the astute clinician. Antihistamines used to treat hay fever may be important for the narrow-angle glaucoma patient and for the patient with chronic allergic conjunctivitis.

Recreational Drugs

In this day and age, it is important to know if there is any type of street drug use occurring and the route of administration. Intravenous drug use is now the most common cause of acquired immunodeficiency syndrome (AIDS) infection in the United States. Such a history should alert the clinician to be particularly vigilant for cytomegalovirus retinopathy or human immunodeficiency virus retinopathy.

Family Ocular History

This is the area in which the patient is likely to give more information than is really wanted. All too often, the patient will explain that both of his or her parents and all but one sibling wear glasses; this is not particularly useful information. The physician should be seeking information about ocular hereditary conditions and communicable diseases within the family or about those that are endemic to where the family lived.

Hereditary Conditions

Several hereditary conditions are relevant to the eyes, such as diabetes mellitus, color-vision deficiency, migraine, retinitis pigmentosa, and macular degeneration. The clinician's emphasis may shift with the demographic characteristics of the patient, but this is vital information for proper care of the patient.

Conditions that Might Be Transmitted from One Family Member to Another

A toxoplasmosis infection in a mother could lead to transplacental infection of the fetus with a subsequent chorioretinal scar in the child. It is the responsibility of the clinician to make the appropriate linkages between the patient's condition and the concomitant manifestation of the disease in a family member, because the link is not always evident to the patient.

Conditions that Are Endemic to Where the Child Was Living with His or Her Family

Histoplasmosis is one condition that comes to mind in this category. Knowing that the brother of the patient had a scar on the back of his eye and that the patient grew up in Illinois might create a high level of suspicion that the white spot on this patient's retina may be evidence of ocular histoplasmosis syndrome.

Family Medical History

Hereditary Conditions

This category involves any systemic hereditary condition that could affect the health status of the patient at the time of examination. For example, connective-tissue disorders can have ocular effects, but they are considered primarily systemic conditions.

Conditions that Might Be Transmitted from One Family Member to Another

A good example of this type of condition is tuberculosis. Public health officials thought that they had tuberculosis in check until the mid-1980s; it is now reaching epidemic proportions in the urban population as a concurrent rise in the number of new cases of AIDS is seen. It would be a significant finding that the patient's father has tuberculosis and has had it for 10 years, during which time the patient lived with his or her father. A large percentage of tuberculosis cases in the population are recurrent inflammatory reactions or reactivation of the mycobacterium.

Social History

Information here is related to the patient's habits and vocation.

Occupation

It is well established that certain visual needs are associated with certain occupations. For instance, pilots have rigorous standards for their visual status. Other vocations might demand normal color vision. However, beyond the vision system, some occupations involve enormous health hazards. The health care worker and the implementation of "universal precautions" by the Occupational Safety and Health Administration is a classic example. Furthermore, this area has obvious ramifications for the eye care practitioner, because working distance is critical for the presbyopic patient. In fact, there are special occupational bifocals for persons whose work area might be at or above eye level and, more recently, for persons who work with computer monitors during a significant portion of the work day.

Marital Status

Single young adults live a different lifestyle than do married young adults. Although this may seem irrelevant, the potential for exposure to sexually transmitted disease is vastly different for the two groups. There are differences in the life expectancies for single males versus married males, even when ignoring the potential for sexually transmitted disease. It seems that married males consume a better diet than single males and, therefore, have a longer life expectancy than do single males.

Avocational Interests

Like occupation, this information is critical for the clinician. Knowledge of hobbies and avocations is a major area in which the astute practitioner will pursue details to assist the patient with correcting his or her vision.

Alcohol Use

Here the clinician may obtain information about the patient's ability to comply with treatment regimens; he

or she may also gain information that has a direct bearing on the patient's overall health. A more recent concern is that of fetal alcohol syndrome and the delayed development that is common in children of mothers who drink.

Tobacco Use

Although many clinicians may think that smoking is not directly related to the eye, the ophthalmic practitioner does gain information that helps with understanding the patient's general health. A 42-year-old male who has smoked four packs a day for 20 years and who also has bilateral papilledema might lead the clinician to suspect a metastatic central nervous system lesion from a primary tumor in the lung. A more pertinent link may be the ocular surface problems that have been linked to smoking and that have further been associated with keratitis, which, among smokers, is more prevalent during the extended wear of soft contact lenses.

Review of Systems

Each of the following areas is an example of how the specific system might affect the ophthalmic practitioner's assessment of the patient.

Ear, Nose, and Throat

Because of the proximity of the nasal passages and actual connections between the two systems, ear, nose, and throat conditions can and do produce ocular signs and symptoms. The most obvious is allergic rhinitis, with its conjunctival component.

Cardiovascular

Hypertension and stroke have serious vision and ocular complications in addition to carotid artery disease, which is a common cause of TIAs and their associated visual symptoms. Marfan's syndrome is a hereditary anomaly with cardiac signs and symptoms and an ocular component that features subluxated crystalline lens as a result of zonular dysgenesis.

Endocrine

Diabetes mellitus and its ophthalmic complications are the most obvious potential problems involving this system. It would be remiss to not mention thyrotoxicosis, which can produce devastating visual complications, including blindness. Conjunctival hyperemia and mild proptosis may be the only signs of Grave's disease, which can lead to blindness in relatively rapid order from a compression neuropathy of the optic nerve.

Dermatological

The lids and lashes are often the site of more diffuse dermatological disease. Careful history taking in this

area can lead to better diagnosis. A good example is atopic dermatitis, which can be manifested in the palpebral conjunctiva.

Gastrointestinal

Not many diseases affect the gastrointestinal tract and the eye, but there are a few. Hermansky-Pudlak syndrome is a hereditary condition with gastrointestinal symptoms and ocular albinism as an eye manifestation. There is increasing evidence that diet and ocular conditions may be linked; gyrate atrophy is but one example.

Genitourinary

Reiter's syndrome is a condition of young males with sterile urethritis, arthritis, and conjunctivitis as the ocular involvement. The clinician must also remember that a history of sexually transmitted disease should place the clinician on alert for ocular problems. The ocular problems might be interstitial keratitis in patients with syphilis, iritis in those with disseminated gonorrhea, or cytomegalovirus retinitis in patients with AIDS. Also, the clinician should be highly suspicious of other forms of sexually transmitted disease when faced with a diagnosis of sexually transmitted disease.

Psychiatric

The first condition that a practitioner might list in this area is hysterical reaction, with tunnel vision as its classic symptom. Although startling, hysterical amblyopia is not the most common form of psychogenic visual problems of which the eye care clinician must be aware; rather, stress-related illnesses are the most common form of this type of condition. The alert clinician should be vigilant for this, because it can save con-

siderable time, cost to the patient, and frustration to identify functional disorders early on during the patient encounter.

For example, spouses of practitioners commonly have conditions that are related to the type of practice of their spouse. A case example is the wife of an optometrist who noted shimmering light in an oval shape to the temporal side of vision in her right eye during the week preceding Thanksgiving. She complained that this had been present for about a week and that, if she shut her left eye, she would lose the right three lanes of the highway. She underwent a workup with the appropriate ocular and neurological evaluations and was found to have an enlarged blind spot; all other test results were within normal limits. The symptoms continued unabated through the end of December and then disappeared. The symptoms reappeared during the spring of the following year, but this time they disappeared much sooner. Things were relatively quiescent until Thanksgiving of that year, when the scotoma and shimmering light reappeared and remained until the end of the year. It was then that the pattern became evident; when the spouse was to visit her mother-in-law, the condition would flare, only to resolve after the stressful situation would pass. This one was called "Mother-in-law's syndrome."

A more critical situation would be a patient whose compulsion was toward self-enucleation with his hands. He eventually was successful with one eye and came close with the other eye. This is not a common problem, but it is one that vision-care providers might learn of during the taking of the case history and when assisting the psychiatrist with the management of a patient.

A Simulated Case History

Although it would not be appropriate to use an actual case history, the following illustrates the form and content of a case history for a first-time patient. It is important to see a whole, intact case history so that the new physician has a concept on which to build his or her own case histories.

History of Present Complaint

This 22-year-old white female presents with a chief complaint of blur at distance. This blur has developed gradually over the last year. It is constant in nature and slowly progressive. It is not related to bodily function or activity. The blur seems to be worse at night, when she is driving. She does not notice an increase in blur when she is watching television at night. The blur is now causing problems reading road signs in time to make

the appropriate driving maneuvers while driving on city streets.

Secondary Complaint

The patient also complains of headache. She first noticed the headaches about 18 months ago and has seen her primary care physician about them, who told her that the headaches were migraines. These headaches occur about once a month, but they are not related to her menses. They involve unilateral, severe, boring pain (8/10) in the left temporal area. The patient can tell when the headache is coming by a feeling of euphoria that is followed by an aura of flashing lights, which expand to form a central relative scotoma. The aura lasts for 20 minutes, and then the headache begins. The pain seems to get more intense over the next 30 minutes or

so to the point that the patient may get nauseated and vomit. During the headache, the patient is hypersensitive to light and sound and usually seeks a quiet, dark room in which to lie down. The actual headache may last from 1 to 6 hours. Sleep is possible during this phase and often brings relief. The patient has not noticed any association with any bodily activities and has not noticed any relationship to foods that she has eaten during the 24 hours prior to the headache.

Past Ocular History

The patient has worn glasses since she was 12 years old to correct nearsightedness. Her last eye examination was 2 years ago, when she was given a new spectacle correction that she has worn constantly. Her optometrist had no other recommendations for her at last visit. She denies any ocular surgery or significant trauma. She did have an episode of "pink eye" when her little sister also had it about 14 years ago, which healed without sequelae.

Past Medical History

The patient had mumps and chickenpox as a child with no sequelae. She has had no other significant diseases or surgeries.

Drugs and Medications

The patient takes over-the-counter multivitamins as a dietary supplement and birth-control pills for contraception. The birth-control pills are low dosage, and she has been taking them for 4 years.

Family Ocular History

The patient's father (56) underwent cataract extraction with intraocular lens insertion in both eyes last year. Otherwise there is no relevant ocular history to report.

Family Medical History

The patient's father has a long history of asthma for which he uses inhalers. The inhalers are both bronchial dilators and steroids. Her mother (52) underwent lumpectomy of the left breast followed by radiation therapy of the breast about 3 years ago. No metastasis or recurrences to date.

Social History

The patient is the last of three siblings, all alive and well. She is a senior majoring in psychology at the local university. She enjoys racquetball and running when not studying. She is single and plans to pursue graduate work in political science. She has never smoked, and she drinks alcoholic beverages only rarely and never to excess.

HOW TO APPROACH THE PATIENT

Open-Ended Question

To obtain a clear case history, it is necessary to ask questions that the patient can respond to with more than a yes or no. These questions should be of the type that allows the patient to tell about his or her problems without the physician interrupting the storytelling. What is desired is to gain information about the patient's problems with the patient doing most of the talking. Judgmental statements or comments should be avoided. The clinician is acting as a recorder of the history and a guide to the patient on this journey. The clinician's moral and ethical beliefs have no place in the process.

Some examples may be, "Why did you make your appointment?", "Tell me about your visual problem," or "Have you had problems with your eyes in the past?" If previous issues are indicated, the physician could ask, "What might those have been?" Alternatively, the physician may inquire, "What type of eye problems have you had in the past?"

Minimal Direction

The above implies that the physician needs to direct the patient without leading him or her into responses that he or she thinks the physician wants to hear. It is an interac-

tive dialog between the clinician and the patient. The physician wants to be as polite and friendly as possible so that the patient feels that he or she can express this personal information to someone who will protect his or her privacy and who is vitally interested in his or her concerns.

Active Listening

The term *active listening* should apply to good history taking. The physician must be attentive to the patient while trying to record notes about what the patient is telling him or her. The physician should respond intermittently to what the patient is saying and then ask questions that indicate that he or she is listening to and understanding of the concerns and information that the patient is conveying. Asking good follow-up questions is key to making the patient feel that the physician has not only listened but has understood what the patient has said and that he or she is interested in the patient.

SYMPTOMS

Headache

Now that the structure and techniques of history taking have been outlined, the results that a careful history taking will yield need to be investigated. First to be

addressed is headache, because it is a common complaint. The worst thing that any practitioner can do to a patient is to tell a patient that the complaint is "all in the head"; this goes double for headache. Not only is it a bad pun, but it is also bad practice.

Most patients over the age of 8 years have had a headache at some point during their lives. In the majority of practices, more than 90% of these complaints are not related to the patient's eyes or visual system. It is the patient, thorough clinician who can differentiate between the types of headaches and their causes.

A *headache* is pain that occurs in the cranium, the nape of the neck, or the forehead. Most patients do not include ear, tooth, jaw, or eye pain in this complaint. Headache can originate from the musculature surrounding the cranium, pressure in the paranasal sinuses, or stretching of or traction on the intracranial or extracranial vasculature or pia mater. The substance of the brain (gray and white matter) is, for all intents and purposes, not pain sensitive. Most of the pathophysiology associated with headache helps with localizing the site of the cause of the headache. However, when it is traction or displacement of the associated structure, it is not possible to localize from the site of the pain to the site of the lesion; this is because space-occupying masses are the major source of traction or displacement, and the traction or displacement may be distant to the site of the lesion. Therefore, the site of the pain may be and usually is remote from the location of the mass.

Stress Headache

Almost everyone, at one time or another, has experienced a headache caused by stress, anxiety, or tension; this is the most common headache that a clinician encounters in practice. These headaches are much more common in adults. Patients complain of pain in the occipital region or the nape of the neck. Sometimes the patient presents with frontal pain, which is related to the same mechanism as the pain at the base of the occiput but which is transferred to the frontal region through the aponeurotica. The headache is often accompanied by a feeling of tightness that leads to a band headache. Classic stress headaches usually happen at work or school and occur during the late afternoon or toward the end of the work period. Also, these headaches may be related to vision problems. It has been demonstrated that a three-dimensional prism with a vertical orientation placed before the eye can produce a muscle tension headache after about half an hour of wear. The pain is usually constant, comes on gradually, and can build for hours. The pain is usually dull in the beginning and may proceed to a moderate degree. The patient is not disabled, and he or she may continue to function normally. Nausea and vomiting are not commonly associated with the pain. On palpation, the musculature of the back of the neck is taut and may be in

spasm. Salicylates provide relief for most patients; massage and support of the head also provide relief. In fact, this is the *only* type of headache that is relieved by support of the head, and the pain is not augmented by coughing or straining at the stool. Both are good diagnostic pearls.

To summarize, stress or tension headaches are characterized by pain in the nape of the neck, come on late during the work period, and are relieved by support of the head; the pain is not augmented by things that raise the intracranial pressure.

Vascular Headache

This type of headache is often confused with migraine, and, indeed, it may be a migraine. It is thought that the mechanism of this headache is the same as that seen in patients with migraine; the difference between the two headaches is in the etiology and some of the symptoms. The mechanism of a vascular headache is related to segmental constriction of an intracranial artery followed by dilatation of that segment. The pain is the result of the stretch receptors responding to the increase in caliber of the affected vessel during the dilation phase. The difference between this headache and migraine is that the vascular headache is most often due to a reaction to trigger substances. Most commonly, it is something that the patient has ingested during the prior 24 hours. The following items are most commonly linked to vascular headaches: red wine, dark chocolate, cheddar cheese, and crustaceans (e.g., shrimp, lobster). All of these items have vasoactive enzymes that produce localized vasoconstriction and rebound dilatation in sensitive persons. These headaches differ from migraine headaches in that the aura is rudimentary or absent in the vascular type.

Vascular headaches are usually throbbing in nature, at least during the early phase. The pain does build for about the first hour, and it may become constant as the intensity increases. The pain is usually isolated to a given region of the head, but it may radiate as the headache progresses. The pattern of location of pain repeats from episode to episode. Nausea and vomiting may occur later in the headache as the pain becomes severe. The pain lasts for hours and is not relieved by salicylates or support of the head. The pain is worse upon reclining because of gravitational effects on the intracranial blood pressure. The pain is also worse when coughing or straining at the stool. Like migraine sufferers, these patients often seek a quiet, dark room and attempt sleep. It is not known if sleep ameliorates the pain or if it is just the passage of time, but it does seem that some relief is gained upon awakening. Antihistamines relieve this headache rather promptly. Often patients relate that the headache is gone within 20 to 30 minutes after taking pseudoephedrine or a similar preparation. Unfortunately, migraines do not respond in the same manner.

Migraine Headache

Migraine is known by many names: migram, hemicrania, and sick headache, to name a few. It is characterized by pain on one side of the head, although simultaneous bilateral pain may occur. It is a familial disorder in which the child has a pattern of headache similar to that experienced by the parent. Migraine syndrome usually starts during the second or third decade of life. The attacks seem to appear fairly regularly; then, during the fourth decade of life, they subside for a period of 10 to 15 years, only to recur during the fifth and sixth decades of life. Although the mechanism was alluded to earlier, the trigger for the migraine incident is not known. It has been related to the menstrual cycle in females, the phases of the moon, stress, and other obscure causes. The classic model of migraine involves four phases: (1) the prodrome, (2) the aura, (3) the headache, and (4) post headache.

The Prodrome. This phase is the least well defined of the four. The astute patient notes that he or she may feel euphoric or depressed during the hours preceding the headache. The sensation may be less well defined, and the patient just has a "feeling" that the headache is going to happen. Some patients evidently do not experience this phase in their syndrome or, if they do, they cannot or have not linked the feelings with the migraine. Some patients note that they "retain water" on the day preceding the headache. Again, this phase is the most difficult to document.

The Aura. This phase is the eye care practitioner's friend, because a significant number of patients report the symptoms to the eye care practitioner first. The aura is usually visual, with something that Helmholtz named a *fortification scotoma* being the most common presentation. The fortification scotoma is a jagged, bright, margined visual phenomena that starts in the center of vision and gets progressively larger over a 5- to 10-minute period; it then collapses in the reverse order of progression. The name *fortification* is derived from the design of towers in European castles and the resemblance of the margins of the scotoma to that structure. The margins are usually reported to be colored, bright lights that have a shimmering quality. The aura is often called a *scintillating scotoma*. The visual image is usually bilateral, which indicates occipital origin, and it is most likely the result of ischemia from the vasoconstriction of early migraine. The pattern of the aura is usually consistent from episode to episode for a given patient. The aura lasts from 15 to 30 minutes, with most patients reporting that it lasts about 20 minutes. The aura is complete before the headache commences. If a patient should report that the aura persists into the headache, the headache is not a migraine.

The Headache. Unilateral pain in the cranium is the most typical complaint. This is severe, throbbing, boring pain on one side of the head. The pain can be so intense

that the patient becomes nauseated and may vomit (hence the name "sick headache"). Again, the pain starts after the aura ceases. During the headache phase, the patient may be hypersensitive to visual, auditory, and olfactory input. The patient may be in so much pain that he or she is stuporous. The conjunctival blood vessels on the affected side may be engorged. The headache can last from 1 hour to 3 days. The patient usually seeks a quiet, dark room, and he or she may apply cold compresses to the forehead. The patient is able to sleep during the headache, and sleep may ameliorate some of the pain. At one time, preparations of ergot were thought to be the treatment of choice if taken during the aura. In recent times, beta-blocking agents have been used with only partial success. Salicylates are of limited benefit. The pain eventually subsides, and the patient enters the final phase.

Post Headache. At this point, the patient often feels as if he or she has done mortal combat. The patient is lethargic and listless and sometimes undergoes diuresis. This period lasts for a few hours, until the patient can regain normal strength.

Now that classic migraine headaches have been addressed, there are some common variations to the classic presentations that the clinician should be able to recognize.

Ophthalmic Migraine

This variant of migraine occurs in about 10% of migraine sufferers. It is similar to classic migraine during the aura, but, after the aura, there is no headache. Depending on the age and physical status of the patient, this form of migraine could be confused with a TIA, but a TIA does not usually last 20 minutes, and the vision loss is not from the center out. Most other types of acute vision loss are discussed later, but they all last longer than 20 to 30 minutes. Certainly an ophthalmic migraine in someone with a history of the same is not cause for diagnostic concern.

Ophthalmoplegic Migraine

This is a rare but spectacular variant of migraine in which the patient actually experiences the paralysis of extraocular muscles during the aura. Although rarely encountered in clinical practice, these patients deserve some careful neurological evaluation to rule out some potentially devastating diseases. The differential diagnosis includes cavernous sinus thrombosis, leaking aneurysm in the circle of Willis, and less harmful diabetic cranial nerve palsy. An important item to remember is that family members tend to have the same symptoms in their migraine manifestations. Case history is helpful for guiding the urgency with which these patients are evaluated.

Hemianopic Migraine

Here the patient notes that he or she has a hemianopic visual field defect during the aura. It usually does not have the shimmering borders of the classic aura, but, in all other characteristics, it is the same as the classic presentation. The clinician is again encouraged to perform a thorough case history and then proceed to a neurological evaluation the first time that the patient presents with such symptoms.

Hemiplegic Migraine

This variant of migraine is rare, and it usually occurs in young females. They exhibit frank hemiplegia for a period of a few minutes and up to 3 days. This is more a form of paraesthesia than paralysis, and it is associated with an increased Babinski reflex on the affected side. This condition tends to get the "million dollar workup" because of the gravity of the symptoms in a young patient. It can be isolated in later episodes, but, for the primary care provider, the first episode is a condition of great concern and urgency.

Hypertensive Headache

This condition is basically a nonentity. Although much has been made of headache occurring in patients with hypertension, it is not a valuable symptom for the clinician. There is little to no predictive value in any of the headaches that result from hypertension. The headaches that occur in hypertension appear to be due to dilatation of branches of the external carotid artery, because occlusion of the external carotid artery alleviates the pain. However, the attributes of this headache do little to differentiate it from other headaches, and therefore it has little diagnostic value.

Cluster Headache

These headaches are also known as *histamine headaches*. This headache is named for the pattern of presentation. Episodes tend to cluster together over days or weeks with long, irregular intervals between the clusters of headaches. The typical patient is male, in the fifth decade of life, and a "type A" personality. The headache is unilateral in the frontal region with conjunctival engorgement, lacrimation, and nasal congestion on the affected side. The attack can last from 15 minutes to 1 hour, and it may recur several times a day. The pain is similar to migraine in that it is severe, deep, and boring. In recent years, propananol has been used to reduce the frequency and severity of this type of headache. The headache is brought on by dilatation of the internal carotid artery on the affected side. This phenomenon can be simulated by the injection of histamine into the internal carotid artery, but the existence of histamine in the clinical presentation of cluster headache has not been demonstrated.

EYE SIGNS AND SYMPTOMS

Let us now turn to the specific complaints that are reported in clinical eye practice. The list of these complaints is long, but just a few are common. Box 6-1 is a list of complaints that account for the vast majority of reasons for an office visit. Although the entire list will be addressed, the following complaints are the most common reasons for a visit to the ophthalmic office:

- Blur at the near point
- Nonspecific ocular discomfort and fatigue
- Burning or tearing of the eyes
- Blur at far point
- No complaint: routine examination
- Appliance-related visit (i.e., spectacles or contact lenses)

Near-Point Blur

Blur during near-point activities is a more common complaint in the adult population as a consequence of the onset of presbyopia or loss of accommodative amplitude in farsighted persons. Presbyopia is first noticed in patients as intermittent blur at near and a subsequent blur at distance when their view goes from

Box 6-1 Common Ocular Complaints in Ophthalmic Private Practice in Order of Frequency

- Blurred vision at near point
- Nonspecific ocular discomfort and fatigue
- Burning or tearing of eyes
- Blurred vision at far point
- No complaint: request for routine checkup, new frames, etc.
- No complaint: broken or lost lenses or spectacles
- Headache (relation to eyes not specified)
- Headache following use of eyes
- Conjunctivitis or blepharitis (crusting and flaking)
- Twitching of lids, itching of eyes
- Photophobia
- Ocular pain
- Loss of vision (uniocular, binocular, and scotomas)
- Exophthalmos (uniocular and binocular)
- Diplopia
- Anisocoria
- Photopsia and halos
- Strabismus
- Jumping of words and other difficulties when reading
- Disturbance of color vision
- Vertigo
- Foreign body in eye

near to distant. The blur at both distances is fleeting in that it clears within seconds of the shift in gaze. These symptoms are usually noted at about 40 years of age, when the patient still has sufficient accommodative reserve to read without a reading addition at near and when he or she is probably due for a decrease in accommodative facility. During the subsequent 5 years, the patient begins to notice that, the further away from the body that he or she holds the material, the clearer the material is. The classic complaint of an early presbyopic patient is that his or her "arms are getting too short." In these cases, it is best to refer the patient to an orthopedic surgeon or to prescribe a first pair of multifocals.

When a complaint of blur at near is the principal reason for the patient visit in a child or adolescent, the clinician should strongly suspect binocular rather than refractive problems. The patient could have refractive problems (e.g., high hyperopia, astigmatism), but, in terms of incidence and the patient's age, these conditions are certainly less common reasons for presentation. In these patients, the clinician should be keenly aware of any symptom that might be related to ocular discomfort or fatigue during the case history. Questioning the patient about the type of blur may be particularly fruitful in this situation. The patient with binocular motor problems may confuse diplopia with blur during the case history. The clinician can differentiate between the blur and diplopia by having the patient describe exactly what is seen when working at near. When reading, the patient may have trouble finding his or her place when going from the end of one line to the beginning of another, or he or she may notice that the letters begin to split apart when reading for longer time periods. Asthenopia or headache is a frequent companions to these motor problems, particularly on school days and, more particularly, toward the end of the day.

Nonspecific Ocular Discomfort and Fatigue (Asthenopia)

Asthenopia is pain, discomfort, or fatigue in or around the eyes. The causes of asthenopia are refractive error, motor anomalies, and integrative problems. A combinational etiology of asthenopia is probable, in which accommodation and convergence are both at play in the development of these symptoms. Whenever there is imbalance between the eyes (e.g., anisometropia, aniseikonia, high phoria), eyestrain is highly likely. The clinician should document the nature of the complaint as thoroughly as possible with the patient in an effort to isolate the underlying etiology as nearly as possible. A child with near blur must be questioned about accompanying signs and symptoms so that causal hypotheses may be formed.

Small to moderate refractive errors cause most of the symptoms in patients complaining of asthenopia. With

large refractive errors in which the patient cannot compensate, he or she usually resorts to monocular or learns to tolerate the resultant reduced visual acuity. Questions related to the symptoms of small to moderate refractive errors then become appropriate. Does the patient have blurred vision (when, where, and how severe)? What is the patient's age? Are there times when the blur is worse, or is it constant? Is the patient having trouble at a particular distance (usually near point)? During reading, does the patient lose his or her place when going from the end of one line to the beginning of the next line? Do words seem to blur or double with prolonged reading? Do the symptoms occur later in the work period? Do the symptoms only occur on work or school days? Has the teacher noticed a reluctance on the patient's part to do certain activities? All of these lines of questioning lead the clinician to a better understanding of the type of refractive error involved.

Burning and Tearing of Eyes

Burning and tearing are frequent complaints in the elderly population; they are most often related to dry eye in this age group. In a younger population, they are most commonly a complaint that accompanies seasonal allergy. Burning and tearing can also be the first symptoms of acute bacterial conjunctivitis. The way to differentiate between the many causes of burning and tearing is to ask about the circumstances that surround the complaint. In what conditions does the patient notice the symptoms? An older, dry-eyed patient may notice the tearing more in the winter, when out of doors on a windy day, when using the air conditioner in the car, or when in the presence of other drafts that desiccate the cornea. The seasonal allergy sufferer usually can tie the symptoms to a particular time of year, usually spring or late summer. The conjunctivitis patient notes that this is a new symptom or, even if this is a second or third episode, that the complaint is not seasonal or periodic.

During the fifth and sixth decades of life and after, dry eye becomes a significant problem for patients. They need reassurance that the clinician understands their problems, because the tearing and dry eye do not seem congruous. The tearing is a reflex tearing in response to irritation of the cornea. The problem is that these tears are aqueous, and they are deficient of the mucin and oils needed for proper tear-film mechanics. Many of these patients note that the tearing increases during reading. This may be due to decreased blinking from concentration on the reading material, which is a common result of near work. The dry eye is, therefore, intensified while reading, and increased reflex lacrimation may result.

A related syndrome is pain, burning, and tearing upon awakening. This can occur at any age and affects

males and females equally. It is often associated with a condition in which the lids do not fully close during sleep, called *lagophthalmos*. Because of Bell's phenomenon, the lower portion of the cornea dries, and there is a semilunar area of desiccation at the limbus and across the exposed inferior cornea and conjunctiva. The patient awakens and is fine until he or she opens the eyes. When the eyes open, pain, burning, and tearing occur. This is almost always more severe in one eye, but both eyes are generally involved. In more extreme cases, recurrent corneal erosion may become a part of the syndrome. Patients sleeping under ceiling fans or where there are nocturnal drafts are more prone to this condition. In temperate climates, it is also worse in the winter, when the humidity is lower inside the home.

Blurred Vision at the Far Point

This complaint is most commonly associated with myopia, although it may occur in decompensated phorias in older adults, certain cranial nerve palsies, some oculomotor imbalances, and high astigmatic refractive errors. "Blur" reported by the patient can be the result of lateral binocular diplopia and monocular diplopia, in addition to refractive or accommodative causes. The vast majority of patients with blur at distance are uncorrected or undercorrected myopes. The classic complaint in the child is the inability to read what is written on the board at the front of the room. The child may have a history of being moved to the front of the class to compensate for the blurred vision.

In some elderly patients, vertical phorias become tropias with certain conditions. Because the vertical phoria is usually smaller in magnitude than are horizontal phorias, the complaint may be one of blur at distance rather than frank diplopia. The classic complaint of this type of patient is that they see blurred taillights on the cars in front of them. The complaint is actually diplopia, but the magnitude is so small that the taillights look blurred rather than doubled. The reason taillights have this appearance and headlights do not is that, at night, peripheral cues for binocular lock are significantly reduced. Oncoming headlights produce more binocular lock cues for the driver, and, therefore, the tendency is to notice problems when viewing taillights. It is believed that these patients are the same patients who break into tropic responses when "fatigue ductions" are performed on them during their earlier years.

Diabetes mellitus is a common cause of intermittent blur that should not be ignored. These patients usually complain of blurred vision that lasts for a day or so. When questioned further, they can link the blurred vision to an increase in blood sugar levels. Although this is a complaint that is usually elicited from an established diabetic, it is possible that the ophthalmic prac-

titioner will be the first to suspect diabetes in such a patient. The refractive error shift is usually in the minus direction, and it may have an astigmatic component. The refractive shifts are generally binocular and of approximately the same degree in each eye, but they are occasionally unocular or more pronounced in one eye.

No Complaint: Request for Routine Check-Up or New Frames

The patient who presents with this complaint seems to be the easiest to serve. There is no real complaint, but he or she wants an eye examination. The clinician should be on guard and ask himself or herself if this the true situation or whether the patient is stoic and hiding some underlying reason for the visit; it is the responsibility of the person taking the history to be alert for this possibility. Often something is bothering the patient, but it may not be revealed during the initial questioning. The patient may not be consciously obscuring the reason for the visit, but, with adequate questioning, the reason for the visit comes to the fore. Perhaps the patient wants to see if the examination reveals an eye problem without prior notification of his or her problem. Unfortunately, some patients enjoy testing the physician while at the same time making their care harder to deliver. Alternatively, the patient actually may not have an underlying complaint, and a baseline examination and updated spectacle prescription may serve this patient well.

No Complaint: Broken or Lost Lenses or Spectacles

This is usually a prior patient of the clinician's, but not always. In either case, the taking of the history is requisite, because the clinician must know the circumstances surrounding the dispensing of the last pair of spectacles and when they were dispensed. With a new patient, a full history is appropriate. For an established patient, an update suffices. These patients—as opposed to those who are coming in for a new pair of glasses—generally do not have other underlying symptoms that are causing the visit, but the physician cannot assume that to be the case.

Headache Not Related to the Use of Eyes

This topic has been thoroughly covered in the previous Headache section.

Headache Following Use of the Eyes

The most common locations of headaches associated with use of the eyes are frontal and occipital. Brow aches and frontal headaches are most often ascribed to refrac-

tive problems or convergence excess. The causes of occipital headaches are not as clearly defined, with convergence insufficiency and vertical imbalance being the primary causes. However, refractive deviations and presbyopia have been noted to cause these headaches. Furthermore, it is sometimes difficult to determine if the occipital headache is actually visual in nature as opposed to being related to stress. The third location of which the clinician should be aware is the temporal area, where uncorrected oblique astigmatism is the primary cause. Although uncommon, this is a clinical pearl worth remembering.

With vision-related headaches, the patient usually notices that the pain begins after reading or use of the eyes and that it is preceded by eyestrain. At times, the headache comes on toward the end of a work period, much like the classic tension headache. In fact, tension-like headache can be induced by the introduction of loose prisms in front of one eye for a period of 15 minutes. The character of the ocular-induced headache is usually dull, steady pain. Severe pain or other associated symptoms should lead the clinician to seek another etiology for the headache. The pain can wax and wane with use of the visual system. Resting the visual system generally relieves the pain. If the underlying etiology is the binocular system, patching one eye will relieve the symptoms.

Conjunctivitis and Blepharitis (Crusting and Flaking)

Patients rarely complain of conjunctivitis or blepharitis, but they do complain of crusting and flaking of the eyelashes. Sometimes this is normal drying of ocular secretions in the inner canthus overnight, but at times it may be the harbinger of impending, full-blown, acute conjunctivitis. The best way to differentiate between the two is a good inspection of the conjunctiva. The most common problem involving crusting and flaking that an ophthalmic practitioner sees is chronic blepharitis. Patients are sometimes oblivious or resigned to the condition and do not mention it during the case history, but it is during that time that the observant clinician first notices it. The base of the lashes have collarettes, and the lid margins are reddened. These patients have usually had the problem since childhood, with the condition varying in severity. This disease is caused by chronic staphylococcal infection, and it is best treated with lid scrubs. Left untreated, this condition can progress to marginal corneal ulcers and ulcerative blepharitis.

The other form of blepharitis that is known as squamous blepharitis is not as easily noted, but it is easier to treat. Also known as seborrheic blepharitis, this condition presents with flakes (scurf) on the eyelashes, mild erythema of the lid margin, flakes in the eyebrow,

and seborrheic flakes in the hair of the scalp. Treatment for this malady is regular use of an antidandruff shampoo.

Twitching of the Eyelids and Itching of the Eyes

Myokymia, or twitching of the eyelid, is a common complaint for which there is no known remedy. It is thought to be related to psychological stress, but that is only a hypothetical cause. It is usually in the lower lid and is not visible to others, even though the patient is sure that it is apparent. Reassurance is the order of the day.

If the cause of twitching is obscure, itching is almost pathognomonic of allergy. The only exception is the itching that is present in herpes simplex lesions before they vesiculate and rupture. The itch of allergy is often accompanied by a stringy,ropy discharge; a burning sensation; and a red conjunctiva. Depending on the stage of the disease process, it can be treated successfully with mast-cell inhibitors or antihistamines.

Photophobia

Sensitivity to light is the hallmark of acute anterior uveitis. It can be seen in cases of keratoconjunctivitis and conjunctivitis as well as with corneal abrasions. The pain seems to be related to contraction of the iris sphincter and ciliary body. When a patient complains of sensitivity to light, the astute clinician immediately starts to look for the cause; the differential diagnosis includes hyperacute bacterial keratoconjunctivitis, herpetic keratoconjunctivitis, significant corneal abrasion, trapped foreign body, and iridocyclitis.

Some persons claim photophobia as a chronic condition and subsequently want to wear sunglasses continually. Many believe that this is an attempt to mask drug abuse by wearing dark glasses so that health care practitioners and others cannot judge pupil size or reactivity during casual contact. One way to ascertain whether the perpetual daylight use of sunglasses is necessary is with the direct ophthalmoscope test. The test is performed during ophthalmoscopy with a halogen direct ophthalmoscope and a patient with an undilated pupil. If the patient does not lacrimate, the patient probably does not need sunglasses in a normal environment.

Ocular Pain

Superficial pain of the eye can be the result of trauma or inflammation of the tissues of the corneal epithelium, conjunctiva, or episclera. Corneal abrasion, retained foreign body, and lid concretions are common traumatic causes of ocular pain. The pain with trauma is usually proportional to the extent of trauma. It can vary from a sandy, gritty feeling to frank, severe pain. Inflammatory causes of pain are many and, here again,

the amount of pain is usually consistent with the degree of inflammation. Herpetic keratitis is an exception to this rule because of the decreased corneal sensitivity associated with this viral infection. Episcleritis and scleritis can produce anything from a burning pain to a deep boring pain; again, the amount of pain parallels the disease process. One of the more common and intense ocular pains is that induced by the trapping of a foreign body under a rigid contact lens on the eye.

Frank pain in the globe is an uncommon complaint. One of the most commonly cited types of ocular pain in textbooks is that found on rotation of the globe, which is associated with retrobulbar optic neuritis. Although it is true that this is a complaint in patients with acute optic neuritis and therefore multiple sclerosis (MS), it is not commonly encountered in practice. Furthermore, it is unusual for that particular complaint to be the chief complaint in a patient with MS; it is much more likely that the patient will complain of vision loss associated with heat (Uhthoff's sign) and other neurological deficits. It is probably the critical nature of the diagnosis that leads authors to give such attention to the symptom. Retrobulbar pain is also associated with tension headache. However, tension headache involves no pain on rotation or loss of vision.

Many patients complain of a sharp, stabbing pain in the eye that can stun the patient. The pain comes on unexpectedly and suddenly. It is short in duration, lasting for 1 to 2 seconds. This pain does not recur frequently, but it does recur. Most patients can remember the episodes with vivid detail, stating where they were and what they were doing at the time of attack. The cause is unknown, and there is no known treatment. Fortunately, the pain is fleeting, and the episodes leave no sequelae.

Acute narrow-angle glaucoma is another source of deep ocular pain. When present, the pain is excruciating and debilitating, and it can produce nausea and vomiting. The pain subsides as the attack is broken. Pain may be absent in patients with longstanding glaucoma or in those with absolute glaucoma, even in the face of significant intraocular pressure rises. The clinician should be aware that a carotid aneurysm may produce similar unilateral pain to that found in acute glaucoma. The differential here is the lack of redness and normal intraocular pressure in the carotid aneurysm.

Intraocular inflammation can cause pain on accommodation in cases of anterior uveitis. This pain is dull, and it is similar to the photophobic pain experienced by these patients. This pain may also be described as an ache. The pain decreases with the withdrawal of the near-point stimulus. Many patients with iritis who do not manifest photophobia do have pain on accommodation; the mechanism of both pain with reading and pain with bright light is probably the miosis induced by both stimuli.

Posterior uveitis, endophthalmitis, orbital disease, and traction or displacement of extraocular muscles are other causes of deep ocular pain. When the physician is faced with a patient with unexplained orbital pain, some clinical pearls help in the differential diagnosis. The pain of uveitis is usually worse at night; the pain of diabetic neuropathy is followed by signs of ophthalmoplegia. In Tolosa-Hunt syndrome, the pain is accompanied by involvement of the third, fourth, and sixth cranial nerves and diminished corneal sensitivity. Ocular pain can be present in temporal arteritis. In this condition, an elevated sedimentation rate, claudication on mastication, and a prominent temporal artery on the affected side help to differentiate it from other causes of deep ocular pain.

Pain associated with the trigeminal nerve is common in the ophthalmic practice setting, particularly in the geriatric setting. Trigeminal neuralgia is common in females during the sixth decade of life. It is characterized by paroxysms of severe pain in the distribution of one of the branches of the trigeminal nerve that are of sudden onset and brief duration. There is often a "trigger" area, which, upon stimulation, produces a paroxysm. There is a reflex spasm of the facial muscles in response to the pain from which its alternative name, *tic douloureux*, was derived. Herpes zoster can produce trigeminal pain as part of its clinical presentation. The pain is preceded by a vesicular eruption in the distribution of one of the branches of the trigeminal nerve; these vesicles rupture to form multiple ulcers. The pain occurs at approximately the same time that the vesicles rupture, but it is not thought to be related to the open lesions on the skin. This pain can persist for months to years, particularly in the elderly. In fact, there seems to be a positive correlation between the patient's age and the duration of the postneuralgia pain. All elderly patients with herpes zoster should undergo workup by an internist to rule out coexisting cancer, because this disease is often seen in patients with depressed immune systems.

Loss of Vision (Unicocular, Binocular, and Scotomas)

When one is considering a real estate purchase, the first three criteria to study are location, location, and location. When a clinician is faced with a complaint of loss of vision, the first three characteristics that he or she should determine about the loss of vision are duration, duration, and duration. Table 6-1 lists most of the major conditions that produce loss of vision, along with the duration of loss.

The preceding paragraph was not in jest: duration is the hallmark symptom of these conditions. This is not to say that other symptoms are not important in the differential diagnosis of loss of vision, but duration is the

TABLE 6-1 Causes of Loss of Vision and Duration of Symptom

Cause of Vision Loss	Duration of Scotoma
Transient ischemic attacks	Few seconds
Migraine headache	20 minutes
Multiple sclerosis	Hours to days
Retinal detachment	Until repaired or permanent
Tumor	Permanent
Nonarteritic ischemic optic neuropathy	Months to permanent
Central retinal vein occlusion	Months with residual loss
Central retinal artery occlusion	Permanent
Cerebral vascular accident	Weeks to permanent

most important. Those other symptoms and how they help with the making of the tentative diagnosis will now be addressed.

Transient ischemic attacks are a common complaint among males who are 50 years old and older. It is a complaint that is frequently missed unless the clinician is looking for the appropriate history. TIAs are manifested almost exclusively as symptoms, which are often visual and fleeting. The classic sign is a graying out of vision, which then returns to normal in 5 to 10 seconds. Depending on where the blockage is in the central nervous system, the nature of the scotoma varies. The scotomas are usually bilateral, and they vary from complete anopsia to a vague complaint that one side of the vision was blurry. The pathophysiology of TIA is probably similar to that of a Hollenhorst plaque in the retinal circulation in that a small cholesterol plaque breaks off of the wall of an artery and temporarily lodges further downstream. This blockage results in ischemia of the nervous tissue and a temporary loss of function. What differentiates TIAs from cerebrovascular accidents (CVAs) or strokes is that TIAs are transient, as the name implies.

Transient ischemic attacks begin as an isolated event, but, over time, they may become frequent. If the frequency or duration is in a crescendo pattern, it is cause for concern and immediate referral to a vascular surgeon or an internist to determine the origin of the emboli and to initiate treatment. An isolated event or a single TIA every 3 to 4 months is not cause for such concern, but it should be a reason for referral nonetheless.

Migraine has been discussed previously. However, just to reiterate, the aura of migraine rarely lasts for more than 30 minutes and is classically reported as being 20 minutes in duration.

Vision loss attributable to MS is a central scotoma during the summer, after a hot shower, or after intense physical exertion. As noted earlier, this is known as Uhthoff's sign, and it is characteristic of vision loss in patients with MS. This vision loss usually lasts 1 to 2 days, and then vision is spontaneously recovered. This is not the same as the vision loss from retrobulbar optic neuritis, which is classically described as "the patient sees nothing and the physician sees nothing," because there is vision loss and an absence of signs on the optic disk. Here, vision loss can be extensive and last for weeks. Smith⁸ describes the fleeting loss of visual acuity as a sign of MS in 20- to 40-year-old women. Here the patient reports that in one instant he or she can read the 20/20 line and in the next he or she can only read 20/80. Again, the vision may be normal 2 minutes later. This is not a TIA, but rather it is probably related to impaired function of the optic nerve secondary to MS. The fleeting vision loss is subtle and difficult to document unless it occurs in the examination chair.

Retinal detachment, tumor, ischemic optic neuropathy, central retinal artery occlusion (CRAO), central retinal vein occlusion, and CVA all have differing presentations, but they share a common characteristic: the vision loss is longstanding or permanent. Retinal detachment is often perceived as a curtain falling over the visual field. Tumor produces a slow, progressive loss of vision. Ischemic optic neuropathy is a rapid-onset, usually altitudinal, visual-field defect that is associated with sectoral optic nerve head swelling. CRAO manifests itself as a sudden and complete loss of vision in the affected eye. Sometimes there is an island of vision in the centrocecal area, which is due to the presence of a patent cilioretinal artery. The fundus appearance is that of an ischemic retina (pale) and a cherry-red macula. The vision loss in central retinal vein occlusion is not as sudden as that of CRAO, but it is a vision loss that progresses over 30 to 120 minutes, with the end result being very reduced vision in the affected eye. CVA, or stroke, is a rapid visual loss that is usually hemianopic and bilateral. Often the patient reports that the vision in the eye on the side of the visual field loss is the problem, without realizing that the field loss is bilateral. This field loss, if present at the time of examination by an eye care practitioner, is permanent. This qualification has been made because of a recent change in the treatment of CVAs. It is possible that, with the treatment of strokes with "clot-busting" agents during the early hours of the stroke, the visual field loss will not be a permanent feature of this condition.

Exophthalmos (Unicocular or Binocular)

Bilateral exophthalmos is by far the most common form of this condition seen in ophthalmic practice. The most common cause of binocular exophthalmos is Graves'

disease from hyperthyroidism. Although the presentation is bilateral, it is not uncommon for the degree of proptosis to differ between the two eyes. The clinician is reminded to be on guard for this sometimes subtle presentation, because it can be vision threatening if unrecognized and untreated. The vision loss is caused by a compression neuropathy. The exophthalmos is produced by swelling of the extraocular muscles and retrobulbar fat. If there is any indication that the patient may have Graves' disease, it is imperative that the patient undergo accurate testing for visual acuities, contrast sensitivity, and threshold visual fields and B-scan ultrasonography of the orbit (if available); the patient should be referred back to his or her primary care physician for a thyroid workup and scheduled for close ophthalmic evaluation. A patient who had undergone thyroid ablation approximately 30 years previously was taking a levothyroxine thyroid hormone-replacement drug and developed Graves' disease with exophthalmos, marked conjunctival hyperemia, and vision loss. Because the patient had had bilateral exophthalmos and had been taking synthetic thyroid replacement for years, this finding was missed by the physician who was treating the patient, thus resulting in more visual loss. The condition (exophthalmos) does *not* totally depend on the state of the thyroid gland function.

The most common cause of unilateral exophthalmos is also Graves' disease. The unilateral presentation is usually binocular exophthalmos, with one eye preceding the other. Although the proptosis may be caused by thyrotoxicosis, the clinician must be vigilant about other causes. Space-occupying lesions of the orbit are common progenitors of this condition. If the globe is displaced along the X or Y axis, it is as a result of a space-occupying lesion of the orbit that is located outside of the muscle cone. Diplopia is a common accompanying complaint with mass lesions of the orbit. Computed axial tomography of the head with orbital emphasis is mandatory for patients with a suspected mass lesion of the orbit. Pulsatile proptosis is a sign of arteriovenous aneurysm of the orbit. Auscultation of the orbit or globe may yield a bruit. These patients often hear rushing water sounds in their heads. Fortunately, this is a rare anomaly in practice, but it is one worth mentioning.

Diplopia

Double vision of recent onset is cause for concern for the clinician, because diplopia can be the harbinger of tragedy. Central nervous system vascular anomalies, aneurysm, and stroke are possible etiologies for sudden-onset diplopia. The first task is to make sure that the patient is actually describing diplopia. It is not uncommon for someone with significant blur to report double vision. Once satisfied that the complaint is valid, the clinician should determine the exact nature of the com-

plaint. The diplopia should be immediately categorized as being binocular or monocular.

In terms of duration, a fresh complaint of double vision without prior episodes is more critical than a longstanding problem. As noted earlier, in the adult population, the most likely cause of a sudden onset of binocular diplopia is vascular. It is possible that an intracranial tumor could cause diplopia, but this is not as likely. In the case of the intracranial tumor, the onset is gradual rather than sudden. Neuropathy is another cause of the sudden onset of diplopia; diabetes mellitus is the leading cause of these lesions. In patients with diabetes, the following nerves are affected in order of frequency: VI, III, and IV. One sign that differentiates a third-nerve palsy caused by diabetes from one caused by a leaking aneurysm in the circle of Willis is that the pupil is generally spared in a diabetic presentation. Usually a diabetic patient knows that he or she has the disease, which will aid the physician with the differential diagnosis. Furthermore, the diabetic may have had the condition before. If so, the nerve palsy associated with diabetes usually resolves in approximately 6 to 12 weeks.

A muscle paresis or palsy is noncommitant when it first develops, and it produces varying degrees of diplopia in different fields of gaze. The greatest deviation between the lines of sight occur in the field of gaze of the affected muscle. Often the patient has already noticed this, and he or she can offer details about the field of gaze in which it is most apparent.

If the diplopia disappears when covering either eye, the clinician can be sure that the patient has "binocular" diplopia, which is produced by a misalignment of the lines of sight. When the patient covers one eye and the diplopia remains, the clinician must assume that there is a monocular cause for the diplopia. Monocular diplopia is generally caused by the ocular elements of the eye. High astigmatism and changes in the crystalline lens are the two most common causes. In rare cases, wrinkling of the internal limiting membrane of the retina can cause monocular diplopia.

Vertical muscle imbalances are common causes of diplopia. Images may be split diagonally, but it is the vertical imbalance that often leads to the diplopia. This can occur at any age, but persons with high hyperphoria who are compensated may report diplopia when driving at night and in other situations in which fusional cues are minimal. Ophthalmic practitioners will remember that the range of clear single binocular vision is restricted in the vertical plane as compared with the horizontal plane. It does not take a large imbalance to overcome what fusional reserves exist in the vertical plane. After fusion is broken, any horizontal imbalance is also manifested, thereby making pure vertical diplopia rare; however, the root cause is the vertical phoria decompensation.

Anisocoria

Anisocoria is not a common complaint, but it does exist. In the young, healthy patient, it is probable that the difference in size between the two pupils is physiological. If the condition is longstanding, it is also probable that the condition is within normal limits. Recent-onset anisocoria with a relative difference in pupil size in light and dark settings indicates the need to search for a central nervous system cause.

Photopsia and Halos

Photopsia is a common complaint in the aura of migraine, as has already been discussed. Flashes of light are common in the area of retinal tears, and these are considered to be indicators of impending retinal detachments. Quick eye movements can cause flashes of light (Moore's lightning streaks), which are caused by pressure phosphenes and are of no clinical significance. Nonarteritic ischemic optic neuropathy has been known to present with bright purple light centrally in the visual field and preceding frank vision loss. Some patients with dry, age-related macular degeneration complain of colored light patches in their central vision; these patients sometimes also complain of a spinning propeller image at fixation.

Halos are produced by the optical elements of the eye. They can be physiological because of the fibers of the crystalline lens. The physiological halos are smaller and usually yellow, whereas the pathological halos are larger. The most commonly sited pathological halo is that produced by acute, narrow-angle glaucoma. In clinical practice, patients rarely complain of halos around lights when they have this condition; it does make sense that the high pressure in the eye leads to corneal edema, but it is not a good presenting symptom for differential diagnosis. These patients have more pressing and obvious symptoms to present to the clinician, such as pain, redness, nausea, and vomiting.

Halos are often reported by patients wearing contact lenses. In conditions of high contrast, such as when driving at night, the limiting optical zones and edges of the rigid contact lens can create noticeable flare and glare. Under very hypoxic conditions, such as after overnight wear of rigid lenses and when the lenses have been coated or filmed by deposition, halos are seen at night around bright lights, stars, the moon, and so on. Also, when soft contact lenses are coated or filmed with deposition, halos can be seen in high-contrast situations, such as when watching a movie in a theater. Soft contact lenses induce more hypoxia than do rigid contact lenses, so that halos around lighted objects are also seen by patients, especially under high-contrast conditions. The visual optics of contact lens wear is further discussed in Chapter 26.

Strabismus

Strabismus is common in ophthalmic practice, and it can be coupled with another complaint, which is lazy eye (amblyopia). Strabismus may be hereditary or acquired. The hereditary variant may be manifested very early in life, or it may become manifested later in life. The best way to differentiate hereditary from acquired strabismus is through a thorough case history of the complaint. Hereditary strabismus patients have a family ocular history that is consistent with the patient's condition. Acquired strabismus is usually associated with neuropathological processes that tend to occur later in life and that tend to have a sudden onset.

If strabismus develops during early childhood, there is usually an accompanying amblyopia. Suppression is thought to be the mechanism for the development of the amblyopia. The bottom line is that patients with strabismus amblyopia do not report diplopia. The characteristics and management of strabismus are discussed in detail in Chapter 31.

Jumping of Words and Other Difficulties When Reading

Patients who complain of words jumping or losing their place when shifting from the end of one line to the beginning of the next have been discussed. This type of problem usually occurs in young children and young adults, and it most likely represents binocular vision anomalies. Difficulty when reading can be caused by numerous conditions, including the following:

- Binocular vision problems
- Refractive error
- Presbyopia

Disturbances of Color Vision

The layman's "color blindness" is well known to be hereditary color-vision deficiencies that are sex-linked and that are predominately found in males. More severe forms of color-vision deficiencies may truly be color "blind," but these are exceedingly rare in the population, and they are usually well documented in the patient's medical history. All hereditary forms of color-vision defects are bilateral, and the severity is usually equal in the two eyes. The case history offered by the patient is often that he or she has been told that he or she has a color-vision defect, but the patient does not believe that he or she is different than persons with normal color vision. To hear such patients tell it, the tests that detect color-vision abnormalities are just too sensitive. Others with greater defects can relate stories of wearing clothes that clash, socks that do not match, and the like. They tell of confusing green and brown and of having trouble with traffic control signals, particularly if the orientation of the light is not what they are

used to seeing. Although this is an important piece of information, it does not have dire consequences for the patient from a health standpoint. The clinician should provide vocational guidance to young patients and patient education to all age groups about available coping mechanisms.

Acquired color-vision deficiencies, on the other hand, are associated with significant health issues. The clinician should be aware that a complaint of a washing out of colors or a change in the perception of colors is cause for a more thorough neurological examination. The cause for changes in color vision is related to changes in the retinal receptors or disease processes that are more medial to the receptors. Neurological disease should be the first thing that the practitioner considers, but these changes could also be caused by local retinal changes or even pharmaceutical toxicities or reactions. Drug toxicities are more likely to be bilateral, whereas the neurological and local retinal problems are mostly monocular in their presentation.

Vertigo

Dizziness or vertigo is a complaint that is usually not ocular in origin, but it is sometimes heard by the ophthalmic case historian. The most common cause of dizziness is related to the inner ear, and it requires a referral to otolaryngologist (ear, nose, and throat specialist). Accompanying symptoms usually differentiate inner-ear inflammation from other forms of vertigo. The other common locus for vertigo is the posterior fossa. Neurological disease or vascular disease in this area of the cranium often leads to a complaint of vertigo. Again, as with the inner-ear problem, accompanying signs and symptoms often help in the differential diagnosis.

Foreign Body in Eye

Foreign-body sensation is a relatively common complaint during examination. When it is an acute symptom and the patient can recount a situation in which something got in the eye, the clinician should suspect that it is indeed a foreign body. Inspection usually supports that diagnosis.

However, a few other conditions may cause foreign-body sensation without the presence of a foreign body. Recurrent corneal erosion is a condition that follows a large corneal abrasion. In this case, the healing process commences, but, for some reason, it does not progress to completion. Adhesions are thought to form between the lid and cornea, and, when the patient arises after a long period of sleep, the adhesion produces a recurrent corneal erosion upon opening the eyelids.

Exposure keratitis can cause many of the same symptoms without the history of foreign body or an antecedent corneal abrasion. This condition is usually

caused by lid lag (lagophthalmos) during sleep. The patient will have been told by others that his or her eyes are open during sleep. The mechanism here is ocular surface desiccation. The epithelium in the area of desiccation may have regions of superficial punctate keratitis, or it may actually abrade upon arising and changing lid position.

Map-dot-fingerprint dystrophy (Cogan's microcystic dystrophy) also produces a type of foreign-body sensation, which may or may not be present upon arising in the morning. The pain is thought to be caused by the rupture of microcysts in the corneal epithelium and a resultant small corneal erosion. The classic signs of map-dot-fingerprint dystrophy should be present for the clinician to be able to differentiate this from other causes of foreign-body sensation.

Corneal abrasion from any cause may give rise to foreign-body sensation in the eye. If a foreign body was present, produced a corneal abrasion, and then was flushed from the eye by lacrimation, the pain persists for hours. The pain usually radiates to the lateral aspect of the upper lid in the affected eye. If the pain persists for longer than 24 hours, a frank foreign body must be suspected.

Floatters

Vitreous floaters are common in the population. The sources are many and diverse. Many persons have floaters from birth due to incomplete absorption of the hyaloid artery. The phenomenon of floaters is not what it appears to be to the patient. The patient complains of something floating in space that appears to move with eye movement and then slowly settles to its original position. In fact, what the patients are seeing are shadows of opaque objects in the vitreous fluid. The closer the object is to the retina, the darker and more clear the outline of the shadow. If the physician harkens back to the geometrical optics discussion of umbra and penumbra, the optics will become clear.⁹

Often children think that a floater is dust on the cornea and do not realize the exact nature of the problem. However, by the time that they reach adulthood, patients just describe the phenomenon as floaters or "something in my vision." It is up to the practitioner to find out why they have floaters. The most significant question to be asked in this case is, "How long have you noticed these things floating in your vision?" If the answer is "For as long as I can remember," the problem is chronic and not something that is urgent. In the adult population, asteroid hyalosis is a common clinical finding. It seems to occur during the fifth decade of life, and it can be visually debilitating. In the past, patients with asteroid hyalosis were said to not notice the floaters from the condition. This is not true, but often

the patient's symptoms do not match the amount of material noted on ophthalmoscopic examination. This may be the result of more calcium soaps being present in the anterior vitreous than in the posterior portion. Again, most of the patients with asteroid hyalosis do have complaints of floaters if they are pressed by the case historian during the interview.

New floaters, on the other hand, represent a problem that must be investigated with diligence. The most common causes, in order of frequency, are as follows:

- Posterior vitreous detachment (PVD): the separation of the vitreous face from its attachment to the retina at the optic nerve head
- Vitreous detachment: the separation of the vitreous base from its attachment to the retina at the ora serrata, with or without round hole formation
- Retinal detachment (RD): frank detachment of the retina
- Vitreous hemorrhage: hemorrhage into the vitreous cavity, for any reason

PVD has classic symptoms. The patient describes a spider web or a large circular object, which is usually temporal to the line of sight. This object may have been larger when it first suddenly appeared, depending on the time since onset and the patient's powers of observation. Flashes may have preceded the actual vitreous detachment, but these rarely persist after detachment. The shadow is cast by the vitreous face, which is now above the retina and which is wrinkled so that the folds cast more of a shadow on the retina. Over time, the symptoms subside, but they never completely disappear, and the patient can describe the PVD's current nature to the clinician in great detail many months after they occur. Again, the greater the distance from the object to the retinal surface, the less well the shadow is formed and the less it is apparent to the patient. Because the vitreous face is drawn away from the retina over time, the patient notices the PVD less and less. Except for trauma-induced PVDs, the age of onset is during the fifth decade of life and beyond.

Vitreous detachment at the ora serrata is fairly common in patients during the fifth decade of life and beyond. It occurs in the same manner as the PVD, except the area of vitreous condensation is smaller and is not as often noticed by the patient. The symptom that should place the clinician on guard is a floater that is peripheral and that is preceded by flashes in the same peripheral region. These vitreous detachments must be thoroughly investigated with the binocular indirect ophthalmoscope and scleral depression. There is a high incidence of operculated tears and round holes of the retina, which can lead to retinal detachments with this type of vitreous detachment. If a blood vessel is in the area of these holes or tears, hemorrhage into the vitreous can also occur, leading to a shower of floaters in the

region of the vitreous detachment. With the round hole of the retina, a tuft of retina is in the vitreous, which leads to a floater that is usually evident to the patient.

Retinal detachment is known to be preceded by flashes and floaters; these have been described. The added feature with retinal detachments is a "curtain" coming down over the patient's vision; this is the hallmark of a RD. The clinician should be acutely aware of the potential harm that can result to the retinal integrity when the patient complains of flashes or new floaters; he or she should investigate these symptoms to prevent a more dramatic and vision-threatening RD.

Vitreous hemorrhage is the last of the acute-onset origins of vitreous floaters. This condition can produce anything from a small floater to a total obscuring of vision; the symptoms depend on the severity of the hemorrhage and its location. Vitreous hemorrhage can occur secondary to a vitreous detachment, retinal vascular disease, trauma, or other causes; the age of onset is linked to the underlying cause of the hemorrhage. With the advent of laser panretinal photocoagulation therapy for proliferative diabetic retinopathy, the incidence of vitreous hemorrhage has diminished significantly.

SUMMARY

This journey has been a short one, but no chapter about this subject can cover all of the possible patient complaints and their origins. This chapter began with a rationale for eliciting a thorough case history and proceeded to discuss the structure that a clinician would follow to build a rational history of the patient and his or her visual and medical problems. The discussion then used a list of common visual complaints to illustrate the type of knowledge base that the clinician must bring to the table when rendering health care in the managed-care scenario. It is hoped that the reader has accumulated some clinical pearls and enhanced his or her skills while studying this chapter and that, when this chapter is combined with all of the others, new and exciting clinical techniques will be available to the vision care practitioner.

The single most important fact that a clinician can take away from this chapter is that, with adequate time and care given to the process of interviewing the patient, the clinician will be well guided in the physical examination of the patient and in the accurate and timely resolution of the patient's problems.

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7

Visual Acuity

Ian L. Bailey

Visual acuity is the spatial resolving capacity of the visual system. It expresses the angular size of detail that can just be resolved by the observer. The limits to visual acuity are imposed by optical and neural factors or their combination. In the normal eye, the limitations imposed by optical factors and neural factors are of similar magnitude.¹

OPTICAL LIMITATIONS

When the eye is in ideal focus, a point object is imaged on the retina not as a point but as a small circular patch with faint surrounding rings; this is the *diffraction pattern*. The central circular patch is called the Airy disk, and it has an angular size of $\omega = 2.44 \lambda/p$ (where the diameter ω is expressed in radians, λ is the wavelength of light, and p is the pupil diameter). The smaller the pupil is, the larger is the Airy disk. When the quality of optical imagery is only limited by diffraction, the Raleigh criterion for resolution says that two Airy disks can just be resolved when the center of one lies at the edge of the other. In other words, the angle (β_{\min}) between the points is $\beta_{\min} = 1.22 \lambda/p$. A useful approximation to this equation is $\beta_{\min} = 2.3/p$ (where β_{\min} is in minutes of arc and p is in millimeters). Applying the Raleigh criterion, a 4.6-mm pupil would be required to achieve a minimum angle of resolution (MAR) of 0.5 minutes of arc (minarc); a 2.3-mm pupil is required for MAR equaling 1 minarc, and a 1.1-mm pupil allows MAR to equal 2 minarc. High resolution cannot be achieved with very small pupils or pinhole apertures.

Obviously, resolution suffers when image quality is degraded by focusing errors, such as myopia, hyperopia, astigmatism, or the failure to optimize focus by appropriate accommodation or spectacle lenses. Even with optimal refractive correction and focusing, there still may be image degradation as a result of the chromatic and monochromatic aberrations of the eye. Image degradation from aberrations increases with large pupil diameters. With very small pupils, the optical limitation

on resolution is imposed by diffraction; however, with large pupils, it is the aberrations that limit optical performance.¹⁻³ For maximum visual acuity, the optimal pupil size is about 2.5 mm, and the resolution limit is just under 1 minarc.

NEURAL LIMITATIONS

The neural limit to resolution is imposed by the packing density of the retinal receptors and the neural interactions in the retina and subsequent visual pathways. In the foveal region, where the retina achieves best resolution, the separation between centers of neighboring cones is about 2 μm . Thus, 4 μm would separate the images of two points when they fall on the centers of two receptors that are separated by one unstimulated receptor. Assuming that this situation represents the anatomically imposed limitation to resolution and that the nodal point of the eye is 16.67 mm from the retina, it is predicted that the neural limit to resolution should be 0.82 minarc. This is similar in magnitude to the optical limit.

TESTS OF VISUAL RESOLUTION

A variety of different tests of visual performance measure some aspect of the limits of the visual system's ability to discern detail or to recognize detailed targets.

Minimum Detectable Resolution

The minimum detectable resolution is the threshold size of a spot or a line required to detect its presence against its background. Consider a light spot displayed against a dark background. If the spot is very small, the width of the retinal image is determined by diffraction. The width of this image is independent of the width of the spot. If the geometrical image of the spot is smaller than the diameter of one receptor, further reduction of

the spot size simply reduces the total amount of light falling on that receptor. The task of the visual system now becomes one of contrast discrimination. The visual system has to distinguish that the amount of light falling on that receptor is greater than that falling on its neighbors. The functional question becomes, "What is the size of the smallest spot that can cause a detectable elevation in the total illuminance on the receptor?" A similar argument would apply to the detection of a light line on a dark background or a dark spot or line against a light background.

Minimum Separable Resolution

The minimum separable resolution is the least separation between two adjacent points or adjacent lines that allows the two to be seen as separate. The minimum separable value is often used to evaluate the performance or quality of optical systems, and it can be used to measure the resolution capacity of the human visual system. Popular alternative targets for measuring the minimal separable resolution are gratings or sets of three lines. For such gratings or three-line targets, the alternating dark and light lines are of equal width (duty-cycle, 1.0). For the three-line target, the observer's task is to determine the minimum separation of lines that allows them to be distinguished as three different lines. For grating targets, the task is to determine the finest grating that can just be distinguished from a uniform field of the same average luminance. Some laboratory tests of vision present displays of gratings in which the luminance distribution across the grating has a sinusoidal profile. For grating targets, the resolution limit is usually expressed in cycles per degree (cpd). At 30 cpd, there are 30 dark and 30 light lines within each degree so that the average line width is $\frac{1}{60}$ degree (equal to 1 minarc).

Sometimes, with periodic patterns, "spurious resolution" occurs. If, for example, the angular size of a three-line target is progressively reduced, the three lines eventually become unresolvable. A further reduction in angular size may cause the target to become indistinct, but it might appear that there are two lines rather than three. This depends on the luminance profiles of the images of each line and their combination when they overlap. The presence of lines is detectable, but the resolution is spurious, because three lines appear as two. For grating targets, spurious resolution can cause paradoxical reversals of threshold, because the presence of the grating can become more detectable as the spatial frequency is increased. This effect is more likely to occur when the eye is not in clear focus and the limits to resolution are being determined by optical rather than neural factors.

Some instruments designed for screening visual acuity use checkerboard targets wherein the patient is presented with four square areas, three of which contain

a uniform gray or a fine halftone pattern, whereas the fourth square contains a relatively coarse checkerboard or dot pattern. The patient's task is to determine which of the four areas contains the checkerboard. The mean luminance of the gray halftone squares is matched to that of the checkerboard pattern so that the four squares appear to have equal luminance when the checkerboard cannot be resolved.

Recognition Resolution

Most clinical tests of visual acuity are recognition tests that determine the smallest symbols, letters, or words that can be identified correctly. Test targets used for these tests are often called *optotypes*. The Snellen chart (Figure 7-1) uses letters as the optotypes.

Landolt Rings

The Landolt ring target—or "Landolt C"—consists of a circle with a break in it (Figure 7-2). The external diameter of the ring is five times the stroke width of the circle so that the internal diameter is three stroke widths. The break or gap is one stroke-width wide. For most Landolt ring tests, the gap is presented in four alternative locations: up, down, right, or left. Sometimes, there are eight alternative gap positions (four cardinal and four oblique). The observer's task is to determine the location of the gap for each Landolt ring presented. Unlike most other optotypes, the critical detail in the Landolt ring is well defined and unambiguous: it is the gap in the ring. Thus, the critical detail is one-fifth the height of the optotype. At threshold or near-threshold levels, the observer does not necessarily see the target as a ring with a gap in it. Rather, the target appears as a small spot or blob with a region that is marginally asymmetrical or lighter, and it is this irregularity that identifies the gap position.

Letter Optotypes

Most letters designed for visual acuity tests are based on grid patterns that are five units high. They have usually been five units wide although letter widths of four or six units have sometimes been used. The stroke width of the letters is usually a fifth of the height and, as much as is practical, the spacing between adjacent strokes is made equal to the stroke width. Snellen⁴ introduced the letter chart (see Figure 7-1) for visual acuity measurement, and he designed his optotypes so that the major limb strokes were one-fifth the letter height. Many of the acuity charts that followed⁵ used a similar approach, and, like the original Snellen design, most used serifs (short lines or blocks added at an angle to the ends of limbs of the letters) on the letters. More modern letter charts use sans-serif letters. Today, the most commonly used sans-serif letters are the Sloan letters,⁶ which are based on a five-by-five grid. In the Sloan letter set, there

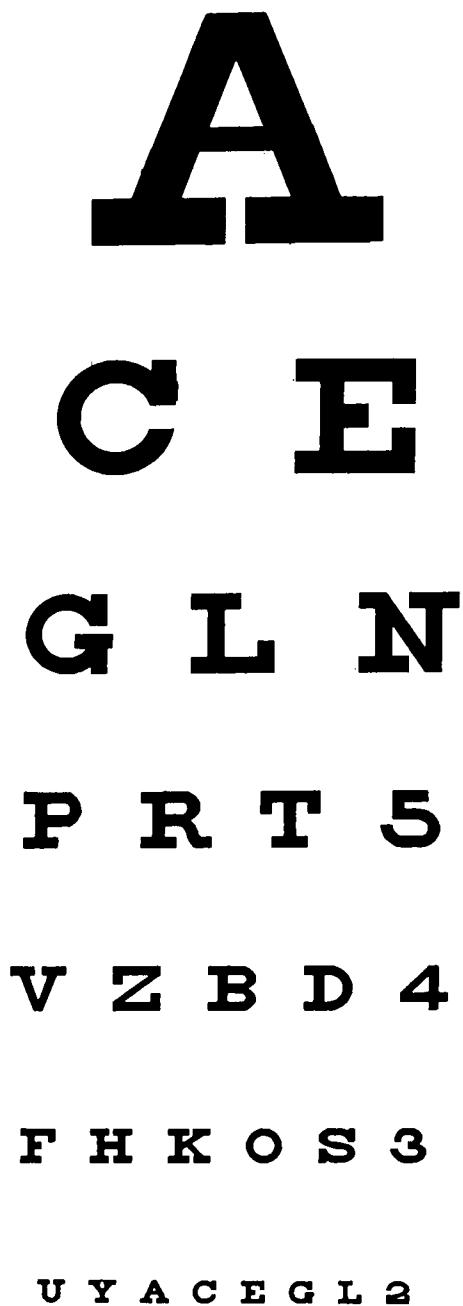


Figure 7-1

Snellen's original chart, shown at about 40% of its actual size.

are 10 letters (C, D, H, K, N, O, R, S, V, Z), with specified angles and curvatures for each. A previous British standard of optotypes⁷ used a different set of 10 letters (D, E, F, N, H, P, R, U, V, Z) based on a five-by-four grid. Figure 7-2 shows examples of a Landolt ring, a five-by-five serif letter, a Sloan (five-by-five) letter, and a 1968 British (five-by-four) letter.

The 2003 British standard on optotypes⁸ introduced a new set of 12 sans-serif letters that is also based on

the five-by-five grid. Five of these letters are identical to the Sloan letters (C, H, N, V, Z). There are a new K and a new R with different limb angles, and a new D with different curvatures. There are four letters in addition to the Sloan set (E, F, P, U), and there are two Sloan letters that do not appear in the new British series (O, S). The selection of limited letter sets and the specification of the letter designs are intended to reduce the variability of legibility between letters. However, within each letter set, there always remains some variation in the legibility of the individual letters. Chart designers should arrange the mixtures of letters at each size so that the average legibility is similar at each acuity level. A comparison of the Sloan and British Standard⁸ letters is contained in Table 7-1.

For the recognition of letter targets at or near threshold sizes, a variety of clues or combinations may be responsible for the correct letter identification. For example, the letters N and H are similar in their general shape, and, when close to threshold size, they might be distinguished from most other letters with relative ease. The patient might see a squarish letter and narrow down the choice to H or N. For the final distinction, the critical cue for correctly identifying the N might be the detection of its diagonal limb, seeing that there is an offset of the notch in the upper and lower edges of the square, or recognizing that there is a concentration of darkness in the upper-left and lower-right corners of the square.

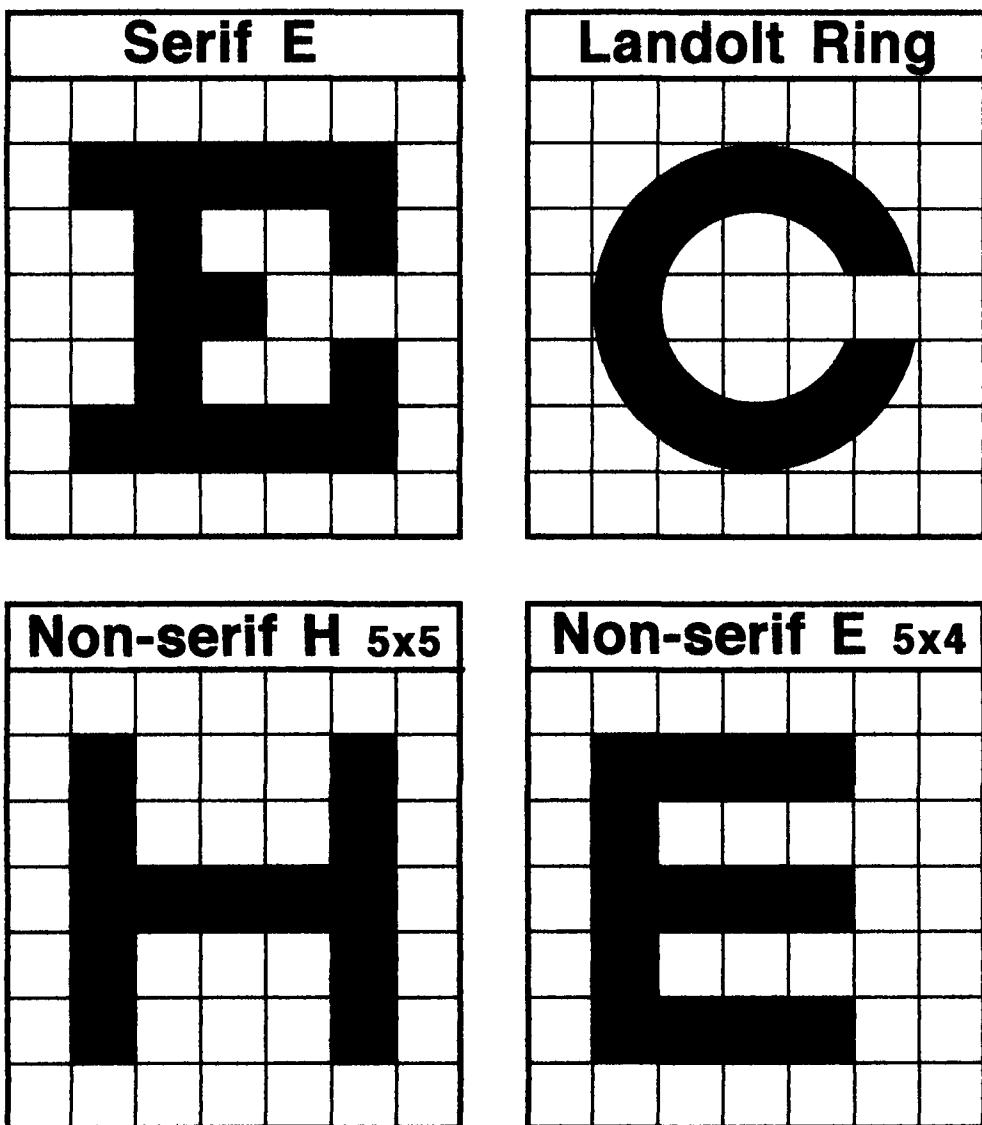
Tumbling E

The tumbling E target, sometimes called the "illiterate E," is based on a five-by-five grid. The E is presented in different orientations at every acuity level, and the patient's task is to identify the direction to which the limbs of the E point. Most commonly, there are four alternative directions: up, down, right, and left. Some tests, however, use eight alternatives, with the addition of the four oblique directions. The letter E usually has three limbs of equal length. The recent British standard⁸ specified an illiterate E with the central limb one unit shorter than the external limbs. Tumbling E targets are most useful when measuring acuity in toddlers or other persons who are not familiar with the alphabet.

Numerical and pictorial targets are available, and they are mainly used with pediatric and illiterate populations.⁹ These are further discussed in Chapter 30.

DESIGNATION OF VISUAL ACUITY

Visual acuity expresses the angular size of the *smallest* target that can just be resolved by the patient, but there are several different ways in which clinicians specify this angular quantity (Table 7-2).

**Figure 7-2**

Examples of optotypes constructed on a grid framework.

Snellen Fraction

The Snellen fraction expresses the angular size of optotypes by specifying the test distance and the height of the letters. In the Snellen notation, the number used to indicate the height of the letters is the distance at which the letter height subtends 5 minarc. In other words, a 20-foot (or 6-m) letter is one with a height that subtends 5 minarc at 20 feet (or 6 m). The Snellen fraction is written with the test distance as its numerator and the letter size as its denominator:

$$\text{Visual acuity} = (\text{test distance}) / (\text{distance at which letters subtend 5 minarc})$$

A visual acuity score of 20/200 means that the test distance was 20 feet and the smallest letters that could

be read would subtend 5 minarc when at a distance of 200 feet. The angular size of such letters at 20 feet is 50 minarc. Provided the retinal image is kept in good focus, the visual acuity should not change with test distance. Thus, 20/200, 40/400, 10/100, 5/50, and 6/60 are all visual acuity scores that represent the same angle (letters subtend 50 minarc); the test distances and the threshold print sizes are different, but they remain in proportion. In the United States, distances are expressed in feet, and clinicians almost invariably use the Snellen fraction with 20 feet as the numerator. In most other countries, metric units are used, with 6 m being the most common test distance. Thus, 20/20 is equivalent to 6/6, 20/25 to 6/7.5, 20/40 to 6/12, 20/100 to 6/30, 20/200 to 6/60, and so forth (see Table 7-2).

TABLE 7-1 Comparison of the Sloan Letters and British Standard (2003) Letters

Sloan Letters	British 2003 Letters	Letter Height	Letter Width	Stroke Width	Exterior Radius	Interior Radius	Angles	Relative Legibility
C	C	5	5	1	2.5	1.5	—	0.99
D		5	5	1	1.5	0.5	—	1.01
	D	5	5	1	2.5	1.5	—	—
—	E	5	5	1	—	—	—	—
—	F	5	5	1	—	—	—	—
H	H	5	5	1	—	—	—	1.06
K		5	5	1	—	—	37/128 45/135	0.99
N	N	5	5	1	—	—	131	1.05
O	—	5	5	1	2.5	1.5	—	0.90
—	P	5	5	1	1.5	0.5	—	—
R		5	5	1	1.5	0.5	116	0.97
	R	5	5	1	—	—	127	—
S	—	5	5	1	1.5	0.5	—	0.93
—	U	5	5	1	2.5	1.5	—	—
V	V	5	5	1	—	—	112/68	1.05
Z	Z	5	5	1	—	—	41	1.10
n = 10	n = 12							

Five letters are identical (C, H, N, V, Z) in both families.

Three letters (D, K, R) are in both families, but the shapes are not identical.

Two letters (O, S) are only in the Sloan series.

Four letters (E, F, P, U) are only in the British series.

In the British F and E, one horizontal limb is 1 unit shorter than the other(s).

Legibility data are not available for the 2003 British letters.

Decimal Notation

The decimal notation effectively reduces the Snellen fraction to a decimalized quantity. Thus, 20/20 (or 6/6) becomes 1.0, 20/200 (6/60) becomes 0.1, 20/40 (6/12) becomes 0.5, and so forth. Decimal notation is most widely used on the European continent; it gives a single number to quantify an angle, and it does not indicate the test distance.

Minimum Angle of Resolution

The MAR is typically expressed in minutes of arc, and it indicates the angular size of the critical detail within the just-resolvable optotype. For letters, the critical detail is taken as one fifth of the letter height. For a visual acuity of 20/20 (or, in metric units, 6/6), the MAR is equal to 1 minarc. For 20/40 (or 6/12), the MAR is 2 minarc; for 20/200 (or 6/60), the MAR is 10 minarc. The MAR in minutes of arc is equal to the reciprocal of the decimal acuity value.

Logarithm of the Minimum Angle of Resolution

The logarithm of the MAR (logMAR)¹⁰ is the common logarithm of the MAR. When visual acuity is 20/20 (or 6/6), the MAR is equal to 1 minarc, so the log MAR equals $\log_{10}(1.0)$ equals 0.0. For 20/40 (or 6/12), the MAR is 2 minarc, so logMAR equals $\log_{10}(2.0)$ equals 0.30. For 20/200 (or 6/60), the MAR is 10 minarc, so logMAR equals $\log_{10}(10)$ equals 1.0.

When the visual acuity score is better than 20/20 (or 6/6), the logMAR value becomes negative. For example, for 20/16 (or 6/4.8), MAR equals 0.8 minarc and $\log_{10}(0.8)$ equals -0.10. For charts that have a size progression ratio of 0.1 log units and five letters per row, each letter can be assigned a value of 0.02 on the logMAR scale.

Visual Acuity Rating

The visual acuity rating (VAR)¹¹ is derived from the logMAR values:

$$\text{VAR} = 100 - 50 \log\text{MAR}$$

TABLE 7-2 Conversion Table for Visual Acuity Scores

LogMAR Notation	VAR Notation	MAR Exact	MAR Notation*	Decimal Notation*	Grating cpd	VE% Notation	DISTANCE VISION				SNELLEN FRACTIONS				NEAR VISION			
							Based on 20 ft*	Based on 6 m*	Based on 4 m*	Snellen notation 0.40 m*	M	Units	N points*	x-Height (mm)	"Reduced Snellen"**	Jaeger (approximate)	AT 40 cm	AT 14 INCHES
-0.30	115	0.501	0.50	2.00	60	109.4%	20/10	6/3	4/2	0.40/0.20	0.20	1.6	0.29	10			14/7	
-0.20	110	0.631	0.63	1.60	48	106.8%	20/12.5	6/3.8	4/2.5	0.40/0.25	0.25	2.0	0.36	20/12.5			14/8.8	
-0.10	105	0.794	0.80	1.25	38	103.6%	20/16	6/4.8	4/3.2	0.40/0.32	0.32	2.5	0.47	20/16			14/11	
0.00	100	1.000	1.00	1.00	30	100.0%	20/20	6/6	4/4	0.40/0.40	0.40	3.2	0.58	20/20	J1		14/14	
0.10	95	1.259	1.25	0.80	24	95.6%	20/25	6/7.5	4/5	0.40/0.50	0.50	4.0	0.73	20/25	J1-J2		14/17.5	
0.20	90	1.585	1.60	0.63	19	89.8%	20/32	6/9.5	4/6.3	0.40/0.63	0.63	5.0	0.92	20/32	J1-J4		14/22	
0.30	85	1.995	2.0	0.50	15	83.6%	20/40	6/12	4/8	0.40/0.80	0.80	6.3	1.16	20/40	J2-J5		14/28	
0.40	80	2.512	2.5	0.40	12	76.5%	20/50	6/15	4/10	0.40/1.00	1.00	8.0	1.45	20/50	J3-J6		14/35	
0.50	75	3.162	3.2	0.32	9.5	67.5%	20/63	6/19	4/12.5	0.40/1.25	1.25	10.0	1.82	20/63	J4-J8		14/44	
0.60	70	3.981	4.0	0.25	7.5	58.5%	20/80	6/24	4/16	0.40/1.60	1.60	12.5	2.33	20/80	J5-J9		14/56	
0.70	65	5.012	5.0	0.20	6.0	48.9%	20/100	6/30	4/20	0.40/2.0	2.0	16	2.91	20/100	J8-J12		14/70	
0.80	60	6.310	6.3	0.160	4.8	38.8%	20/125	6/38	4/25	0.40/2.5	2.5	20	3.64	20/125	J9-J13		14/88	
0.90	55	7.943	8.0	0.125	3.8	28.6%	20/160	6/48	4/32	0.40/3.2	3.2	25	4.65	20/160	J10-J15		14/110	
1.00	50	10.00	10.0	0.100	3.0	20.0%	20/200	6/60	4/40	0.40/4.0	4.0	32	5.82	20/200	J14-J18		14/140	
1.10	45	12.59	12.5	0.080	2.4	12.8%	20/250	6/75	4/50	0.40/5.0	5.0	40	7.27	20/250			14/175	
1.20	40	15.85	16	0.063	1.9	6.8%	20/320	6/95	4/63	0.40/6.3	6.3	50	9.16	20/320			14/220	
1.30	35	19.95	20	0.050	1.5	3.3%	20/400	6/120	4/80	0.40/8.0	8.0	63	11.6	20/400			14/280	
1.40	30	25.12	25	0.040	1.2	1.4%	20/500	6/150	4/100	0.40/10.0	10.0	80	14.5	20/500			14/350	
1.50	25	31.62	32	0.032	0.95	0.4%	20/630	6/190	4/125	0.40/12.5	12.5	100	18.2	20/630			14/440	
1.60	20	39.81	40	0.025	0.75		20/800	6/240	4/160	0.40/16	16	125	23.3	20/800			14/560	
1.70	15	50.12	50	0.020	0.60		20/1000	6/300	4/200	0.40/20	20	160	29.1	20/1000			14/700	
1.80	10	63.10	63	0.016	0.48		20/1250	6/380	4/250	0.40/25	25	200	36.4	20/1250			14/880	
1.90	5	79.43	80	0.013	0.38		20/1600	6/480	4/320	0.40/32	32	250	46.5	20/1600			14/1100	
2.00	0	100.0	100	0.010	0.30		20/2000	6/600	4/400	0.40/40	40	320	58.2	20/2000			14/1400	

*Numbers rounded to simplify sequences. Rounding errors do not exceed 1.2%.

On this scale, a score of 100 corresponds with 20/20 (6/6). A VAR that equals 50 corresponds with a Snellen fraction of 20/200 (6/60). The VAR equals 0 when the visual acuity is at the 20/2000 (6/600) level. The VAR is greater than 100 when visual acuity is better than 20/20 (or 6/6). For example, for 20/16 (or 6/4.8), VAR equals 105. On charts that use a 0.1-log unit-size progression, the VAR score changes by 5 for each size increment. If, in addition, there are five letters per size level, each letter carries a VAR value of 1. The VAR scale can facilitate the scoring of visual acuity. On the VAR scale, a difference of 15 points represents a twofold change in the MAR, and a 5-point change represents a change with ratio of 5:4 in the MAR. The VAR scoring system has been used in the *Guides to the Evaluation of Permanent Impairment*.¹² A functional acuity score (FAS) is obtained by adding the VAR for the right eye, the VAR for the left eye, and three times the binocular VAR and then dividing the sum by 5:

$$\text{FAS} = (\text{VAR}_{\text{OD}} + \text{VAR}_{\text{OS}} + 3 \text{VAR}_{\text{OU}})/5$$

Visual Efficiency

The visual efficiency (VE) scale was introduced in 1925 by Snell and Sterling^{13,14} for use when quantifying visual loss for legal and compensation purposes. The scale was developed on the basis of experiments in which visual resolution was degraded by adding a series of diffusing filters before the eyes, and it was assumed that vision was degraded by the same amount as each additional filter was introduced. The VE was deemed to be 1.0 (or 100%) when visual acuity was 20/20 or 6/6. Arbitrarily, 20/200 or 6/60 was said to represent a VE of 0.2 (20%). Given these two chosen benchmarks, a good fit of their experimental data was obtained by the following relationship:

$$\text{VE} = 0.2^{(\text{MAR} - 1)/9}$$

It is more common for this relationship to be expressed in the following form:

$$\text{Log(VE\%)} = 2.0777 - 0.0777 (\text{MAR})$$

The American Medical Association (AMA)¹⁵ adopted the Snell-Sterling scaling of VE. The system was expanded by developing VE ratings to quantify losses of visual fields and ocular motility. The AMA system for the evaluation of permanent visual impairment¹⁶ allows the calculation of an overall rating of VE that is the product of acuity, field, and motility efficiency scores. The AMA system combines the monocular VE for the two eyes, giving three times more weight to the VE of the better eye. This system became obsolete with the AMA's recent publication of its *Guides to the Evaluation of Permanent Impairment*, 5th Ed.¹²

VISUAL ACUITY CHART DESIGN

Snellen Chart

Snellen's original chart⁴ had seven different size levels. There was only one letter at the largest size level, and the number at each size level increased progressively to eight optotypes (seven letters and one number) at the smallest size (see Figure 7-1). The size sequence in feet was essentially 200, 100, 70, 50, 40, 30, and 20 (or, in metric units, 60, 30, 21, 15, 12, 9, 6.) Many modifications were made to Snellen's original chart design, and detailed descriptions of many of these are provided in the Bennett's of ophthalmic test types.⁵ Despite significant deviations from Snellen's original design (i.e., differences in letter design and selection, size progressions, spacing relationships, and number of letters at the various size levels), it is still common to apply the term "Snellen charts" or even "standard Snellen charts" to charts that have a single letter at the top and increasingly more letters at the smaller sizes.

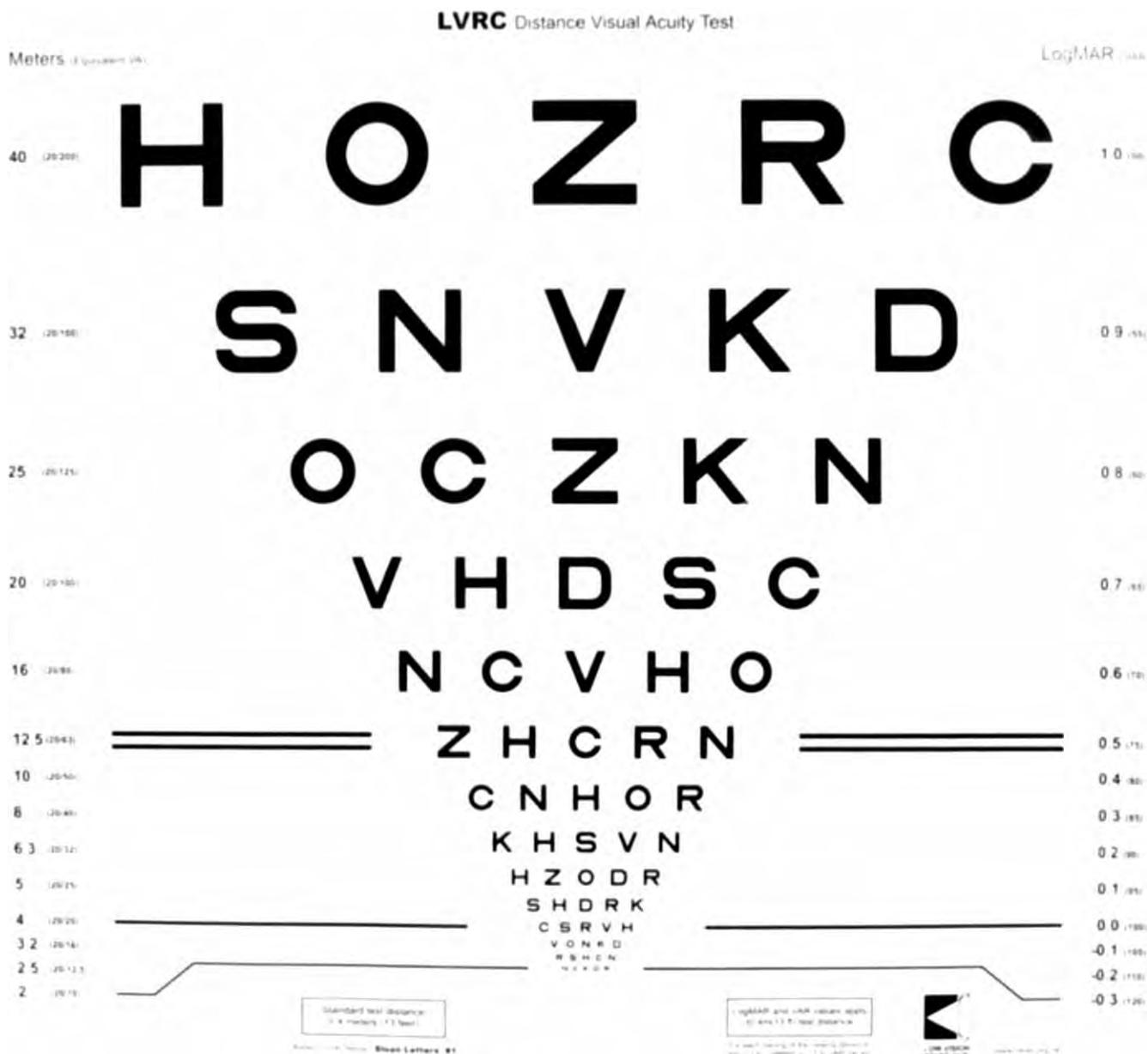
Bailey-Lovie Design Principles

Bailey and Lovie¹⁰ proposed a set of principles for the design of visual acuity charts, and these make the task essentially the same at each size level (Figure 7-3). Thus, size becomes the only significant variable when changing from one size level to the next. Such standardization of the visual acuity task requires the following:

1. A logarithmic size progression (constant ratio from one size to the next)
2. The same number of letters at each size level
3. Spacing between letters and between rows that is proportional to letter size
4. Equal (or similar) average legibility for the optotypes at each size level

Along with these chart design principles, they introduced the clinical scoring of visual acuity in logMAR units as well as a method for giving equal additional credit for each additional letter that is read correctly.

Several charts have since been developed in accordance with these principles. Taylor¹⁷ prepared a tumbling E chart. Ferris and colleagues¹⁸ made a chart for the Early Treatment of Diabetic Retinopathy Study (ETDRS) using Sloan letters rather than the British letters that were used in the original version of the Bailey-Lovie chart. Strong and Woo¹⁹ arranged Sloan letters with sizes progressing in columns rather than rows, and they added masking bars to the ends of the columns and rows. Johnston²⁰ prepared a version using Chinese characters, and Hyvarinen and colleagues²¹ prepared charts using abstract "LH symbols" for testing children. The Bailey-Lovie chart design using four-position Landolt rings is shown in Figure 7-4. The same principles have been used for charts with Arabic, Indian, and Thai characters.²²⁻²⁴

**Figure 7-3**

Visual acuity chart designed according to the principles of Bailey-Lovie (also known as a LogMAR chart design), shown at 20% of its actual size. The optotypes used here are Sloan letters, as in the ETDRS charts. LogMAR, Logarithm of the minimum angle of resolution; VAR, visual acuity rating. (Courtesy of the Low Vision Resource Centre, Hong Kong Society for the Blind, Kowloon, Hong Kong.)

Design Features for Visual Acuity Charts

Logarithmic Size Progression

Logarithmic scaling of size on visual acuity charts has long been advocated by Green,²⁵ Sloan,⁶ and many others,⁵ and it is now broadly accepted. Westheimer²⁶ provided evidence and argument that logarithmic scaling is more appropriate than other alternatives. He measured peripheral visual acuity at different retinal

eccentricities, and he found that, across the range of measured visual acuity values, the variance of measurement was virtually constant if visual acuity was expressed on a logarithmic scale. Thus, just-noticeable differences are about equal in size if the scale is logarithmic. Although several different logarithmic scaling ratios have been suggested, common practice today uses a size progression of 0.1 log unit ($10^{0.1}$). With such a size

LVRC Near Visual Acuity Test

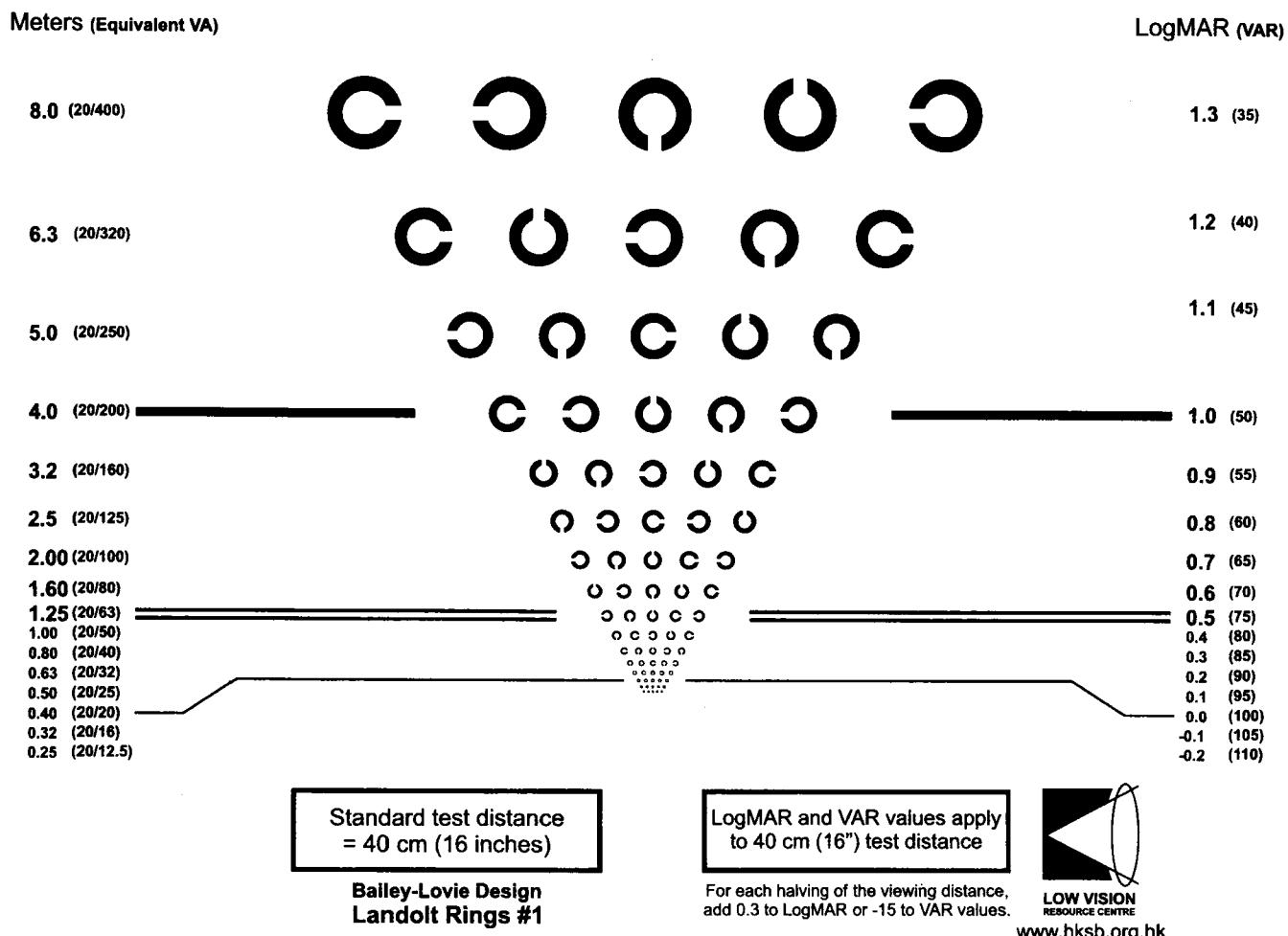


Figure 7-4

A Landolt ring chart following the Bailey-Lovie design, calibrated for a viewing distance of 40 cm. This chart is printed on a card that is 228 mm × 176 mm. (Courtesy of the Low Vision Resource Centre, Hong Kong Society for the Blind, Kowloon, Hong Kong.)

progression, each successive step represents a change in size by the ratio 1.2589:1 (approximately 5:4). A change of 10 increments on this scale represents a change of exactly 10 times, and a change of three steps represents a change of approximately two times. With a small amount of rounding to give more convenient numbers, the sequence progresses as follows: 1.0, 1.25, 1.60, 2.0, 2.5, 3.2, 4.0, 5.0, 6.3, 8.0, 10, 12.5, 16, and so on. For a test distance of 6 m, the sequence becomes 6.0, 7.5, 9.5, 12, 15, 19, 24, 30, 38, 48, 60, 75, 95, and so on. A more exact sequence for the logarithmic progression is shown in the second column of Table 7-2. This table also shows the approximations as they are usually applied when scoring visual acuity in terms of MAR, decimal notation, or Snellen notation based on 20 feet or 6 m.

Letter Legibility

The Landolt ring target has been recommended by the NAS/NRC Committee on Vision²⁷ and by the Concilium Ophthalmologicum Universale²⁸ as the reference optotype against which the legibility of all other optotypes should be calibrated. It is usually assumed that, for Landolt rings, the gap position is equally detectable for all four alternative orientations of the ring, but the gap position is slightly more difficult to detect when it is located in oblique positions. However, the EDTRS charts with the Sloan optotypes have been so commonly used in research studies around the world that this chart and its optotypes have effectively become the "gold standard" to which alternatives should be compared.

Typically, clinicians prefer letters as the visual acuity test targets rather than targets such as Landolt rings or

tumbling Es, which require the patient to identify orientations. When patients are naming orientations, it is more difficult for the clinician to keep track of which optotype is being read at any given instant. It is particularly difficult when the patient skips, repeats, or corrects their reading of an optotype or of a whole row. In addition, some patients make mistakes when calling left or right (e.g., gesturing right but calling left). When there are only four possible orientations, the probability of guessing correctly is relatively high (0.25). Although letters of the alphabet show variability in their individual legibility, they do offer many advantages that appeal to clinicians. The probability of guessing correctly is small, being 1 in 26 for random guessing but greater if the patient realizes that not all 26 letters are used on the chart. It is easier for clinicians to recall letter sequences and to verify that a row has been read correctly, even when the clinician temporarily loses track of which letters are being read by the patient.

The Sloan letters are most widely used today. The 10 five-by-five letters show some small variability in their individual legibility.^{5,6,29,30} When there is significant variation in the relative legibility within a set of optotypes, it is desirable to select the group of optotypes at each size level so that each group has approximately the same average difficulty. This has been done for the ETDRS chart with Sloan letters and for the Bailey-Lovie chart with 1968 British standard letters.

Number of Optotypes at Each Size Level

The reliability of visual acuity measures increases with increased number of letters at the near-threshold sizes.³¹⁻³³ Doubling the number of letters at each size level should reduce the standard deviation of measurements (and, correspondingly, the confidence limits for detecting change) by a factor of $1/\sqrt{2}$ (i.e., 0.71). Similarly, a finer size progression would also improve the reliability of measurement. Provided that the size pro-

gression is not excessively coarse, the reliability of visual acuity measurements is inversely proportional to the square root of the average logMAR value per letter (i.e., the size progression ratio in log units/number of letters at each size). This represents the sampling frequency

$$SD = k\sqrt{p/n}$$

where SD is the standard deviation of visual acuity measurement in logMAR units; p is the size progression ratio in log units; n is the number of letters at each size; and k is a constant that depends on the optotype and chart design. For five-letter rows and a 0.1-log unit size progression, standard deviation of letter chart acuity is about 0.028. To detect or identify change, it is necessary to establish confidence limits. This is the range of differences between test and retest values that, if exceeded, is taken as being caused by a real change rather than the result of noise in the measurement. The standard deviation of test-retest discrepancies is equal to $\sqrt{2}$ times the standard deviation of the measurement. The 95% confidence limits for change may be taken as 1.96 times the standard deviation of test-retest discrepancies. If visual acuity is scored on a letter-by-letter basis, to apply 95% confidence limits, the criterion for change should be taken as the next scale increment beyond that which contains the 95th percentile. Table 7-3 presents a few examples to show how size progression ratios and number of letters per size level can affect the standard deviation of measurement, the standard deviation of the test-retest discrepancies, and the criterion for change.

Spacing Between Letters and Between Rows

Spacing between neighboring letters reduces their legibility. Flom and colleagues^{34,35} coined the term "contour interaction" to describe the effect that neighboring spatial contours have on the discriminability of small detail. They conducted experiments using Landolt rings

TABLE 7-3 Letter Chart Design and Confidence Limits for Change

Progression logMAR	No. of Letters at Each Size	LogMAR per Letter	Standard Deviation of Measurement	Standard Deviation of Test-Retest Differences	Confidence Limits Calculated	Criterion for Change
0.100	5	0.02	0.028	0.040	0.078	5 letters (0.10 log MAR)
0.100	10	0.01	0.020	0.028	0.055	7 letters (0.07 log MAR)
0.050	5	0.01	0.020	0.028	0.055	7 letters (0.07 log MAR)
0.200	10	0.02	0.028	0.040	0.078	5 letters (0.10 log MAR)
0.200	5	0.04	0.040	0.057	0.111	4 letters (0.16 log MAR)

Standard deviations and confidence limits in the first row are all based on empirical measurements.

The latter four rows give projections made according to the sampling frequency.

The sampling frequency (logMAR/letter) is determined by the size progression and the number of letters at each size.

with "masking bars" located above, below, to the right of, and to the left of the ring. They found that the discrimination of the gap position depended on the separation of the masking bars from the ring. This is consistent with the clinical observation that letter legibility is increased if the letters are isolated or widely separated from their neighbors. Contour interaction should be distinguished from the "crowding" effect, which is related to the difficulty of reading letters caused by the requirement of finer eye movements to read letters when they are in a tightly packed array.³⁶ From experiments in which the spacing between optotypes was varied from 0.5 to 3.0 times the height of the optotype, Bailey and Raasch^{11,29} found that a twofold change in spacing altered the visual acuity score by 0.03, 0.04, and 0.07 log units for British letters, Sloan letters, and Landolt rings, respectively. For low-contrast (10% Michelson) charts, spacing had little effect on visual acuity scores.³⁷ Although spacing arrangements within a chart may influence visual acuity scores, the choice of the spacing ratio is arbitrary. The space between adjacent rows and between adjacent letters is usually made equal to the letter width. Visual acuity is better when the spacing is wider. It should be recognized that eye-movement control and fixation tremor may contribute to the reduction of visual acuity when the letters are tightly spaced and that the influence of such motor factors is greater when the threshold print size is smaller.

CLINICAL TESTING OF VISUAL ACUITY

Chart Formats

Visual acuity charts may be prepared as printed panels or as slides to be projected onto a screen, or they may be generated for video display. The chart panel, projection screen, or video screen is often viewed directly, but, when the room dimensions do not permit the desired test distance, mirrors may be used to lengthen the optical path from the chart to the patient.

Printed Panel Charts

Printed panel charts come in a variety of forms. Many are printed on opaque card or plastic, and these are directly illuminated. Others are printed on translucent material and mounted on a light box that provides illumination from the rear (back illumination). The different print sizes on the chart are usually labeled as the distance in feet or meters at which the letters subtend 5 minarc. Most commonly, panel charts are presented at a distance of 20 feet (or 6 m), and the acuity is recorded as the Snellen fraction. Closer test distances are used when the examination room does not permit chart presentation at the standard distance or when the patient

has low vision and is unable to read the largest letters on the chart. To ensure that the patient's resolution threshold lies within the range of the chart, the patient should be able to read the letters at the largest size but unable to read the letters at the smallest size. Clinicians adopting closer test distances typically choose a distance that is a simple fraction of the standard, because this facilitates the comparison of visual acuity scores. For example, 10 feet or 5 feet are preferred close distances for charts designed for presentation at 20 feet.

When one is using printed panel charts, the distance from the patient to the chart and the size of the letters must be known to determine the visual acuity. With some charts, however, the print size is labeled not according to the letter height but rather as the letters' angular size for a specific test distance. Testing with such charts at any distance other than the specific "standard" distance requires an adjustment to the score. However, there is then some risk of error when converting scores to compensate for the use of a nonstandard test distance. For example, the ETDRS charts¹⁸ are designed for a 4-m presentation distance, and the top row is labeled "20/200," although its letters subtend 5 minarc at 40 m (131 feet) rather than at 200 feet or 60 m. Reading this top row at 4 m should earn a score of 4/40 (or, in imperial units, 13.1/131), which, in angular terms, is equivalent to 20/200 (6/60). If the chart is moved to, for example, 1 m (3.3 feet) and a patient can just read the top row (40-m letters labeled 20/200), the clinician might erroneously assign an acuity score of 3.3/200 (1/60), and this would be considered equivalent to 20/1200 (6/360). In this example, however, it would have been correct to record the visual acuity score as 3.3/131 (1/40), and this can be considered equivalent to 20/800 (6/240).

Although 20 feet or 6 m is the most widely used test distance, 4 m has been recommended by Hofstetter³⁸ and, subsequently, by some authoritative bodies.^{27,28} A 4-m test distance facilitates making a dioptric allowance (of 0.25 D) to the refractive correction to allow for the chart being closer than optical infinity. Also, using 4 m as the standard for testing distance vision facilitates comparison with near-vision measurements, in which 40 cm is commonly used as a standard test distance.

Projector Charts

If the projector lens and the patient's eye are equally distant from the projection screen, the angular size of the chart and its component optotypes of the projector chart image are independent of the observation distance. Consequently, the designation of print size on projector charts is usually in angular terms. The equivalent Snellen fraction is used on most American charts, and decimal acuity notation is used on European projector charts. If the viewing conditions are arranged so that observation distance is 20 feet (or 6 m) and the

projector is appropriately positioned with respect to the screen, the expression of visual acuity as a Snellen fraction is straightforward. If, however, the optical path length from the patient to the screen is some other distance, a proportional change needs to be made to the size of the projected letters. For example, if 18 feet (5.4 m) was the observation distance, the projector system should be adjusted so that the row designated as "20/200" has the height of its letters subtending 50 minarc at 18 feet (5.4 m); then all other letters' sizes would be scaled proportionately. If this row is the smallest that the patient can read, the visual acuity as a Snellen fraction should strictly be expressed as 18/180 (or 5.4/54); however, it is usual to record such a visual acuity result as 20/200 (6/60), which is the equivalent Snellen fraction. Projector chart systems are usually arranged so that the distance from the patient to the screen is never varied.

The angular width of chart displays for the common clinical projectors is usually about 2.5 degrees square, and this limits the number of letters that can be displayed in a single row at the larger sizes. If 11 character spaces are allowed to display a row of five characters, then the largest presentable row on a 2.5-degree display has a visual acuity value of about 20/55 (6/16 or logMAR = 0.43). The largest angular size available in most projectors is 20/400 (6/120), and typically only one letter of this size can be presented per display. Standard 35-mm slide projectors can present wider fields, and they can readily allow five letters per row up to the 20/200 (6/60) level.

Charts on Display Screens

Computer-generated displays are not yet widely used in clinical practice, but they offer distinct advantages. They provide the means to select different optotypes, to change letter sequences, and to vary stimulus parameters such as contrast, spacing arrangements, and presentation time. The computer interface provides opportunities for more detailed recording and analysis of responses. Computer-controlled presentation of test targets facilitates repeated measurements with random or semi-random rearrangements of letter sets. This process avoids some of the memorization problems that can occur when using printed or projected charts. There are some brightness limitations in that the luminance levels on cathode-ray tube displays are typically less than 150 cd/m², but some newer cathode-ray tube models and many flat-panel displays provide screen luminance of up to 300 cd/m². The sizes of the display's pixels and of the screen itself impose limits on the extreme sizes (small and large) of optotypes and charts that can be presented. The pixel structure limits the size of the smallest letters, and the screen dimensions limit the size of the largest letters that can be presented in a row or singly.

At least 20 pixels are required per letter height so that the spatial structure or shapes of individual optotypes do not show significant variation from one size to the next. Even with this minimal number of pixels, some compromise must be accepted. Consider Landolt Rings or tumbling E optotypes. To maintain a 5:1 ratio between the height of the optotype and stroke or gap width, the number of pixels per letter height must be an integer multiplied by 5. If the usual logarithmic progression of size is to be preserved, the limb or gap width should be incremented in accordance with the following sequence: 1.0, 1.25, 1.6, 2.0, 2.5, 3.2, 4.0, 5.0, 6.3, 8.0, 10, and so on. The numbers in this sequence are not all integers and multiples of 5, and so an optotype will not appear the same when presented in different sizes. For example, if the very smallest letters had 20 pixels per letter height, the limb or gap width would be 4 pixels; the next larger letters would be 25 pixels high, with a 5-pixel limb width. At the next largest size, a letter must be 30 or 35 pixels high, respectively, to achieve the correct proportions with a limb width of 6 or 7 pixels. However, 32 pixels are required to achieve the desired logarithmic size progression ratio indicating the proper level of acuity. At 32 pixels, for instance, there will be three limbs or spaces of the tumbling E having a width of 6 pixels and two limbs or spaces composed of 7 pixels. The dilemma is that the chart can supply the proper letter size or the proper detail proportion—but not both at the same time—for this acuity level.

Today, it is common for display screens to have a screen resolution of 1600 × 1200 pixels (UXGA), and finer resolutions are available (QXGA = 2048 × 1536; QSXGA = 2560 × 2048). Consider the size range that could be presented in the Bailey-Lovie format on a UXGA screen. If the smallest letters were 20 pixels high and if they were to subtend 2.5 minarc (20/10, 6/3, or 4/2), the vertical height of the screen would necessarily subtend an angle of 2.5 degrees (1200 pixels, each 0.125 minarc). This would require a screen height of 17.5 cm at 4.0 meters. If there were to be five letters on each row, with the space around each letter equal to one letter width, then 11 character spaces would be required for each row of letters. Restricted by the horizontal screen dimension of 1600 pixels, the largest characters could be 145 × 145 pixels. If the largest characters were to subtend angles of 50 minarc (20/200, 6/60, or 4/40), the screen would need to horizontally subtend an angle of 9.2 degrees. Thus, the screen would need to be 65-cm wide at a viewing distance of 4 m. Even with two screens (i.e., a large screen for the large sizes and a small screen for the smaller sizes), it would be impractical to have a continuous chart that maintained a uniform format, and there would need to be some overlap of the size ranges of the two charts.

Chart Luminance

For most purposes, visual acuity measurements are made with the visual acuity chart at moderate photopic luminances, and, typically, the general room lighting is subdued. Recommendations for a standardized chart luminance range from 85 to 300 cd/m². Sheedy and colleagues³⁹ showed that, in this luminance range, doubling the luminance changes the visual acuity score by about 0.02 log units (1 VAR unit), which corresponds to one-fifth of a line or a 5% change in MAR. A compromise chart luminance that is becoming widely used as a standard is 160 cd/m². The British standard requires a luminance of at least 120 cd/m². It can be difficult to achieve specific luminance levels with different projector, light box, and video display systems, so a clinical tolerance of 80 to 320 cd/m² for test chart luminance may be reasonable and practical. To better ensure measurement consistency within a given clinical setting or among sites in a clinical study, the chosen luminance should be maintained within a 15% tolerance. When illuminating charts, one should take care to avoid glare sources within the patient's field of view. The visual performance of certain patients—particularly those with retinal pathology—may be considerably influenced by retinal illumination. The clinician may choose to vary the chart luminance to find the patient's specific lighting dependency.

Contrast is another variable that affects visual acuity. Measurement of visual acuity with low contrast (gray) optotypes is becoming more widely used, mainly for patients with corneal or lenticular disorders or for those who have had refractive surgery. Low-contrast visual acuity and its difference from high-contrast visual acuity are often regarded as measures of contrast sensitivity. The reader is referred to Chapter 8 for a detailed discussion of contrast sensitivity and its assessment.

Refractive Correction

During an eye examination, clinicians are frequently considering whether to recommend that spectacles or contact lenses be worn or whether changes should be made to the corrective lenses that the patient is currently wearing. In recent times, refractive surgery has become part of the range of interventions that may be considered by the clinician and the patient. Visual acuity measurements guide the clinician's decisions and recommendations about these various options for treating refractive errors. The acuity measurements of most relevance are the visual acuity that may be obtained with the best spectacle or contact lens correction, the visual acuity obtained when no spectacles or contact lenses are being worn, and the visual acuity measured with the refractive correction that the patient usually wears while performing common distance vision tasks of daily life. The increasing use of surgical treatments of refractive

error and the expected development of methods to correct higher-order optical aberrations have created the need to modify some of the terminology used when referring to different kinds of visual acuity measurements.

Unaided visual acuity is defined as visual acuity measured without any spectacles or contact lenses (i.e., with "lenses off"). It can apply to eyes that have had refractive surgery and those that have not. The unaided visual acuity becomes a benchmark against which the benefits of using a refractive correction may be referred. Care must be taken to ensure that the patient does not squint or narrow the palpebral aperture to reduce the blur created by defocus or optical irregularity. Unaided acuity is relevant when predicting how well or how poorly patients can see if deprived of access to their refractive correction. Dimming the ambient illumination causes pupil dilation, which is likely to reduce the uncorrected visual acuity when there is uncorrected refractive error or optical irregularity.

In the past, the term *uncorrected visual acuity* had been widely used to mean the same thing as unaided visual acuity; however, when refractive error has been corrected by refractive surgery, the term *uncorrected visual acuity* literally means the visual acuity without spectacle or contact lenses before the surgical intervention. After the surgery, vision is of course no longer uncorrected. This type of unaided acuity must be measured before the surgery; if recorded, it can be a useful reference against which the visual acuity benefits of the surgery may be quantified.

Habitual visual acuity is defined as the visual acuity measured under the refractive conditions that the patient habitually uses when performing distance vision tasks of daily life. Whether or not the current spectacles, contact lenses, or postsurgical refractive status are optimal corrections of the refractive error is irrelevant. The question is simply, "What is the visual acuity habitually being obtained by the patient?" The habitual visual acuity becomes a benchmark against which the benefits of changing the refractive correction may be compared.

For patients who do not usually wear eyeglasses or contact lenses for distance vision, the habitual visual acuity is simply the unaided visual acuity. Often the optical corrections being worn will be ideal or close to ideal, but it is not uncommon for patients to be wearing optical corrections that are distinctly inappropriate; these may include old corrections prescribed many years ago or spectacles obtained at a flea market, from a relative, or over the counter. Sometimes patients will be habitually combining two or more different means of refractive correction. For example, spectacles may be used over contact lenses to correct residual astigmatism, and spectacles may be used to provide an additional improvement in visual acuity after refractive surgery. Many persons who use monovision created by contact

lenses, refractive surgery, or natural anisometropia will choose to wear spectacles for tasks such as driving and watching television.

Corrected visual acuity is defined as the visual acuity obtained with the patient wearing the best available refractive correction obtained by conventional spectacle lenses or contact lenses (i.e., with "lenses on"). The best available refractive correction is usually established by determining the best spherocylindrical spectacle correction over and above any other refractive correction that may be present, such as contact lenses or refractive surgery. Thus, the corrected visual acuity could be obtained with a full optical correction in the form of a spectacle lens or with the combination of a spectacle lens over a contact lens. In the case of refractive surgery, a spectacle correction over the surgically modified eye—and perhaps even in combination with a contact lens—could provide the best available correction.

If there are no significant optical irregularities or opacities, the corrected visual acuity indicates the best resolution achievable by the patient's visual system. In the presence of corneal surface irregularities (e.g., keratoconus, traumatically induced corneal distortion, some cases of distortion resulting from refractive surgery), a spectacle correction over a rigid contact lens might be necessary to provide the best measure of corrected visual acuity (see Chapter 34).

The corrected visual acuity provides a benchmark or reference for determining whether visual acuity has changed as a result of disorders affecting the optical or neural components of the visual system. Changes in corrected visual acuity can be critically important when making diagnoses, when determining whether additional vision loss has occurred, and when deciding whether changes should be made to eye disease treatments.

During the past decade, there have been significant advances in the technology required to measure and correct optical aberrations of the eye (see Chapter 19). There is some promise that optimizing the quality of the retinal image may lead to measurable and significant improvements in the visual acuity as compared with that which can be obtained through the use of conventional spectacle lenses and contact lenses. Optimal control of aberration might be achievable by surgical shaping of the corneal surface or through having individualized asphericity built into the surface configurations of the patient's spectacle lenses, contact lenses, or intraocular lenses.

Optimal visual acuity can be defined as the visual acuity that will be obtained when the optical quality of the retinal image is optimized. It is a long established and common clinical practice to use the term "best-corrected visual acuity" to mean the acuity that is obtained with best correction in the form of conventional spherocylindrical lenses. Thanks to technological advances in the control of higher-order aberrations, the concept of what is "best correction" is changing. Although "best"

and "optimal" may be synonyms, it seems appropriate to use "optimal visual acuity" to refer to the very best possible visual acuity and to possibly discourage the future use of the term "best-corrected visual acuity" to avoid confusion between the old and new meanings of "best correction." The term "best-corrected spectacle acuity" is now sometimes used to indicate the best acuity derived from the wear of the spectacle refraction.

Pinhole acuity refers to visual acuity measured using pinhole apertures (usually having a diameter of 1.0–1.5 mm) placed before the patient's eye to determine whether a reduced visual acuity is a result of optical defects. The pinhole increases the depth of focus so that the blur created by optical irregularities or refractive error becomes reduced; consequently, visual acuity improves. Pinhole tests are used when the best corrected visual acuity is poorer than expected or when there is reason to suspect optical irregularities. A pinhole can be expected to improve visual acuity in patients with keratoconus or with cortical or posterior subcapsular cataracts, because it can channel light through a better region of the eye's optics. Defocus and optical irregularities become less important as depth of focus is increased. However, a pinhole should not have any significant impact on visual acuity that is reduced because of amblyopia or some retinal disorders. The pinhole does reduce the illuminance of the retinal image; through this mechanism, the pinhole may sometimes reduce visual acuity, especially in patients with retinal diseases that make visual performance particularly sensitive to changes in retinal illumination.

The Potential Acuity Meter (PAM) by Marco Ophthalmic Instruments (Jacksonville, Fla) is an instrument that presents an image of a visual acuity chart to the eye using a Maxwellian view optical system. Maxwellian view, explained in Chapter 1, confines the beam entering the eye to an area that is smaller than 1 mm at the plane of the pupil. In theory, using the PAM is similar to using a pinhole, except there is better control of the retinal illumination.⁴⁰

Visual acuity measurement under special illumination conditions may be indicated to evaluate the potential functional difficulties that depend on illumination conditions. Bright conditions cause pupil constriction, and this may have adverse effects on visual acuity in cases of centrally located optical opacities or irregularities. On the other hand, more peripherally located optical defects—as often occur after refractive surgery—might cause a visual acuity reduction when illumination is reduced and the pupils dilate to expose the regions of optical irregularity. In some patients (especially those with retinal disease), visual performance may be strongly affected by retinal illuminance, and the clinician may choose to vary the chart luminance over a wide range to identify and quantify the patient's specific lighting dependencies.

Testing Distance

In a given clinical setting, a standard testing distance is established. It may be a specific distance such as 20 feet, 6 m, or 4 m to facilitate scoring in Snellen notation. With projector displays, the test distance is usually chosen according to spatial constraints of the examination room. Most examination rooms are too short to allow a direct observation path of 20 feet (6 m), so mirrors are used in both the projection and observation paths to achieve longer testing distances. In most circumstances, the test distance is close to 20 feet (6 m). Although variations from the "standard" are not uncommon, they rarely fall outside of a 10- to 30-foot range.

If patients cannot read all letters at the largest angular size available at the standard distance, a shorter test distance should be used. Short test distances are most easily achieved using printed panel charts. For patients with very low visual acuity, close distances such as 5 feet, 1 m, 1 foot, and 40 cm might be considered. When patients cannot read the largest letters available on a projected chart, a printed panel chart should be used.

When close test distances are used, it may be necessary to modify the refractive correction by adding the appropriate plus lens power to ensure optimal focus on the retina. If a plus lens is used to cause the image of the chart to be at optical infinity, some presbyopic patients might not achieve their best possible acuity, because proximal accommodation may create some defocus. Proximal accommodation is only of potential significance when testing patients with good visual acuity at distances closer than 10 feet. If short viewing distances are adopted to enable the testing of patients with very low vision, it is not usual to make any refractive compensation; modest levels of defocus are not likely to affect the legibility of their threshold-size letters, because they are so large in angular size.

Testing Procedure

Monocular visual acuities are tested with one eye viewing the test chart while an occluder is placed before the other eye. If the hand of the patient or the clinician is being used to occlude the other eye, care should be taken to use the palm, because otherwise the patient might look through a narrow gap between the fingers. Usual practice is to measure the right eye first, but the left eye might occasionally be measured first if it is known that the patient has poorer vision in that eye. At almost every eye examination, the visual acuity of the right eye (OD) and the left eye (OS) are measured separately. Typically, clinicians measure the binocular (OU) visual acuity as well. This is measured with both eyes open, and it is usually expected that the binocular visual acuity will be marginally better than—or at least equal to—the visual acuity of the better eye. Rarely is the

binocular visual acuity poorer than the better of the two monocular acuities. This may happen in some cases of binocular vision disorders, nystagmus, or metamorphopsia, and it can occur in monovision when the patient is unable to alternate central suppression from one eye to the other (see Chapter 28).

Some clinicians ask patients to read from the largest letters at the top of the chart through to the smallest that can be read. More commonly, the patient is asked to begin reading at a size level that is expected to be a little larger than patient's resolution limit. For example, a patient expected to have an acuity of 20/20 or better might be asked to begin at the 20/40 level. The patient is instructed to read down the chart as far as possible. There is often a hesitancy that indicates that the patient is earnestly struggling to read as many letters as possible. For clinical testing, it is common practice to ignore an occasional error if all letters at the next smallest size are read correctly. When reading letters at sizes close to threshold, the patient should be encouraged to guess. One widely used rule is that, if patients correctly identify 50% or more of the letters correct at a given size (e.g., three of five letters), they should be obliged to guess the remaining letters at that size level and then to guess at all letters at the next smallest size. Carkeet⁴¹ modeled visual acuity responses and supported guessing when 40% or more of the letters were read correctly at the previous size level. When there was a high probability of guessing correctly (e.g., as with four-position Landolt rings), a more stringent 20% criterion (i.e., one out of five) was deemed appropriate. To ensure that the patient has been tested at sizes both larger and smaller than the threshold size, all optotypes at a larger size should be read correctly, and no optotypes at the smallest size should be read at all.

Special problems sometimes arise in patients with disorders affecting macular function. Patients with macular scotomas may miss letters at many different size levels, and patients with amblyopia may behave similarly. There may be a tendency to completely miss letters at the start or end of rows. Some patients appear to have to search for individual letters, and they may name the letters out of sequence. The clinician may help such patients keep their bearings by pointing to individual letters. Eccentric viewing helps some patients with macular scotomas achieve better visual acuity scores. The clinician may encourage eccentric viewing by having the patient look above, below, to the right, and to the left of the letters being read; this may improve visual acuity performance. Patients with amblyopia or macular disorders are likely to achieve better resolution if presented with isolated single letters rather than a series of letters in a row or chart.

Flip charts are sometimes used to isolate different size levels and to facilitate the isolation of individual letters. With a view to expediting testing, Rosser⁴²

designed charts with an abbreviated size range and fewer letters per row. Camparini and colleagues⁴³ recommended having the patient read only the first letter in each row until difficulties or errors were encountered. These quick testing procedures will generally reduce the reliability and validity of the test results.

Assigning Visual Acuity Scores

Row-by-Row Scoring

Unfortunately, it is a common practice to assign a visual acuity score on a row-by-row basis. The visual acuity score records the smallest size at which at least a specific proportion (typically 50%, but up to 80%) of all of the letters of that size are correctly identified. The possible scores correspond with the size levels on the chart. Scoring row by row is too coarse to reliably detect small changes in visual acuity. For example, when using a chart with five letters per row, a one-row change in visual acuity score could be caused by as little as a one-letter difference or as much as one letter short of two full rows. With row-by-row scoring, the visual acuity score must change by at least two size levels for clinicians to be confident that there has been a significant change.³² Despite its relative insensitivity, this is the method that remains the most widely used by eye-care practitioners.

Many clinicians do give partial credit, qualifying a visual acuity score by adding plus or minus signs to indicate that the patient actually did a little better or a little worse than the performance indicated by the numerical value recorded. A patient reading all letters in the 20/25 (6/7.5) row and correctly identifying two letters on the 20/20 row could be given a score of 20/25⁺² (6/7.5⁺²).

Letter-by-Letter Scoring

Giving credit for every letter read provides more sensitivity for the detection of changes in acuity. Clinicians may record a visual acuity score followed by a plus sign with a number to indicate the number of letters read at the next smallest size or a minus sign with a number to indicate the number of letters missed at that size level; for example, 20/25⁺², 20/25⁻¹, 20/30^{-1,+2}. If the chart has the same number of letters on each row, the qualifiers (e.g., ^{-2,+1,-1,+2}) carry the same value at all levels of the chart. By giving credit for every letter, 20/25⁺¹ can be considered equivalent to 20/25^{-1,+2}. If the number of letters at the different size level varies throughout the chart, the weight given to the qualifying number depends on the specific number of letters in the rows concerned.

If visual acuity is being recorded in logarithmic units (logMAR or VAR), each letter can be assigned a value that is added to the score when that letter is read correctly. On charts with five letters per row and a size pro-

gression of 0.10 log units, each letter can be assigned a value of 0.02 logMAR units. For each additional letter read, 0.02 is deducted from the logMAR score. Similarly, scoring in VAR units gives a value of one point per letter so that each extra letter read adds one extra point to the score. Table 7-4 provides three examples of how letter-by-letter scoring can be used to give scores in terms of logMAR, VAR, or Snellen fractions with qualifiers. For these three examples, it has been assumed that the chart complies with the Bailey–Lovie design principles so that each letter carries equal value. Table 7-5 also shows how credit can be assigned for each letter, even when charts do not have a regular size progression and the number of letters varies per row. Then, for each size level, the per-letter value in logMAR or VAR units is determined by subtracting the logMAR or VAR values for that row from that of the proceeding row and dividing this difference by the number of letters.

Visual Acuity Measurement in Research

In many research projects involving visual acuity measurement, visual acuity tests are likely to be administered frequently, and more sampling is likely to be required. When only one or a small number of charts is available, there can be problems caused by patients memorizing letter sequences, particularly in the threshold region. There are several ways to reduce or eliminate this problem; these include having more charts available or using modest variations in the test distance so that the patient's resolution threshold moves to a new region of the chart. For charts that use British or Sloan letters with five letters by row, chart pairs can be designed so that there is no replication of any of the 10 letters at each of the size levels. This allows for the presentation of 10 letters at each size without there being any repeats of letters that might be more difficult or easier for the patient. Computer generation of new letter sequences provides a good solution to problems that result from patients memorizing letters or sequences.

When visual acuity is being measured for research purposes, it is important to have the testing conditions and procedures rigidly defined. Standard refraction procedures may be required. Testing conditions such as chart luminance and contrast, viewing distance, and criteria for changing to alternate viewing distances should be specified. There should be standard instructions for advising the patient that the chart contains letters only, that all letters should be attempted, reading should be at a steady pace, and that guessing is permitted. The examiner should not point to individual letters or rows, all errors should be recorded, and patients may not make correct a response once the next letter has been read. Procedures to encourage guessing and rules for stopping should be applied when the visual acuity threshold is approached.

TABLE 7-4 Letter-by-Letter Scoring of Visual Acuity in Units of LogMAR, VAR, and Snellen Fractions for an ETDRS or Bailey-Lovie Chart

SIZE LABELS ON CHART		PATIENT A		PATIENT B		PATIENT C			
LogMAR	VAR	Snellen	No. correct	LogMAR	VAR	No. correct	LogMAR	VAR	
0.40	80	20/50	6/15	5 of 5	0.40	80	5 of 5	0.40	80
0.30	85	20/40	6/12	5 of 5	0.30	85	5 of 5	0.30	85
0.20	90	20/32	6/9.5	5 of 5	0.20	90	4 of 5	0.22	89
0.10	95	20/25	6/7.5	1 of 5	0.18	91	2 of 5	0.18	91
0.00	100	20/20	6/6	0	0.18	91	0	0.18	91
-0.10	105	20/16	6/4.8	0	0.18	91	0	0.18	91
Each additional letter adds -0.02 to the LogMAR score and 1 to the VAR score.		Snellen = 20/32 ⁺¹		log MAR = 0.18 VAR = 91		log MAR = 0.18 VAR = 91		log MAR = -0.04 VAR = 102	
<i>Three examples of scoring visual acuity letter-by-letter on a chart with five letters per row and a 0.1 log unit size progression.</i>		Snellen = 20/32 ⁺¹ or 20/32 ⁺²		Snellen = 20/32 ⁺¹ or 20/32 ⁺²		Snellen = 20/20 ⁺¹ or 20/20 ⁺²		Snellen = 20/20 ⁺¹ or 20/20 ⁺²	

Three examples of scoring visual acuity letter-by-letter on a chart with five letters per row and a 0.1 log unit size progression.

TABLE 7-5 Letter-by-Letter Scoring of Visual Acuity in Units of LogMAR, VAR, and Snellen Fractions for a "Standard" Snellen Chart

SIZE LABELS ON CHART		PATIENT X		PATIENT Y	
Snellen	LogMAR	No. of Letters per Row	LogMAR per Letter	VAR	VAR
20/200	6/60	1.00	50	1	1.00
20/100	6/30	0.70	65	2	-0.15
20/70	6/21	0.54	73	3	-0.05
20/50	6/15	0.40	80	4	-0.04
Each additional letter carries a value that depends on the number of letters in that row and on the increment of size.		log MAR = 0.85 VAR = 58		log MAR = 0.65 VAR = 68	
<i>Two examples of scoring visual acuity letter-by-letter using a traditional Snellen chart.</i>		Snellen = 20/100 ⁺¹ or 20/200 ⁺¹		Snellen = 20/100 ⁺¹ or 20/200 ⁺²	

S-Charts

The S-chart test is a Landolt ring test designed by Flom and colleagues^{34,35} for clinical research. The S-chart test consists of a series of 35-mm slides. Each slide contains a panel of 25 symbols arranged in a five-by-five square grid. The 16 external symbols and the central symbol are all tumbling Es in randomized orientations. Forming a square around the central E is a series of eight Landolt rings, with their gap orientations (right, left, up, down) arranged randomly. The observer's task is to begin with the top left Landolt ring and progress in a clockwise direction, naming the location of the gap for each of the eight Landolt rings. Each 35-mm slide presents a new size. Originally the size progression followed the Snell-Sterling visual efficiency scale, but later a logarithmic size progression was used. Visual acuity score was determined by fitting a sigmoid or S-shaped psychometric function to a plot of percent letters correct to letter size. Probit analysis methods achieve the same result. With four alternative gap orientations, chance performance is represented by 25% correct. Consequently, the print size that allows 62.5% of letters to be read correctly represents the 50% probability of seeing point. The 50% probability of seeing point is commonly taken as the threshold for the visual acuity value.

PEDIATRIC TESTS OF VISUAL ACUITY

A wide variety of visual acuity tests are available for measuring visual acuity in infants, toddlers, and others who have a limited ability to respond to standard test stimuli.⁹ The clinician selects a visual acuity test that is appropriate given the response capability of the patient. There is a hierarchy of tests,⁴⁴ ranging from visually evoked potentials for the least responsive patients and progressing to techniques of preferential looking; observation of optokinetic nystagmus; and responses to picture flash card tests, picture charts, symbol and letter flash cards, symbol and letter charts, and reading charts.

Grating Acuity Tests

Striped or checkered grating targets are used in some of the more elementary tests of visual resolution in infants. The size of the detail is varied to determine the finest pattern that can elicit a response from the infant. The patient's responses may be determined by objective or subjective means. The angular size of the detail within this pattern is expressed as cycles per degree, and it is taken as the visual acuity. Within a grating pattern, one cycle embraces a dark and a light stripe. An equation used to convert grating acuity score (cpd) to MAR is as follows:

$$\text{MAR} = 30/\text{cpd}$$

For a 30-cpd grating, each cycle is one-thirtieth of a degree, so one cycle is 2 minarc. Each dark and each light stripe is 1 minarc. Thus, a 30-cpd grating has a nominal equivalence to 20/20 or 6/6 (MAR = 1 minarc), a 3-cpd grating is equivalent to 20/200 or 6/60 (MAR = 10 minarc), and so forth.

Visually Evoked Potential Tests

Visually evoked potential tests involve measuring electrical potentials from the back of the head as the patient looks toward a screen on which flickering striped or checkered patterns are presented. The magnitude of the electrical response declines as the detail within the pattern is made finer. The size of the detail (expressed as spatial frequency in cycles per degree) in the finest target that evokes a measurable response is taken as the visual acuity.⁴⁵

Preferential Looking Tests

These tests evolved from research procedures developed by Dobson and Teller⁴⁶ and McDonald and coworkers⁴⁷ for studying the development of vision in infants. The infant is presented with two target areas: one containing a black-and-white spatial pattern, the other containing uniform gray. The clinical adaptation of the laboratory technique uses Teller Acuity Cards,⁴⁷ which are large, rectangular, gray cards with a black-and-white striped grating pattern off to one side. When the card is presented to the patient, the clinician observes whether the patient (usually an infant) moves the eyes to fixate to the side with the grating. Within a test card series, there is a progression of spatial frequency (fineness of grating), and the task of the clinician is to determine the spatial frequency of the finest grating that still attracts the patient's attention.

Cardiff cards⁴⁸ are somewhat similar in concept to the Teller cards. Each Cardiff card presents a line drawing of an object. The picture is formed by a line that consists of a central white line with finer black flanking lines on either side. The luminance averaged across the black-white-black line matches the luminance of the gray background. Consequently, when the lines are too fine to be individually resolved, they become indistinguishable from the gray of the background. The clinician determines the finest line drawing that still attracts the child's attention.

Optokinetic Nystagmus

Striped patterns are presented on a video screen, on a rotating drum, or by other methods, and they are moved in one direction in front of the patient. If the striped pattern is visible, the patient's eyes will make "railroad nystagmus" eye movements as they follow the movement of the stripes. The clinician determines the size of the finest grating having motion that elicits the nystagmus response when it is moving.

Flash Card Tests

Flash cards with pictures or symbols as targets can be used for patients who have some ability to respond to instructions. Patients may be asked to point to or to name pictures or symbols on flash cards, to find a matching target, or to play other matching games. At the simplest end of the range of such tests is the Bailey–Hall cereal test,⁴⁴ in which pairs of flash cards are presented. One card of the pair shows a picture of a cereal ring (Cheerio), whereas the comparison card simply has a square. The patient is asked to identify which card has the picture of the cereal ring. On successful identification, the patient is rewarded with a piece of cereal to eat. The size of the picture of the cereal ring and the furthest distance at which it can be recognized provide the basis for the visual acuity estimate. More advanced tests involve the use of alternative pictures or symbols that are to be identified. The LH symbols⁴⁹ are an example of such optotypes. The four alternative LH symbols, to which patients can apply their own name, are most commonly called square, apple, circle, and house. Other flash card series similarly call for identifications or matching responses; these include Lighthouse flash cards (umbrella, apple, house); the broken wheel test,⁵⁰ which requires distinguishing a car with wheels shown as Landolt rings; and the Allen picture cards, which contain a series of simple line drawings of easily named objects.

Letter Flash Cards

There are several alternative series of letter flash cards for which the patient is required to name the target letter or letters or to make a match.^{9,51} Some use selected letters, including the mirror-reversible H, O, T, and V; others use tumbling E or Landolt ring optotypes for which the patient is required to identify the orientation of the symbol. The HOTV set of optotypes is used in the computerized display of the Baylor-Visual Acuity Tester (B-VAT) by Mentor O&O (Norwell, Mass).⁵²

Picture or Symbol Charts

Picture or symbol charts present an array of simple drawings of objects or simple symbols of progressively decreasing sizes, and the targets are to be named by the patient. Such charts are available with LH symbols that include different contrasts to assess contrast sensitivity; others have different spacing arrangements, which are used to identify problems with symbol crowding.

Letter Charts for Children

If the child is capable of making the appropriate responses, it is preferable to use adult charts with letters or equivalent optotypes arranged in the usual chart format. Similarly, if the child is capable of reading,

there may be value in performing reading acuity tests using typeset materials as the test target. Some special series of reading tests using simpler words have been developed for children (e.g., the Sloan reading card series).⁵³

NEAR VISUAL ACUITY

Near visual acuity is measured at distances within arm's length. A testing distance of 40 cm is usually considered to be the standard. If the test chart design and the luminance levels are comparable, the near visual acuity score should be equal to the score of distance visual acuity, provided that the eye is accommodated or optically corrected to provide good focus for the retinal image. However, there are rare exceptions, such as patients with posterior subcapsular cataract whose pupil constriction at near-vision tasks causes the pupil area to become more completely filled with the cataract so that the visual acuity becomes degraded. Most of the tests of near visual acuity do not use letter chart formats that are comparable with the charts used for testing at distance. Usually, the near vision tests use typeset material that is similar in style to the print of newspapers and books. The material may be arranged in sentences or paragraphs or in series of unrelated words.

Designation of Near Visual Acuity

Specification of near visual acuity usually includes the recording of both the observation distance and the size of the smallest print that can be read. Several different methods are used to specify the size of print in near-vision tests; the relationships among them can be seen in Table 7-2.

M Units

M units are a measure of print size introduced by Sloan and Habel.⁵⁴ They are used to specify the size of print by indicating the distance in meters at which the height of the smaller letters (the lowercase x-height of typeset print) of the printed material subtends 5 minarc. Print that is 1.0-M units subtends 5 minarc at 1 m; accordingly, it is 1.45-mm high. Regular newsprint is usually about 1.0 M in size. Visual acuity may easily be recorded as a Snellen fraction in which the clinician records the test distance in meters in the numerator, and the denominator indicates the M-unit size of the smallest print that can be read at that distance. A patient who can just read 1.0-M print at 40 cm would have his or her visual acuity recorded as 0.40/1.0 M. Jose and Atcherson⁵⁵ pointed out that the M-unit rating of a sample of print can easily be estimated by measuring the height of the smallest letters in millimeters and multiplying this number by 0.7.

Points

Points are units used to specify the size of typeset print and are used in the printing industry; one point is equal to $\frac{1}{72}$ of an inch. The point size of a specimen of print essentially indicates the size of the print extending from the bottom of the descenders (as in letters g, j, p, q, y) to the top of ascenders (b, d, f, i, j, k, l, t). For print styles that are most commonly used for newspaper text, the height of the smaller lowercase letters (a, c, e, m, n, o, r, s, u, v, w, x, z) is about half of the total height. Newsprint is often 8 points in size, so the x-height is about 4 points. Because $\frac{1}{72}$ inches is equal to 1.41 mm, 8-point print in a newsprint style font can be given an M-unit rating of about 1.0 M. Thus, for print in font styles similar to common newsprint, the M-unit rating for the lower case letters can be estimated by dividing the point size by 8. Capital letters and numbers are taller than the lowercase letters (often by 1.5 times), and, for these larger characters, 8-point print is equal to 1.5 M rather than to 1.0 M. Common sans-serif fonts such as Helvetica have ascenders and descenders that are smaller as a proportion of the overall size. Consequently, for samples of print at the same point size, the x-height will be larger for Helvetica than for Times Roman. Fonts presented on computer screen displays usually have their sizes expressed in points, which refer to the size when the document is printed as hard copy. The size on the display screen varies with screen size and pixel density. It is handy to remember the following:

$$1.0 \text{ M units} = 1.45 \text{ mm} \approx 8 \text{ points (lowercase, newspaper style)} \approx \text{typical newsprint}$$

N Notation

To standardize the testing of near vision, the Faculty of Ophthalmologists of the United Kingdom^{56,57} adopted the Times New Roman font as the standard font for testing near vision, and they recommended that the print size be indicated in points. The size label "N8" indicates that the standard near test font is being used and that the size is 8 points. The near visual acuity performance is recorded as the smallest print that can be read (recorded in N notation), and the distance is specified (e.g., N8 at 40 cm). A print size recorded in N-notation can be converted to M-units by dividing the number by 8 (e.g., N20 = 2.5 M-units).

Equivalent Snellen Notation

Equivalent Snellen notation (also known as "reduced Snellen") is widely used to indicate the size of print used in testing vision at near. Equivalent or reduced Snellen notation ostensibly expresses the distance visual acuity value that is mathematically equivalent to the near visual acuity. Usually—but not always—a standard test distance of 40 cm is assumed. Thus, a specimen of print

of size 1.0 M presented at 40 cm might be labeled as 20/50 equivalent, because the Snellen fraction 20/50 is equal to 0.40/1.00. At any other test distance, this same 1.0-M print no longer has the same equivalence. Unfortunately, it is a relatively common practice to use the equivalent or reduced Snellen notation as a measure of the height of the test print.⁵⁸ Clinicians who use the equivalent Snellen system might record, for example, "20/50 at 20 cm" to indicate that 1.0-M print (equivalent to 20/50 when the chart is at 40 cm) is being read at 20 cm. This visual acuity performance is actually equivalent to 20/100, and it could be more appropriately recorded as 0.20/1.00 M. Despite its widespread use, "equivalent" or "reduced" Snellen notation is inappropriate for specifying the size of print used for testing near vision. First, it is inappropriate to use an angular measure (Snellen fraction) to specify the height of letters. Second, it is inappropriate to use a term that suggests a test at 20 feet when this distance is not relevant to the near-vision test distance or to the size of the print.

Jaeger Notation

Jaeger notation indicates the size of print by using the letter J followed by a number, and it is widely used, mainly by ophthalmologists. The near visual acuity is indicated by recording the print size and the test distance (e.g., J3 at 40 cm). Unfortunately, there is no standardization of the Jaeger sizes; hence, there is no intrinsic meaning to the number that indicates the print size. Sizes J1 to J3 usually indicate that the print is small, and J5 to J8 indicate fairly small print. Jose and Atcherison⁵⁵ found that, among charts from different manufacturers, there can be as much as a twofold difference in the sizes of print samples that carry the same Jaeger size label. The Jaeger notation should not be used for measurements of visual acuity, but it remains popular in the ophthalmological community.

Acceptable Size Notations for Testing Near Vision

M units and points specify the height of letters, and both are satisfactory. M units have the advantage that they can be used in the traditional Snellen fraction format, which specifies the testing distance and the print size; points have the advantage that they are well known and widely used outside of the ophthalmic professions. Visual acuity is an angular measure, and its specification using M units or points requires that the testing distance be given as well (e.g., 6 points at 40 cm, or 1.5 M at 40 cm). When using point sizes, test material in capital letters should be specified. Otherwise, it should be assumed that the height of the lowercase letters is the critical size dimension.

Currently there is no international standardization of the font style that should be used for testing reading

acuity, but the Times font has long been the established standard in the United Kingdom. Most regular newsprint is in Times or in fonts of similar styles, and it seems reasonable to use such fonts for clinical testing, because they are representative of everyday reading tasks.

Reading Acuity and Letter Chart Acuity

Letter chart acuity at near is directly comparable with letter chart acuity at distance. Reading acuity is a more complex function. Reading acuity tests use typeset print as the test target and the resolution of more congested and complex components arranged in sequences that must be recognized. Patients with disorders affecting macular function (e.g., age-related macular degeneration, amblyopia) are likely to have a reading acuity that is significantly worse than the letter-chart acuity. Task complexity can have a substantial effect on the visual acuity scores. Kitchin and Bailey⁵⁹ studied a group of subjects with age-related macular degeneration and found that visual acuity scores showed substantial differences depending on the task complexity. Their results are summarized in Table 7-6; it can be seen that, for this group of subjects, there was a fivefold difference between their averaged grating acuities and their reading acuities.

Reading charts come in a wide variety of formats. Most use a series of passages of text or simple sentences with print size diminishing with each successive passage. Some charts use a series of unrelated words,^{60,61} because this avoids context being used to help the patient guess at the word. Charts that require the reading of sentences or passages^{62–64} are obviously more

representative of real reading tasks, in which context and syntax contribute to reading accuracy and efficiency. Word reading charts can be said to test the ability to see to read rather than the ability to read.

Near Visual Acuity Versus Near Vision Adequacy

Many tests said to be near visual acuity tests are not really used to measure visual acuity but rather near-vision adequacy. A visual acuity test determines the angular size of the smallest print that a patient can read. A test of near visual acuity requires that some of the presented material be beyond the patient's limit. On most reading charts, the smallest letters are 0.4 M or larger (0.4/0.4 M is equivalent to 20/20 or 6/6). When being tested with such charts at the usual 40-cm test distance, patients with normal visual acuity can be expected to read the entire near-vision chart at 40 cm, and, consequently, their resolution limit is not established. On reading charts, the progression of print size should extend to 0.25 M (2 points) or smaller if visual acuity is to be measured at a test distance of 40 cm (0.25 M at 40 cm is equivalent to 20/12.5 or 6/3.8 performance). Although tests of near vision adequacy should not be confused with tests of reading acuity, they are nevertheless useful, because they do demonstrate that the patient is capable of reading print that is quite small.

Visual Acuity and Resolution Limit at Near

Sometimes it is important for the clinician to distinguish between near visual acuity and the patient's resolution limit. Near visual acuity is determined from the smallest print that can be read when the retinal image is in good focus. The resolution limit simply determines the size of the smallest print that can be read without the requirement of a sharp retinal image. A low-vision patient with a 2.50-D addition might read 4.0-M print at 40 cm and 3.2-M print at 32 cm, with the retinal image being in sharp focus at both distances. However, this patient might have a resolution limit of 2.5 M when the print is at 16 cm. Moving the print closer to 16 cm has enlarged the retinal image, but now defocus is significant, so best visual acuity is not achieved. Reading 2.5-M print at 16 cm represents better resolution (smaller print can be read), but visual acuity is poorer than 0.40/4.0 M or 0.32/3.2 M. To predict near-vision magnification needs for low-vision patients, clinicians should rely on measurements of reading acuity taken when the retinal image is in good focus.

Reading Efficiency

As patients read the reading acuity test charts, reading efficiency decreases as the print size approaches the

TABLE 7-6 Visual Acuity and Task Complexity in Age-Related Macular Degeneration Subjects

Task for Visual Acuity Test	AVERAGE VISUAL ACUITY*	Snellen Notation
	LogMAR (SD)	
Grating display	0.61 (+/-0.11)	20/81
Single letters	0.76 (+/-0.31)	20/115
Landolt rings with masking bars	0.89 (+/-0.36)	20/155
Letter chart	0.97 (+/-0.32)	20/189
Word reading chart	1.31 (+/-0.25)	20/408

*16 age-related macular degeneration subjects.
From Kitchin JE, Bailey IL. 1981. Task complexity and visual acuity in senile macular degeneration. *Aust J Optom* 63:235–242.

patient's resolution limit. For normally sighted subjects, maximum reading efficiency is usually obtained when the print is about three times larger than the smallest resolvable print.^{37,65} When testing patients with low vision, clinicians commonly note the smallest print for which good reading efficiency can be obtained in addition to noting the smallest print that can just be read. The size limit for good efficiency and the resolution limit should both be accounted for when prescribing magnification for near vision. For normally sighted subjects, maximum reading efficiency is usually obtained when the print is about three times larger than the smallest resolvable print.^{37,65,66} This relationship holds over wide ranges of luminance and contrast.

Some near-vision test charts have been designed to facilitate the clinical measurement of reading efficiency.^{60–62} These charts allow for the assessment of changes in reading speed as a function of the print size. Reading speed slows as the threshold size is approached. Bailey and colleagues⁶⁷ pointed out that cognitive and motor demands of the reading task can affect reading speeds and the degree to which speed changes with print size. Bailey-Lovie word reading charts⁶⁰ have 17 size levels that range from 10 M to 0.25 M. For the smallest 11 rows, there are 42 letters (two words each with lengths of 4, 7, and 10 letters). At the six largest sizes, there are fewer words. The MNREAD charts of Legge and colleagues⁶² have 19 different sizes ranging from 8.0 M to 0.12 M; at each size level, there is a simple sentence that has 60 character spaces distributed about evenly over three rows.

Logarithmic Scaling of Reading Charts

If the progression of print size on a reading chart follows a logarithmic or constant ratio of progression, there are special advantages that facilitate the prescribing of near-vision additions or magnifiers for the purpose of allowing the patient to resolve—or read with efficiency—print of a particular size. Several such charts are available.^{60,62,63,68} The labels for the progression for print size on the chart can be used as a sequence of alternative viewing distances or as a sequence of alternative dioptic powers (Figure 7-5). To change the resolution limit by a certain number of size levels on the chart, the viewing distance (or dioptic distance) should be changed by an equivalent number of steps on the scale. Consider the following progression of the M-unit ratings of print sizes on a logarithmically scaled chart:

10, 8.0, 6.3, 5.0, 4.0, 3.2, 2.5, 2.0, 1.6, 1.25, 1.00, 0.80, 0.63, 0.50, 0.40, 0.32, 0.25, 0.20, and so on.

Assume that a patient can just resolve 1.6-M print and can efficiently read 2.5-M print when the chart is at

40 cm. The clinician's goal might be to enable this patient to read 1.00-M print with efficiency, and thus 0.63 M would be the resolution limit. Here, two steps of size separate the efficiency and resolution limits: first with 2.5 M and 1.6 M and second with 1.00 M and 0.63 M. Four steps of improvement are required. The efficiency limit must shift by four steps from 2.5 M to 1.00 M, and the resolution limit must also shift by four steps from 1.6 M to 0.63 M. To achieve this, four steps of change must be made to the viewing distance (and to the dioptic demand). The clinician may use the sequence of size labels on the charts as a guide to the sequence of changes in viewing distance. Since there is a four-step change in size from 4.0 to 1.6, a change in viewing distance from 40 cm to 16 cm will be a change of equal proportion. Thus, the four-step change in the viewing distance required in this example goes from a starting point of 40 cm to 16 cm. Similarly, the dioptic demand needs to be increased by four steps from a starting point of 2.50 DS to 6.25 DS. Provided that the retinal image remains in satisfactory focus, the reading resolution and reading efficiency goals for this patient can be achieved by spectacles or magnifying systems that provide an equivalent viewing distance of 16 cm or an equivalent viewing power of 6.25 DS.

PURPOSES OF VISUAL ACUITY MEASUREMENT

Refraction and Prescribing Decisions

Refractionists use visual acuity charts as test objects for refraction procedures to determine the lens power that provides the sharpest retinal image—and thus the best visual acuity—for the individual patient. Comparison between the patient's habitual visual acuity and the visual acuity with the newly determined refractive correction often influences the clinician's or the patient's judgment on the advisability of obtaining new spectacles or contact lenses. Some insurance programs require documentation of an improvement in visual acuity before they will pay for a change in optical correction. Some worthwhile changes in optical correction do not produce measurable increases in visual acuity. Depending on the patient's accommodation abilities, a correction for hyperopia might serve to relieve an excessive accommodative response while providing little or no improvement in measured visual acuity. Often, for patients with low vision, a change in refractive correction will provide a substantial improvement in perceived clarity without changing the visual acuity score.

The relationship between visual acuity and uncorrected refractive error is complex, and there is considerable variation between individuals, even when optical

**Figure 7-5**

Bailey-Lovie word reading chart with logarithmic size progression, shown at actual size. M, M units; N, N notation points; LogMAR, logarithm of the minimum angle of resolution; VAR, visual acuity rating.
(Courtesy of the National Vision Research Institute of Australia.)

factors are similar. Acuity depends on optical factors such as pupil size, the presence of astigmatism, the axis of astigmatism, and optical aberrations. Atchison and colleagues⁶⁹ studied visual acuity for wide ranges of simulated myopia and pupil size, and they tested at two different luminance levels. There was a significant reduction in uncorrected visual acuity as a function of the magnitude of the myopia and the diameter of the pupil, but both of these relationships are nonlinear. Peters⁷⁰ studied clinic records of patients in the age groups of 5 to 15 years, 25 to 35 years, and 45 to 55 years, with over 2000 subjects in each group. He analyzed uncorrected visual acuity as a function of spherical and astigmatic refractive error and their combinations. His results showed high similarity for all three age groups with regard to myopia. In hyperopia, the two younger age groups obtained better acuities; this was clearly as a result of their ability to use accommodation to fully or partially overcome spherical hyperopic error. There is an approximation or rule of thumb that is compatible with the results of Peters⁷⁰ and Atchison and colleagues,⁶⁹ as well as the results of others: up to about 2 D of refractive error, visual acuity reduces by about 0.1 log unit (one row on the chart) per 0.25 D. This means that there is a visual acuity reduction of 2.5 times (0.4 log units) for 1.00 D of spherical defocus and 6.25 times (0.8 log units) for 2.00 D. In hyperopia, young patients will likely use their accommodation to reduce the amount of apparent refractive error such that the expected visual reduction does not occur in the magnitude expected. For simple astigmatism, the visual acuity reduces at about half the rate. Thus, visual acuity will be reduced by about a factor of 2.5 times (0.4 log units) for 2.00 D of astigmatism and 1.6 times (0.2 log units) for 1.00 D of cylindrical defocus.

Monitoring Ocular Health

Many disorders affecting the optical or neural components of the visual system cause a change in visual acuity. When it is known that a vision-reducing disorder is present, monitoring visual acuity can provide a means of detecting deterioration or improvement in the condition. In many eye diseases, a change in acuity is often a major determinant of whether treatments are implemented, altered, or continued.

When it is clinically important to detect small changes in visual acuity, extra care should be taken with the measurement. First of all, the clinical test conditions and the procedures should be carefully standardized. For better sensitivity to change, the visual acuity scores should be as reliable as is practical; this usually means that acuity should be scored in a manner that gives credit for each letter read. Bailey and colleagues³² showed that, for normally sighted subjects, scoring letter

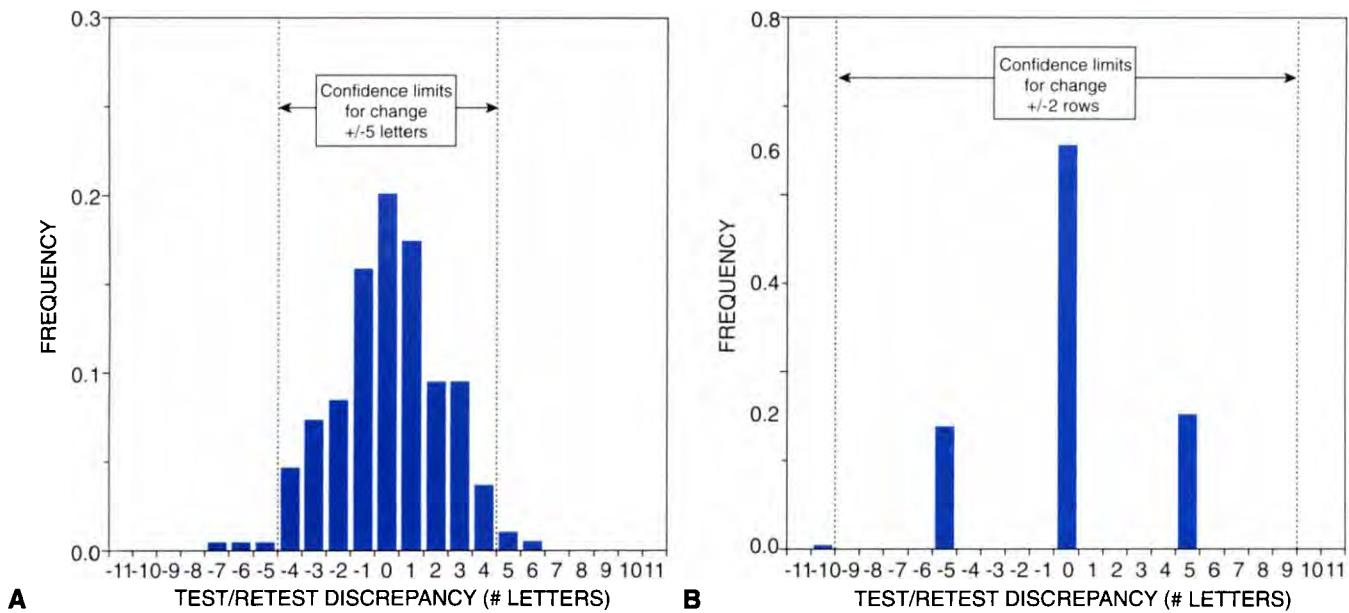
by letter gave 95% confidence limits for changes at ± 5 letters. They compared test and retest scores and found that the discrepancy between the two exceeded ± 4 letters for fewer than 5% of the comparisons. If a five-letter discrepancy between test and retest scores occurs less frequently than 5% of the time by the vagaries of sampling, it is reasonable to consider a five-letter difference sufficient evidence that a real change has occurred. In the results of these researchers, the 95th percentile discrepancy is found at ± 4 letters, so the next largest discrepancy (± 5 letters) becomes the 95% confidence limit—or criterion—for change. If visual acuity is scored row by row, the 95% confidence limit for change is determined to be ± 2 rows, because the 95th percentile discrepancy lies in the next smallest category, which is ± 1 row (Figure 7-6). This means that, when visual acuity is scored by row, at least two rows of change must occur before the clinician can determine that there has been a real change in acuity. Thus, there is a twofold difference in the confidence limits (five letters versus two rows = 10 letters) when scoring letter-by-letter rather than scoring row-by-row (without adding qualifiers to indicate how many letters were read).

Visual Acuity for Normalcy

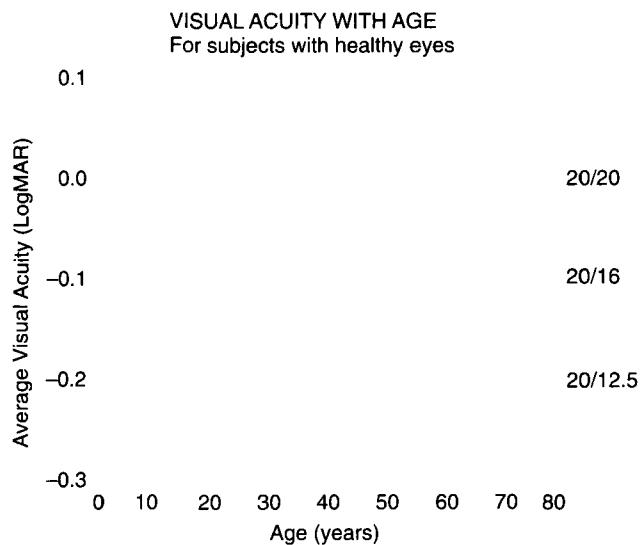
Although 20/20 (6/6) is commonly held to represent normal vision, most normally sighted persons have acuity that is measurably better than 20/20; the traditional 20/20 is more a limit at the poorer end of the normal range. Brown and Lovie-Kitchin⁷¹ emphasized that, when evaluating a given patient, it is important to establish the patient's individual baseline visual acuity and reliability against which subsequent measurements can be compared. For patients with normal or near-normal vision, five letters on a Bailey-Lovie or ETDRS chart is a reasonable criterion for identifying change.

Elliott and colleagues⁷² presented a meta-analysis of data that they had collected in different experiments, and they showed a systematic decrease in visual acuity with age (Figure 7-7). For inclusion in their analysis, they selected only subjects who had no significant ocular disorders and no substantial reduction in acuity. For their groups younger than the age of 50 years, the average visual acuities were better than 20/16 (6/4.8); along with the reported standard deviations, it was seen that, at least up to age 50, visual acuity was expected to be better than 20/20 (or 6/6). Even for subjects who were more than 75 years old, the average best-corrected visual acuity was slightly better than 20/20 (6/6).

Visual acuity scores in patients with significant disorders affecting vision are likely to be less reliable. Depending on the cause of the condition and on individual factors, it can become more difficult to set the confidence limits for change. Taking visual acuity meas-

**Figure 7-6**

A, Distribution of test-retest discrepancies when scoring visual acuity letter by letter. B, Distribution of test-retest discrepancies when scoring visual acuity row by row.

**Figure 7-7**

Visual acuity with age for healthy eyes. (Adapted from Elliott DB, Yang KCH, Whitaker D. 1995. Visual acuity changes throughout adulthood in normal, healthy eyes: seeing beyond 6/6. Optom Vis Sci 72:188.)

urements using letter-by-letter scoring at each clinical visit allows the clinician to accumulate data to identify and document reliability characteristics for the individual patient; real change is then identified when a visual acuity score is significantly outside the range of the usual "noise" in visual acuity scores for that patient.

Comparison between the visual acuity scores for the two eyes can also be useful for identifying deviations from normalcy.⁷³

Visual Acuity Measurement Applied to Vision Standards

Clinicians are often required to provide visual acuity scores that will be used by others to determine whether the patient meets eligibility standards specified for certain occupational tasks, for licenses, or for certain benefits. Visual acuity measurements have been used as the basis for determining the amount of financial compensation in insurance claims or legal suits involving a loss of vision.

Chart design, testing distance, scoring method, and test procedures can significantly affect acuity scores and the consequent decisions. As an example, consider the visual acuity standard for legal blindness, which is "visual acuity should be 20/200 or less." It is usually easier for a patient to meet this criterion if a "standard Snellen chart" is used at a test distance of 20 feet. Such charts have a single letter at the top 20/200 level, there are two letters at the next 20/100 level, and then there are progressively more letters at the smaller size levels on the chart. The patient is given a score of 20/200 for reading the largest letter but failing to read the pair of letters that constitute the 20/100 row. On such a chart, the "20/200 or less" criterion effectively becomes "fails to achieve a visual acuity of 20/100." Decisions might change if this chart were presented at 10 feet. A patient

who did not achieve the 20/100 acuity level might obtain a visual acuity score of 10/70. This performance is equivalent to 20/140, so the patient would no longer meet the legal blindness requirement. An apparent but artifactual improvement in visual acuity has occurred as a result of the chart design. On the other hand, a patient who might have been able to read the two letters at the 20/100 level might be unable to achieve equivalent success when the chart is presented at a 10-foot distance, because the ostensibly equivalent task (10/50) is likely to involve more letters that are more closely spaced and, as a consequence, less legible. If testing for legal blindness is performed with a chart that uses a 0.1 log unit size progression, the next size smaller than the 200-feet letters is 160 feet. To meet the definition of legal blindness, the practical criterion would now become "fails to achieve an acuity of 20/160." A patient who achieves 20/200¹ or 20/200² technically fails to meet the requirement for legal blindness; however, should the clinician use row-by-row scoring without adding qualifiers, the acuity would be recorded as 20/200, and the patient would be considered legally blind. The test chart design, the testing distance, and the scoring method can all significantly affect the visual acuity score and the decision as to whether the patient meets specified visual acuity standards. Until chart formats, scoring methods, and test conditions and procedures are specified or standardized,^{74,75} opportunities for substantial inconsistencies remain.

TOWARD STANDARDIZATION OF VISUAL ACUITY MEASUREMENT

There is still no single accepted international standard for the clinical measurement of visual acuity. Three major authoritative bodies have produced sets of similar—but not identical—principles for chart design. The authorities are (1) the National Research Council's Committee on Vision^{27,75}; (2) the Concilium Ophthalmologicum Universale, Vision Functions Committee²⁸; and (3) the International Standards Organization.^{76,77} They agreed that the standard test distance should be 4 m, that the standard optotype against which others should be calibrated is the four-choice Landolt ring, and that the size progression ratio should be 0.1 log unit (1.259×). There were some disagreements about the number of optotypes at each size, the spacing between adjacent optotypes, and the spacing between adjacent rows. The recent British Standard 4274-1⁸ unfortunately introduced new "standard" optotypes and allowed considerable variations in the size progression, the number of letters at different sizes, the spacing between rows, and the chart luminance. It did, however, require that the space between letters be equal to the letter width. The International Standards Organization standards

were those accepted by the American National Standards Institute.

The ETDRS chart with its Sloan Letters and Bailey-Lovie layout has become the de facto standard for research in Western countries.⁷⁸ Versions are available with Landolt ring or tumbling E optotypes and characters from other languages. It seems probable that the ETDRS chart will become more consolidated as the standard for clinical research, with some variations allowed to accommodate populations that lack familiarity with the 10 alphabetical letters used in the ETDRS charts. The clinical community may slowly come to incorporate this chart design into routine clinical practice. When computerized display screens eventually become widely used for the clinical measurement of visual acuity, the flexibility of manipulating the display will create a need for decisions about chart-design parameters. Then, perhaps, the clinical community will embrace the Bailey-Lovie design principles.

OTHER APPLICATIONS OF VISUAL ACUITY TESTING

Contrast Sensitivity

Low-contrast visual acuity charts (usually light-gray letters on a white background) are sometimes used as a measure to identify changes that affect contrast sensitivity. Visual acuity is poorer when the contrast is lower. The extent to which acuity is degraded by the contrast reduction can identify patients whose general contrast sensitivity has been affected by their visual disorder. The Regan letter charts⁷⁹ are available as a series of visual acuity charts at several different contrasts, and a 10%-contrast Bailey-Lovie chart is available.^{80,81} Haegerstrom-Portnoy and colleagues⁸² produced a low-contrast chart by printing black letters in a Bailey-Lovie format on a dark gray background. This chart, which is known as the Smith-Kettlewell Institute Low-Luminance chart (SKILL chart), was found to be more sensitive for detecting changes in visual function after retinal and optic nerve disease than other tests of visual acuity and contrast sensitivity.

The Small Letter Contrast Sensitivity (SLCS) test has a series of Sloan letters that are all of the same size (5.5 M), and the contrast reduces progressively in steps of 0.1 log units.^{83,84} There are 10 letters at each of the 14 levels of contrast. As compared with standard visual acuity charts of the Bailey-Lovie design, the results of the SLCS test are more sensitive to small changes in refractive error, and they show enhanced performance under binocular viewing. Part or all of this sensitivity advantage may be the result of increased sampling. For contrast levels greater than 10%, the relationship between log contrast sensitivity and log visual acuity is

almost a linear function, with a slope of approximately 7 to 10. In other words, the contrast should be changed by 0.7 to 1.0 log units to produce a 0.1 log unit change in visual acuity. In addition, there are twice as many letters per row, so the SLCS test effectively offers a 14- to 20-fold sampling advantage. Consequently, sensitivity to differences should be increased by about four times ($\sqrt{14}$ to $\sqrt{20}$). To obtain equivalent sampling frequency with a visual acuity chart, it would be necessary to have more rows with much finer increments of size (0.01 to 0.014 log units instead of the usual 0.1 log units) and to double (from 5 to 10) the number of letters per row. Alternatively, an equivalent sampling frequency could be obtained by averaging the results of 14 to 20 independent visual acuity measurements with ETDRS charts. The subject of contrast sensitivity is covered in detail in Chapter 8.

Tests of Disability Glare

Visual acuity may be affected by glare when light is scattered by optical elements of the eye. Light scatter reduces the contrast of the retinal image, which in turn reduces the visual acuity. For a given glare situation, the reduction in visual acuity can serve as an indicator of the severity of light scatter. More subtle levels of light scatter are detected more easily if low-contrast charts are used, because low-contrast visual acuity is more substantially reduced by the additional reduction in contrast that results from the scatter.^{85,86} Disability glare is covered in detail in Chapter 8.

Measurement of Potential Acuity

With cataracts or other optical degradations of vision, it can be useful to know the visual capabilities of the retina, because this may influence decisions regarding surgical intervention. The effect of the optical opacities or irregularities must be eliminated or reduced to assess the retina's potential to achieve good visual acuity. The simplest and best-known procedure is the measurement of visual acuity with a pinhole aperture before the eye, as mentioned earlier in this chapter. Similar in principle was the PAM, which is an instrument that projects a Maxwellian view image of a visual acuity chart into the eye via a narrow beam that is of pinhole size as it enters the pupil.⁸⁷ After surgical treatment, the visual acuity should be at least as good as that obtained with the pinhole of the PAM.

When coherent light is used to form two-point images in the plane of the pupil, interference occurs, and a high-contrast grating pattern is formed on the retina.⁸⁸ The spatial frequency of the grating depends on the separation of the two spots, with wider separations giving higher spatial frequencies. The optical quality of the optics of the eye has relatively little effect on the quality (i.e., contrast) of the interference pattern on the

retina. A similar effect can be achieved by presenting grating patterns of variable frequency to the eye in a Maxwellian view.⁸⁹ Hence, the projection of coherent high-contrast gratings of different spatial frequencies onto the retina through distorted or clouded optics has been a method for the assessment of potential acuity. Elliott and colleagues⁹⁰ used tests of reading speed and Vianya-Estopa and colleagues⁹¹ used flicker fusion tests as alternative methods for the assessment of potential vision behind cataracts or other optical obstructions to vision. Yet another method for the evaluation of potential vision is by measurement of vernier acuity. Vernier acuity measures the accuracy with which the patient can judge whether targets are aligned, such as judging whether two spots are placed one underneath the other. The accuracy of alignment is relatively impervious to optical blur. Enoch and colleagues^{92,93} advocated using vernier acuity to evaluate the integrity of the retina behind dense cataracts as a means of estimating the visual improvement that might result from cataract extraction. Vernier acuity measurements necessarily require that patients make multiple settings, because it is the variance of alignment errors that is the measure of vernier acuity. The average alignment error is expected to be zero, unless there is an optical distortion of the retinal image or some disruption of retinal structure.

SUMMARY

Visual acuity remains the most widely used and most useful single clinical measurement for determining whether a significant abnormality or change is affecting the visual system. It is sensitive to refractive error and to many abnormalities that affect the optical media, the retina, the optic nerve, and the visual pathways. It is used routinely by eye-care practitioners during refractive procedures and during decision making when diagnosing or monitoring ocular disorders that affect vision. Letter charts are likely to remain the test of choice for the clinical measurement of visual acuity.

Since the 1970s, there has been slow progress toward standardizing many of the factors that can affect visual acuity measurements. The clinical research community has generally accepted and now almost exclusively uses charts that standardize the test task, and visual acuity scores are assigned in accordance with the principles proposed by Bailey and Lovie.¹⁰ Practitioners have been slower to change their methods. The chart design principles have not become widely used in clinical practice, except perhaps for low-vision care. Popular projector systems have restricted display areas that are not wide enough to allow five letters per row when the acuity rating is 20/63 or greater; these small display areas are more compatible with the restricted angular size of the viewing apertures of phoropters. These constraints make it unlikely that the clinical-practitioner community will

change to charts that have the same number of letters in each row across all size levels. Alternatively, logarithmic size progressions, standardized spacing ratios (at least between adjacent letters), and using letter sets of approximately equal legibility can readily be incorporated into projector chart formats. Pressures from recommendations published by authoritative bodies and from the practices of clinical researchers may influence the adoption of new chart designs for projector charts for clinicians.

Computer-driven flat-panel displays will gradually become the standard method for presenting visual acuity tests in clinical practice. Having the chart displays generated by computer allows for the randomization of letter sequences and controlled variation of optotype, contrast, luminance, and spacing. Similarly, for testing near vision, computer displays will allow for the easy variation of the test targets and display parameters, and it may become easy to monitor eye movement rather than listen to the patient reading aloud to assess reading efficiency. The use of computers will also enable better scoring of visual acuity, because it will be easy to record exactly which letters were read correctly. Appropriate algorithms can then be applied to generate more precise scores, to provide reliability information, and to enable the analysis of response speeds as a function of angular size.

The use of finer scaling to record acuity scores is even more important than the standardization of chart design. Among the community of clinical practitioners, there is not yet a broad appreciation of the extent to which clinical decision making can be enhanced by the use of finer scaling.^{32,71} Again, the methods of scoring letter by letter are widely used by researchers, and this might eventually influence general eye-care practitioners. One can expect resistance to adopting new units such as logMAR or the more user-friendly VAR. Without changing units, practitioners would gain better sensitivity for detecting changes in visual acuity if they make more frequent and more disciplined use of pluses and minuses to qualify visual acuity scores; alternatively, they could use approximate interpolations (e.g., 20/20⁺ might be called 20/19).

Letters should remain the target of choice for the clinical measurement of visual acuity for distance vision. Landolt rings, tumbling Es, numbers, and grating patterns will continue to be important, especially for population groups who do not use or who cannot read the English alphabet. The standard letters are likely to remain the five-by-five Sloan letters that follow the traditional framework used for earlier serifed letters and Landolt rings. The additional five-by-five letters and the redesigned variants of the Sloan letters introduced by the recent British Standard 4274-1⁸ are unlikely to challenge the broad acceptance of the Sloan letters. Although sans-serif fonts may be more easily dis-

played on pixilated screens, it seems probable that serifed fonts (Times Roman or Times New Roman) will become an official or de facto standard for reading acuity tests that are presented on printed charts or display screens.

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8

Contrast Sensitivity and Glare Testing

David B. Elliott

CONTRAST SENSITIVITY

Visual acuity (VA) and visual field (VF) assessments are traditionally specified in visual standards for driving, legal blindness, and a variety of occupations. However, it is now well established that these assessments do not provide all of the information necessary to indicate how well a person views the world. Numerous studies have shown that contrast sensitivity (CS) provides useful information about functional or real-world vision that is not provided by VA and/or VF,^{1–4} including the likelihood of falling,⁵ control of balance,^{6,7} driving,⁸ motor vehicle crash involvement,⁹ reading,¹⁰ activities of daily living¹¹ and perceived visual disability.^{12,13} It is clear that CS should be included with VA and VF in definitions of visual impairment and visual disability and for legal definitions of blindness.¹⁴ Thus, using CS in combination with VA (and VF, when necessary) gives the clinician a better idea of how well a patient actually functions visually. In addition, measuring CS is a relatively quick and simple procedure, and CS can provide more sensitive measurements of subtle vision loss than VA. There are many clinical situations in which CS can be reduced while VA remains at normal levels, including after refractive surgery,¹⁵ with minimal capsular opacification,¹⁶ with oxidative damage due to heavy smoking,¹⁷ in patients with multiple sclerosis,¹⁸ and in diabetics with little or no background retinopathy.^{19,20} For these reasons, CS measurements have become standard for most clinical trials of ophthalmic interventions, and they have been widely used in the assessment of refractive surgery,^{21,22} new intraocular implants,¹⁶ anticataract drug trials,²³ and potential treatments for age-related macular degeneration²⁴ and optic neuritis.^{25,26}

Theoretical Background

The following discussion briefly reviews the background literature pertinent to the design and measurement procedures of CS tests currently used in clinical eye care practice or in clinical trials.

Definitions

A *contrast threshold* is the smallest amount of contrast required to be able to see a target. CS is the reciprocal value of the contrast threshold. A patient who requires a lot of contrast to see a target has low CS, and vice versa. Before sine-wave gratings were used to measure CS, contrast was calculated in terms of Weber contrast. Weber contrast is defined as $(L_b - L_t)/L_b$, where L_b and L_t are the luminance of the background and the target, respectively. Presently, this measurement is generally used when calculating the contrast of letters or similar targets. For example, Snellen letters are of high contrast (generally over 90%), with black letters of low luminance against the much higher luminance of the white background.

The great impetus to present-day CS measurement came during the late 1950s and 1960s with the fundamental research work of Campbell, Robson, and Blakemore. These researchers began to evaluate CS using sine-wave gratings; these gratings had previously been used to characterize the optical performance of cameras and photographic film by Selwyn in 1948 and of televisions by Schade in 1956.²⁷ *Sine-wave gratings* are repetitive light- and dark-bar stimuli with luminance profiles that have the shape of the simple mathematical function sine (Figure 8-1). *Michelson contrast* is defined as $(L_{max} - L_{min})/(L_{max} + L_{min})$, and it is generally used when calculating contrast for gratings. L_{max} and L_{min} are the luminances of the lightest and darkest points of the grating, respectively. Michelson contrast is thus a unitless quantity, varying from 0 to 1 or 0% to 100%. One adjacent pair of light and dark bars makes up one cycle. This is also called the *spatial period* of the grating, and it is measured between successive troughs or peaks of the luminance profile (see Figure 8-1). The thickness of the gratings is described by their spatial frequency in cycles per degree (c/deg) of visual angle at the eye. When a large number of gratings can fit within a degree of visual angle, the grating has a high spatial frequency, and the gratings are fine. When the gratings are broader, fewer of them can fit within a degree of visual

angle, and the gratings are of lower spatial frequency (Figure 8-2). The grating's spatial phase defines its position in terms of the spatial period. For example, a change in spatial phase of 180 degrees indicates that the grating is displaced by half a cycle so that the light bars assume the previous position of the dark bars, and vice versa.

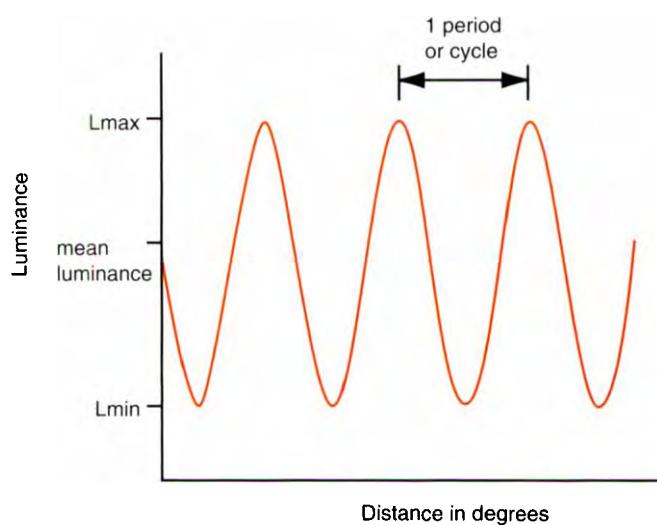


Figure 8-1

The luminance profile of a sine-wave grating.

A plot of CS over a range of spatial frequencies gives the contrast sensitivity function (CSF).^{*} A log scale of CS is used, because psychophysical measurements are logarithmic in nature (i.e., sensation $\propto \log$ contrast stimulus). A normal photopic CSF is shown in Figure 8-2. It shows a clear peak of about 2.3 log CS (CS of 200, 0.5% contrast threshold) at intermediate spatial frequencies, between about 2 and 6 c/deg. There is then a gradual fall-off in sensitivity at lower frequencies and a more rapid fall-off at higher spatial frequencies (this CSF shape is called *bandpass*). Anything in the area outside the curve is invisible to the human eye. The low-spatial-frequency decline is due to lateral inhibitory processes in the neural system. The neural and optical attenuation of high-spatial-frequency CS is about the same, and the optical quality of the eye limits resolution to about the same level as the foveal spacing of cones.²⁸ The point at which the CSF cuts the x-axis is called the *cutoff frequency* (see Figure 8-2). This represents the finest gratings (maximum spatial frequency) that can be seen at 100% contrast, and it therefore represents grating VA. The denominator of Snellen VA can be approximated from the cutoff frequency by dividing it into 600. For example, a cutoff frequency of 30 c/deg

*The imaging performance of lenses is generally assessed using the modulation transfer function. This compares object contrast levels to image contrast levels at a series of spatial frequencies. Changes in phase are measured in a phase transfer function, and the two together make the optical transfer function.²⁸

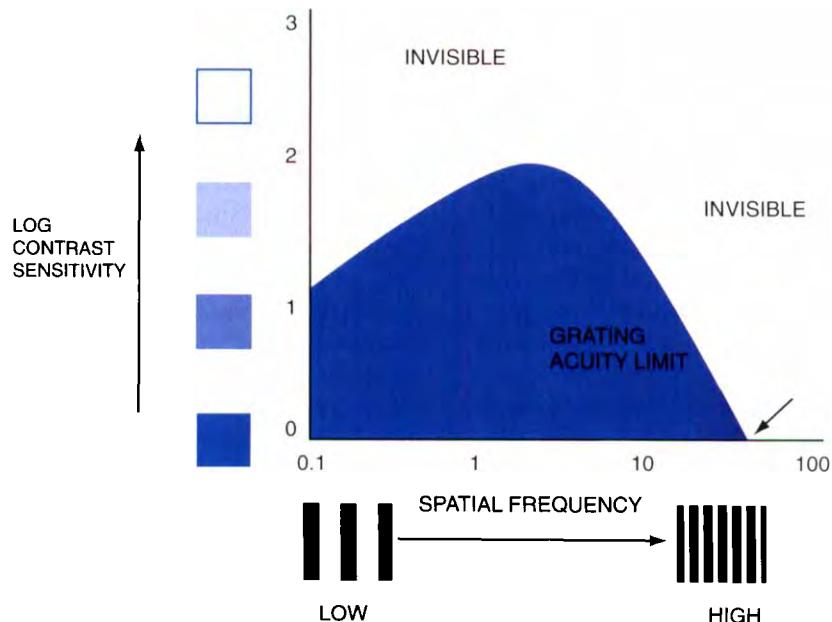


Figure 8-2

A typical photopic contrast sensitivity function. The grating acuity limit or cut-off frequency, shown by the arrow, is the highest spatial frequency grating that can be detected at maximum contrast.

gives a Snellen denominator of 20 and a Snellen VA of 20/20. Cutoff frequencies of 50 to 60 c/deg are often found in subjects with healthy eyes, unless high spatial frequencies are calibrated incorrectly.²⁹

Why Use Sine Waves?

Sinusoidal gratings were first used in the evaluation of optical systems because they are always imaged as sine waves of the same spatial frequency, even when they are degraded by defocus, aberration, diffraction, and light scatter. Only the contrast and the phase (spatial position) of the image are affected; the luminance profile remains sinusoidal. In addition, it is known from the work of Fourier (1768 to 1839) that sine wave gratings constitute the building blocks of complex periodic waveforms. The principle is used in music synthesizers, which can produce all sorts of sounds from sine-wave profiles. Similarly, various spatial light patterns can be broken down into a number of sine waves of certain contrast, phase, and orientation (Fourier analysis). They are therefore a "pure" stimulus that theoretically makes it easier to analyze any response to a sine-wave target (i.e., responses to more complex targets can be driven by responses to any number of components within the target). A Fourier analysis of a square-wave grating indicates the frequencies and amplitudes of the sine waves necessary to make up a square-wave grating. The fundamental sine wave provides the square wave with its essential phase, size, and contrast; what is lacking are the sharp edges between the gratings. These edges are produced by sine waves with higher frequencies and lower amplitudes, which are called the *harmonics*. Square-wave gratings include the odd harmonics of the fundamental. For example, if the fundamental frequency was f , the square wave grating would include harmonic frequencies of $3f$ ($\frac{1}{3}$ the amplitude of f), $5f$ ($\frac{1}{5}$ the amplitude of f), $7f$, and so on.

Channel Theory and Fourier Analysis

Psychophysical experiments by Campbell and Robson³⁰ first suggested that the CSF was an envelope of CS functions of several independent parallel detecting mechanisms. Each channel is highly sensitive to some particular spatial frequency band and virtually insensitive to all spatial frequencies differing by a factor of about two (Figure 8-3). The existence of channels was exciting from a clinical viewpoint, because it suggested the possibility of selective dysfunction in one or a small number of channels in various eye diseases. It was thought that different eye diseases could perhaps have their own pattern of CS loss—an individual "signature." Neurophysiological studies have also shown that, throughout the visual pathway, neurons are often selectively responsive to restricted bands of spatial frequency, temporal frequency, and orientation.^{31,32} The spatial CSF channels could be due to a series of ganglion cells that

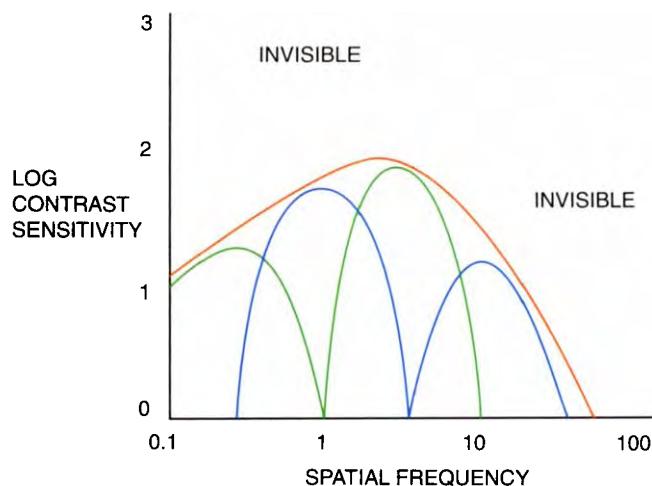
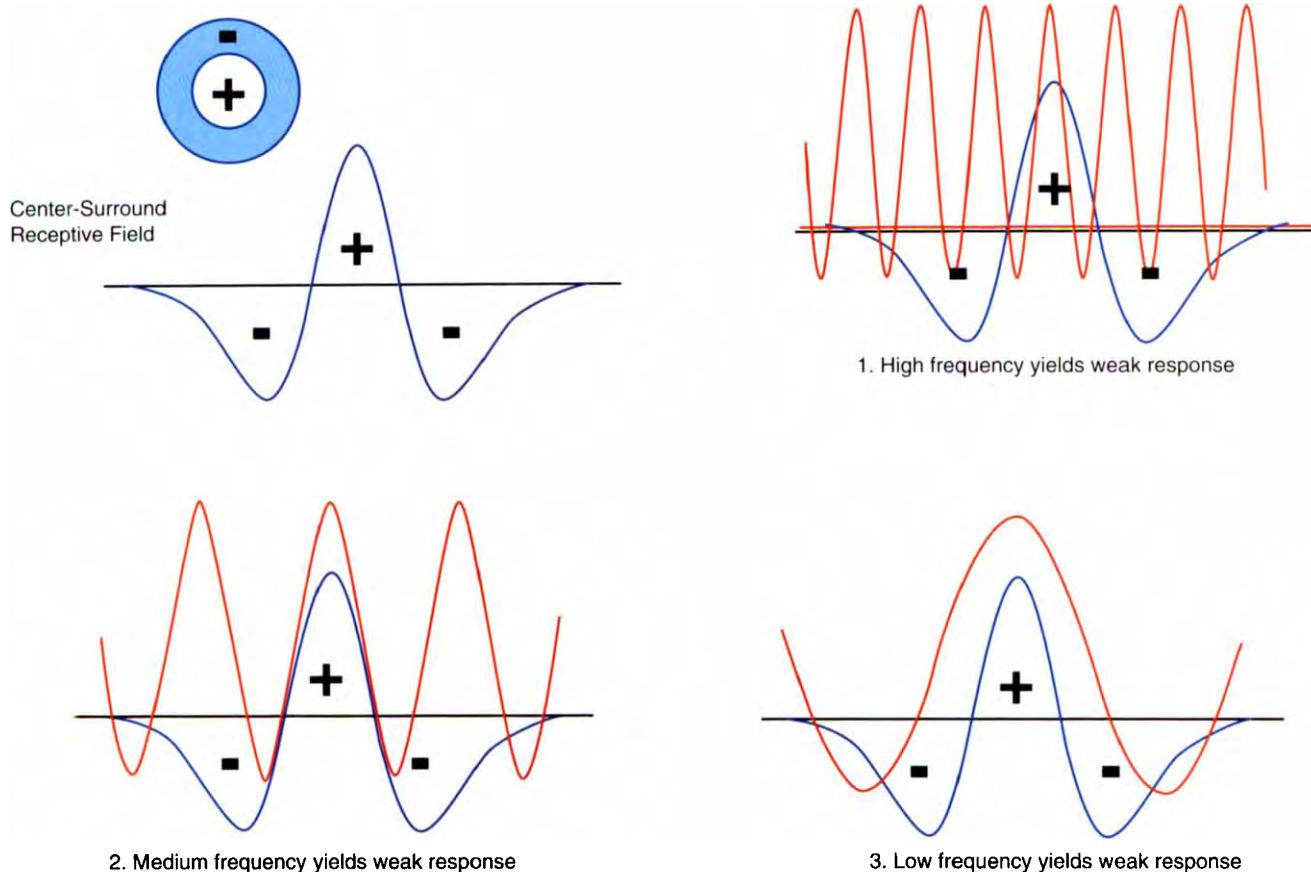


Figure 8-3

Four channels with their own contrast sensitivity functions are shown summed together to illustrate the channel theory of the contrast sensitivity function.

have receptive fields of different sizes so that they are maximally sensitive to different spatial frequencies. The spatial frequency "tuning" of each channel is thought to be caused by the characteristic center-surround organization of ganglion-cell receptive fields (Figure 8-4). A narrow, higher-peaked Gaussian distribution represents the center mechanism, and a broader, lower-peaked distribution represents the antagonistic surround (the "Mexican hat" profile). The difference of the two produce the response profile of the receptive field (difference of Gaussian [DOG]).³³ Stimulation of the receptors in the center of the field produces an increase in the cell's response, whereas stimulation of the surround causes a decrease. Stimuli smaller than the center receptive field (higher frequency) only produce a partial response from the ganglion cell. Stimuli larger than the center receptive field also stimulate the inhibitory surround area so that the overall response from the ganglion cell is progressively reduced.

Campbell and Robson³⁰ further suggested that the neurons in the visual cortex might process spatial frequencies instead of particular features of the visual world. Put simply, rather than piecing the visual world together like a puzzle, they suggested that the visual world is broken down into its separate spatial-frequency components by Fourier analysis, and this information is then passed in separate channels to the cortex, where it is reconstructed. However, the Fourier analysis model is simplistic, and it is incorrect to suggest that *any* spatial light distribution (e.g., a photograph of Babe Ruth) could be synthesized by adding together sine-wave gratings.²⁹ The simple channel theory assumes a linear system in that the sensitivity to complex periodic waveforms should be predictable by the sensitivity of the

**Figure 8-4**

The spatial frequency “tuning” of each channel of the contrast-sensitivity function is thought to be caused by the characteristic center-surround organization of ganglion cell receptive fields. Stimulation of the photoreceptors in the center causes an increase in the cell’s response, whereas stimulation of the surround causes a decrease. This renders particular receptive fields more responsive to some spatial frequencies than to others.

sinusoidal components. Although this has been shown for some complex waveforms,³⁰ it does not hold true for others. There are many examples of nonlinearity in the visual system,²⁹ and recent models tend to incorporate nonlinearities of spatial contrast detection.³⁴ Indeed, a linear contrast detection system is probably not desirable, because it can never be more than the sum of its parts, unlike a nonlinear cooperative system.²⁹ This does not question the use of spatial-frequency-specific sine-wave gratings as stimuli or the concept of parallel channels but rather the complete applicability of a Fourier analysis system. It should also be noted that, in addition to several channels relaying threshold information about sine waves, there are also channels relaying suprathreshold information, as well as channels for color, movement, depth, texture, and disparity.^{32,35}

How Many Channels Are There?

There seems to be reasonable consensus that the visual system consists of four to six spatial frequency channels modeling threshold contrast detection.³⁴ This has had

implications in the design of clinical CS charts. Ginsburg³⁶ designed the original Vistech charts to try to assess each of the contrast detection channels. The Vistech measures CS at five spatial frequencies. The selectivity of these channels is usually given in terms of their bandwidth, which is calculated as \log_2 of f_2/f_1 , where f_1 and f_2 are, respectively, the upper and lower spatial frequencies at which the sensitivity is half the maximum. For example, if a channel was maximally sensitive to a spatial frequency of 4 c/deg and sensitive by half this amount to spatial frequencies of 3 and 6 c/deg, its bandwidth would be $\log_2 (6/3) = 1.0$ octave. Similarly, a bandwidth of x octaves means that the upper spatial frequency is 2^x times the lower one. Blakemore and Campbell³⁷ first suggested a bandwidth of about 1.0 octave; again, this fundamental research has been used in the design of clinical CS tests. Most of the spatial frequencies used on the Vistech and Functional Acuity Contrast Test (FACT) charts increase in 1-octave steps, starting at 1.5 c/deg and including 3, 6, 12, and 18 (for all frequencies to differ by 1 octave, the last

spatial frequency should be 24 c/deg). There is now general agreement that bandwidths are in the 1.0 to 2.0 octave range, and orientation bandwidths are between ± 15 to ± 30 degrees.³⁴ Once again, this fundamental research information appears to have been used in clinical CS test design in that the orientations of the gratings of the Vistech and FACT charts are 90 ± 15 degrees.

What About Gabor Patches, Cauchy Functions, and DOGs?

A problem with traditional sine-wave grating targets is that they are nonlocalized. Peripheral areas of a large sine-wave stimulus fall on peripheral parts of the retina that have different analyzing characteristics than the central retina. For low-spatial-frequency targets, it may not be the central retina that is determining sensitivity, and attempts to model how the visual system is analyzing the stimulus become difficult. Given what is known of the changes in the receptive field size of ganglion cells, it is more likely that any vision channels (or filters) of spatial frequency are regionalized sets of localized channels.³⁴ Gabor patches, Cauchy functions, and DOG stimuli are all luminance profiles that enable the stimulus to be localized in space.²⁹ Gabor and DOG stimuli also resemble receptive field profiles of retinal or cortical cells, and it has been suggested that the visual system may analyze the retinal image using Gabor or DOG filters. DOGs were originally introduced as a quantitative description of the sensitivity profile of cat retinal ganglion cells (see Figure 8-4). Similarly, Gabor functions resemble experimentally obtained receptive field profiles of cortical cells.²⁹ A Fourier transform of the DOG luminance profile resembles the psychophysically determined CSF shape, which suggested to investigators that they were on the right track and that links between psychophysics and neurophysiology were being found.²⁹

Background for Clinical Contrast Sensitivity Measurements

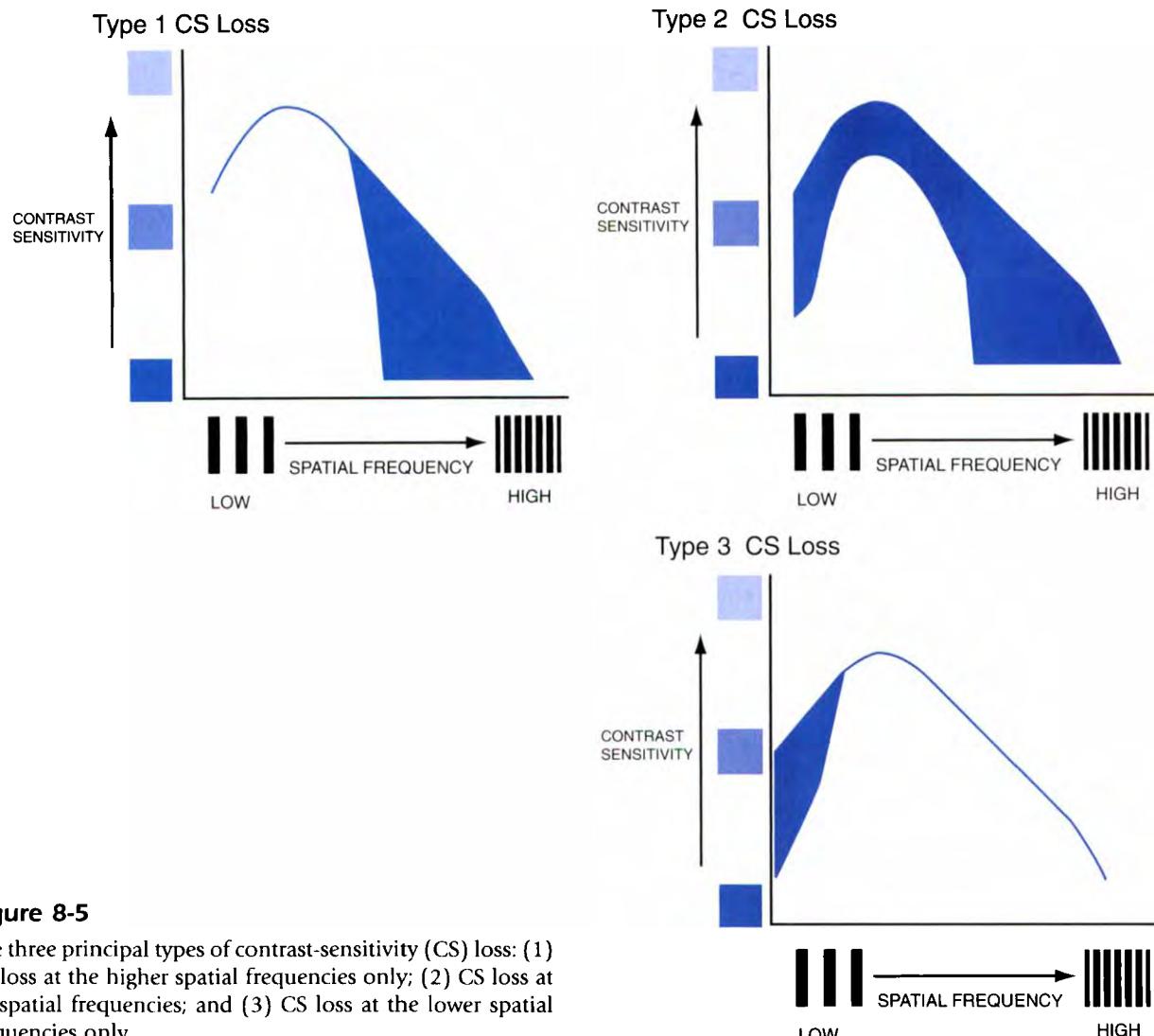
How Can Vision Be Poor if VA Is Normal?

A new clinical test must provide additional information to that already provided by the standard, or it must replace the standard. In the case of CS, the traditional test it must compete against is VA. The point at which the CSF cuts the x-axis indicates the finest pattern that is just detectable at maximum contrast, which corresponds to grating VA (see Figure 8-2); VA can therefore be predicted approximately from this point on the CSF. However, the reverse is not possible. As clinicians who are accustomed to always thinking of a patient's level of vision in terms of VA, it can be difficult to comprehend how vision can be poor if acuity is normal. However, just as the quality of sound is not determined by the highest-pitched note heard, so the quality of vision is

not determined solely by the smallest detail that can be resolved. The loss of low-frequency sound produces a "thin" sound, which has lost its "body" (this can be appreciated with the use of a hi-fi system with a graphic equalizer; it is similar to reducing the bass). The loss of low-frequency spatial frequencies similarly produces a thin, "washed out" picture of the world, yet acuity remains the same. The knowledge that resolution is an insufficient assessment of an optical system has been known for many years. For example, the quality of camera lens performance was once represented by its resolution limit; currently its modulation transfer function (MTF) is used. The MTF is essentially the CSF of an optical system: the ratio of image to object contrast through the system is measured at several spatial frequencies. Smith³⁸ found that, in two camera systems with the same resolution limit, the system with the better MTF produced the superior image.

Various Types of Contrast-Sensitivity Loss in Patients

Initially it was hoped that eye diseases would all have an individual signature CSF to aid diagnosis, but this has not materialized. Different diseases (and abnormalities) such as cataract, age-related maculopathy (ARM), uncorrected myopia, and corneal edema can all show a similar CS loss, and CS has little diagnostic value. Several authors have suggested that CS loss in patients with eye disease or abnormality can be classified into several different types.^{35,39} Three types are common to all classifications, and they are shown in Figure 8-5. Type 1 shows a high-spatial-frequency CS loss with normal CS at lower spatial frequencies. Type 2 shows a CS loss at all spatial frequencies. Often the early stages of eye diseases, such as cataract and age-related maculopathy, show a type 1 loss. Lower spatial frequencies become increasingly affected, and the CS loss can become a type 2 loss at a later stage.⁴⁰ However, the level of VA does not indicate which type of CS loss a patient has. A patient could have 20/30 (6/9) VA and type 2 CS loss or 20/60 (6/18) VA and a type 1 CS loss. In this example, the patient with the better VA may actually have worse vision than the other patient. Type 3 CS loss shows normal high-frequency CS (and likely normal VA) with reduced CS at lower spatial frequencies. Type 3 losses have been found in patients with optic neuritis, multiple sclerosis, primary open-angle glaucoma (POAG), papilledema, visual pathway lesions, diabetes, Parkinson's disease, and Alzheimer's disease^{35,41}; these are largely diseases that affect part or all of the visual pathway. Other CS losses have been described in the literature,^{35,39} but the only other CS loss type to be found in most classifications is the sharp "notch" loss. These losses seem to be relatively rare,³⁵ and they may be due to small amounts of spherical and/or astigmatic blur⁴² or perhaps to the unreliability of some clinical tests.⁴³

**Figure 8-5**

The three principal types of contrast-sensitivity (CS) loss: (1) CS loss at the higher spatial frequencies only; (2) CS loss at all spatial frequencies; and (3) CS loss at the lower spatial frequencies only.

Assessment of Real-World Vision

Until recently, relatively little attention has been paid to the use of clinical vision testing to predict real-world performance. VA testing, for example, has been employed to determine whether an individual is allowed to drive and to categorize a patient as legally blind, yet there is little or no evidence to indicate that a given level of VA is indicative of an individual's ability to drive or perform everyday visual tasks. VA is a poor predictor of driving ability,⁴⁴ and it is likely to be a poor predictor of many aspects of real-world vision, because the visual world is not composed purely of fine objects with sharp edges at high contrast; rather, it is made up of a variety of contrasts, with low contrasts being common.⁴⁵ The loss of CS is roughly equivalent to a loss of image contrast in the normal eye. For example, a twofold loss in CS is roughly equivalent to reducing image contrast in the normal eye by a factor of two,⁴⁶ and a CS deficit can predict a real-world contrast deficit. How much contrast loss is required before performance

is reduced depends on the visual task. Some tasks, such as optimal reading speed and mobility orientation in room illumination, are tolerant of large reductions in contrast⁴⁶⁻⁴⁸; these tasks would likely only be affected in patients with severe losses in CS. However, other tasks, such as reading speed of newspaper-size print and face recognition, are moderately affected by contrast reduction,⁴⁷ and mobility orientation under dim illumination has been shown to be seriously affected by reductions in contrast.⁴⁷ Under low-luminance conditions and when a patient is working near his or her acuity limit, tolerance to contrast loss is reduced.⁴⁶ This suggests that a patient's reported visual disability likely depends on the percentage of time spent functioning near his or her acuity limit and under low-contrast and low-luminance conditions, such as walking or reading small print in dim illumination, night driving, and walking or driving in fog or heavy rain. Although patients may not spend much time actually performing these tasks, these appear to be the very tasks that

many patients complain about. As indicated earlier, numerous studies have shown that CS provides useful additional information about functional or real-world vision that is not provided by VA. Using CS in combination with VA therefore gives the clinician a much better idea of how well a patient actually perceives the real world. CS has been shown to provide significant additional information beyond that provided by VA and even to correlate better than VA with various functional vision tasks, including the likelihood of falling, mobility orientation, balance control, driving, motor vehicle crash involvement, face perception, and reading performance, as well as activities of daily living and a patient's perceived visual disability.¹⁻¹³ CS should always be included with VA and VF in definitions of visual impairment and visual disability, and it will hopefully be included in future legal definitions of blindness and low vision.¹⁴

When assessing real-world vision using CS measurements, it is best to measure CS binocularly, because this is how most patients view the world. The relationship between monocular and binocular CS is not the same as that for VA. Binocular VA is generally about 10% better than monocular VA (about half a line on a logMAR chart) for a patient with two healthy eyes.⁴⁹ In a patient with differing VA in the two eyes, this binocular summation may disappear, and binocular VA tends to be highly correlated with the VA of the better eye.⁴⁹ Binocular CS is about 42% better ($\sqrt{2}$, or 0.15 log CS) than monocular CS in a patient with two healthy eyes.⁵⁰ With increasing differences between the CS of the two eyes, the binocular summation decreases. At a certain level, binocular inhibition—in which the binocular CS is *worse* than the monocular CS—can occur.^{49,51} It has been suggested that some of those patients who see better with one eye closed or whose "bad eye affects their good eye" have binocular inhibition.⁵² Given the small degree of binocular inhibition, current clinical CS tests have not been able to reliably detect it in patients.⁵³ Because of the influence of the difference in CS between the eyes on binocular summation, binocular CS correlates well with the monocular CS from both eyes.⁴⁹ It is therefore better to measure CS binocularly when assessing functional vision rather than to depend on the monocular CS of the better eye.

Measuring Contrast Sensitivity

Contrast Sensitivity Measurement Used in Research

Electronic systems are the most commonly used method in vision-research laboratories. The sine-wave gratings (or letters) are produced on a video monitor or oscilloscope screen, with the spatial frequency and contrast generally under computer control. This allows for great flexibility of testing parameters and procedures. The

computer can calculate and put into effect the required stimulus parameter levels; receive and interpret patient responses; convert numerical data into physical units; and store results in a convenient and easily accessible manner. Television monitors are cheaper than oscilloscope displays, but they lack the resolution and luminance range of the displays. Monitors need regular calibration of luminance (and thus contrast), because luminance does not vary linearly with applied voltage but rather is proportional to voltage raised to the power of gamma. The "gamma correction" of monitors, therefore, refers to their correct calibration. Traditionally, vertical sine-wave gratings are used, because CS is at maximum and approximately equal at 90- and 180-degree orientations, but it reduces to roughly equal minimums at 45 and 135 degrees.

Psychophysical Methods

Psychophysical methods are an important and occasionally undervalued aspect of measurement of any threshold, including CS. The psychophysical methods for CS address how to determine the contrast threshold. Most physiological mechanisms, including vision, do not give simple all-or-nothing responses. A subject's response to some stimulus invariably produces a sigmoid curve, as shown in Figure 8-6. At a very low contrast, the target is never seen, whereas, at very high contrast, it is always seen. Presentations at intermediate contrasts sometimes are seen; at other times, they are not. This type of curve is called a frequency-of-seeing curve or psychometric function. Because there is no sharp transition, a simple solitary value of threshold becomes difficult to define. With a psychometric function that ranges from 0% to 100% detection, the 50% detection level is generally chosen as an appropriate level to represent threshold.

According to signal detection theory, the sloped transition is caused by the presence of noise in the detection situation, and the subject's task is to distinguish signal plus noise from noise alone. Although noise can be external (e.g., slight changes in luminance, working distance, or accommodation), in a well-controlled experiment, the main source of noise is internal; it is caused by the variable activity level of the neural processes. The level of noise is assumed to be random. The introduction of a grating or letter increases the level of neural excitation by an amount that depends on the contrast level. When the patients' task is to indicate if and when they see a target, they set a criterion level at which they believe the target is visible. Remember that psychometric functions have no sharp transition and that the observer could choose anywhere on the function to be the threshold. Unfortunately, observers can use different criterion levels. An observer who is eager to demonstrate his or her ability with regard to the task would tend to give a positive response under conditions

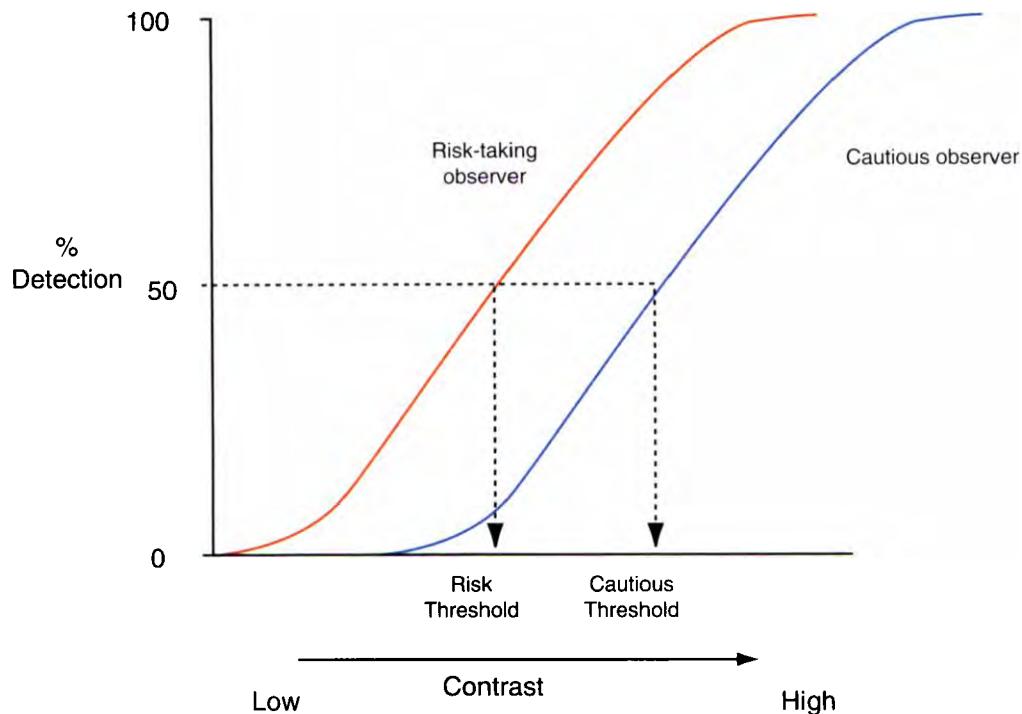


Figure 8-6

As the contrast of a target increases, a subject is more likely to see the target. There is no sharp division between visible and invisible, and the plot of detection versus contrast follows an S-shaped curve or psychometric function. The threshold value can be influenced by a subject's criterion of threshold. For example, cautious observers might wait until they are absolutely certain they can see the target and will have a higher contrast threshold than will observers who take more risks.

of uncertainty; a cautious or less confident observer would tend to only give a positive response if the target was clearly visible. This difference is demonstrated in Figure 8-6, which shows the psychometric functions of such observers. It could be that their actual contrast thresholds are identical, but they demonstrate different apparent thresholds, because one is a risk-taking observer and the other is a cautious one. Techniques that ask patients to indicate when they can just see the target or whether they can or cannot see it (yes/no procedures) are called *criterion-dependent methods*.

For clinical CS measurements, forced-choice techniques are used to try and eliminate criterion effects. The observer is told that the target always appears and is asked to state its position or some other characteristic: for example, "Is it on the right or left or top or bottom of the monitor screen?" or "What letter is it?" Alternatively, the grating can be presented during one or two time intervals in a temporal forced-choice technique, and the patient is asked whether the target was seen in the first presentation or the second. The patient is not allowed to respond "I cannot see anything," and, when they believe this to be the case, they must be asked to guess. In forced-choice procedures, risk-taking

observers may provide some incorrect responses regarding presentations that they believe they can see, and cautious observers are likely to correctly identify positions or letters that they state they cannot see. Forced-choice procedures are said to be criterion free.

Several psychophysical methods of contrast threshold determination are used in both research and clinical tests, and it is important to understand their differences. The method of adjustment involves the patient adjusting the contrast level themselves until he or she can just see it. The patient may be asked to increase the contrast from zero until it can just be seen. The practitioner then records the value, resets the contrast to zero, and then asks the patient to repeat the measurement. Several measurements are taken, and a mean is calculated. Often half of the measurements also involve decreasing the contrast from a high value until the patient perceives it to just disappear. Contrast thresholds measured from unseen to seen tend to be higher (more contrast, lower CS) than those measured from seen to unseen. The method of adjustment is quick and simple, but it is criterion dependent. Measurements that include a decreasing contrast level from a high value can suffer from the effects of adaptation (the cells

that respond to the grating tire and reduce their response). If the patient adapts to a high-contrast grating, the threshold measured is an adapted—and subsequently much higher—threshold.

The method of limits is similar, but the practitioner or a computer increases and decreases contrast. The contrast change is generally in discrete steps if under the control of the practitioner. When computers are used, continuous changes are used, and the technique is often called the *method of increasing contrast*. The speed of increase is obviously important, and a value of approximately 0.14% contrast/sec is often used.⁵⁴ Although this technique depends on the patient's response time (i.e., a motor factor as well as a sensory one) and is criterion dependent, it has been shown to be one of the most repeatable of the simple criterion-dependent methods.⁵⁴ *Bekesy tracking* is a modified automated method of adjustment. The contrast of the target is increased from zero until the patient can just detect it, and then the direction of the contrast change is reversed. The contrast decreases until the patient believes the target has disappeared, and then the contrast is increased, and so on. This is repeated for a predetermined number of reversals, and the threshold is determined from the mean of the reversal points. Although theoretically this appears to be a useful technique (it was proposed as the standard on the Nicolet CS-2000 system), its repeatability has been shown to be poorer than either the method of adjustment or the method of increasing contrast.^{54,55}

The method of constant stimuli presents a series of random contrast levels, and the response to each is recorded. Usually a set number (between about 10 and 20) of contrast levels are used, and the percentage of yes (or correct) responses at each level is calculated. The method of constant stimuli can be used in a yes/no (criterion-dependent) mode or in a forced-choice mode. With a yes/no procedure, the practitioner builds up a psychometric function like the one shown in Figure 8-6, and he or she can estimate threshold as the 50% point on the curve. In a two-alternative forced-choice (2AFC) procedure, the patient may be asked to indicate whether the grating was present on the top or the bottom of the screen; the computer then plots the percentage of correct responses at each contrast level to obtain a psychometric function. Because of the chance of guessing correctly, the lowest point on the psychometric function in a 2AFC procedure should be 50%. The threshold value is therefore generally taken as the 75% point on the curve (halfway between the upper and lower limits). Similarly, in any forced-choice procedure with N alternatives, the percentage lower limit on the curve is $1/N \times 100$, and the threshold is typically taken as halfway between that value and 100. *Weibull functions* are generally used to analyze psychometric functions in

the contrast domain; this method is generally regarded as the standard, but it requires many trials before it provides reliable data.⁵⁶ At least 100 trials are required when using a 2AFC procedure in this way.⁵⁷

In many cases, practitioners are not interested in the shape of the psychometric function but rather only the threshold, so the method of constant stimuli is inefficient, because much time is spent showing contrast levels well below or well above the threshold. Staircase methods are specifically designed to find the threshold value quickly. With a yes/no staircase procedure, contrast is reduced by a predetermined amount if the patient responds positively, and it is increased if the patient responds negatively. The contrast level continually hovers around threshold, and the measurement is ended after a predetermined number of trials or reversals. The threshold can be calculated as the final contrast level reached or the mean of the reversals. Staircase methods can also be used in forced-choice format in which the contrast is decreased after a correct response, and vice versa. A number of conditions need to be determined before staircase procedures can be used; these are the starting contrast level, the number of correct/incorrect responses needed before contrast is changed, the step size, when or whether to change the step size, when to stop, and how to calculate the threshold. The simplest 2AFC staircase procedure involves reducing contrast after two correct responses and increasing it after any incorrect response.⁵⁸ The rules used determine which point on the psychometric function is being estimated as threshold.⁵⁸ More complex staircase routines have been devised, such as parameter estimation by sequential testing (PEST), Best PEST, and quick estimate by sequential testing (QUEST).⁵⁶ These are called *adaptive staircases*, because the contrast level to be presented at any one trial is determined by the patient's responses to some or all of the preceding trials. Computer simulation runs of both Best PEST and QUEST suggest a greater accuracy and speed than the original PEST.⁵⁹ However, patients do not always perform as computer simulations would predict. In comparisons of simple staircases, PEST, and a maximum-likelihood technique on real subjects, researchers have found little difference in test-retest variability between the techniques.⁶⁰ They further suggested that inexperienced subjects (e.g., patients in a clinical situation) may have difficulty with Best PEST and QUEST; because they converge so quickly to near threshold, observers have little chance to familiarize themselves with the detection task. Subjects may also show a loss of motivation if contrast levels remain at or near threshold for too long.²⁹

Forced-Choice vs. Criterion-Dependent Tests

It would seem obvious from the previous discussion that forced-choice methods should be preferred over

criterion-dependent ones. This has been confirmed by several clinical studies of forced-choice versus criterion-dependent methods^{61,62} and is generally true, but a test is not automatically a good one just because it uses a forced-choice technique. For example, a 2AFC test must contain a large number of trials (certainly more than the number recommended for the test if used in yes/no mode),⁵⁶ otherwise its reliability will be poor. This is because, in 2AFC situations, there is a 50% chance of correctly identifying the grating position with your eyes closed. In addition, assumptions made about a yes/no staircase mode should be used with caution in 2AFC mode. Finally, in addition to whether a test uses a forced-choice or criterion-dependent psychophysical method, other factors of a test's design must be considered, such as its step size, number of trials, range of contrast levels, and time taken.

Ideal Contrast-Sensitivity Test Design Features

The American Academy of Ophthalmology⁶³ has suggested the following important test design principles for CS and glare tests. Clinical data have shown that tests that are consistent with these three principles of test design provide reliable data, unlike those that do not.⁶⁴

1. The test should use a forced-choice psychophysical method (see the caveats discussed earlier). Letter targets are particularly useful in this regard, because they provide a choice of 26 (if the patient is unaware that only a subset of the alphabet is generally used) or 10 patient responses. Many clinical charts just use the 10 Sloan letters of D, H, N, V, R, Z, S, K, O, and C or the 10 British Standard letters of D, H, N, V, R, Z, F, P, E, and U, because they have been shown to have similar legibility (at high contrast). The more choices available, the more reliable the responses will be. For example, in a 2AFC situation, the patient has a 50% chance of guessing correctly below threshold. In a 10AFC situation, the patient only has a 10% chance of guessing correctly, and, in a 26AFC situation, the patient has about a 4% guessing rate. It is perhaps useful at this point to think back to the problem of the risk-taking and cautious observers discussed earlier (see Figure 8-6). With a yes/no task, their thresholds can differ substantially because of differences in the patient's criterion of threshold. What happens with the forced-choice letter task (e.g., Snellen acuity)? The risk takers would likely guess at a line of Snellen VA letters and get them all wrong, and a true acuity threshold would be obtained. The cautious observers would probably state that they could not see any more on the chart, but, when pushed to try to read the next line down and guess, they would read it all correctly and perhaps even read some of the letters on the line below that! The risk takers are identified and a true

threshold obtained with the use of the 26AFC psychophysical method. The cautious observers can only be identified by a good measurement method in that all patients must be pushed to obtain a true threshold measurement. Differences in measurements of VA and CS can be obtained using the same chart by different practitioners, and this is probably the result of differences in measurement technique and, in particular, how much patients are pushed to guess.^{65,66}

2. Test targets should follow a uniform logarithmic progression. This scale of progression provides equal perceptual steps. Consider the use of a linear contrast scale on which the difference between 2% contrast ($1.70 \log CS$) and 4% contrast ($1.40 \log CS$) is huge perceptually, because the brain perceives contrast on a logarithmic scale. Alternatively, the perceptual difference between 30% contrast ($0.52 \log CS$) and 32% contrast ($0.50 \log CS$) is minimal and may not even be seen. Using a linear contrast scale would provide a test that was insensitive to CS changes at low contrast levels (around 0% to 5%), and it would contain unnecessary steps at high contrast levels.
3. Several trials should be used at each level, and step sizes should be smaller than the variability that is inherent in patients with normal vision. The number of decisions at each contrast level should take into consideration the number of alternatives available for each decision. For example, a 2AFC test should contain many decisions at each contrast level, because the probability of an incorrect response due to chance would otherwise be too high. One or two decisions per level would give 50% or 25% guessing rates, respectively. The 20/200 (6/60) letter on a Snellen chart is often criticized for its unreliability, because decisions are based on one letter. At least it is a 26AFC task; imagine how unreliable it would be if it was a 2AFC task. The CSV-1000E is a 2AFC test with one decision per level, and the Vistech and FACT tests are 3AFC tests with only one decision per level. The best combination is a multichoice task (e.g., a letter) with several trials (or letters) at each contrast level.

A practical limitation of clinical tests, including CS tests, is size. A vision test chart needs to fit easily into an examination room. This, therefore, tends to limit the number of step sizes, particularly if a test attempts to measure CS at several spatial frequencies. CS tests could provide either a screening service, which would just provide contrast levels around the boundary of normal and abnormal scores (the test would then just determine whether the patient's CS was normal or not), or they could provide a quantification of CS and allow for the monitoring of patients with reduced CS. This would

require contrast levels over the complete range of contrasts from near zero to near 100%. Most CS charts attempt to provide a test that can be used for both screening and monitoring patients, and step sizes can be relatively large. If the step sizes are too large, the test provides poor sensitivity.⁶⁷ For example, imagine a Snellen test with lines of only 20/15 (6/4.5), 20/30 (6/9), and 20/120 (6/36). It would be insensitive to subtle refractive error or disease. This would also be true of any CS test with large step sizes.

How Can the Quality of a Contrast-Sensitivity Test Be Determined?

Several aspects of a test's usefulness are generally considered before it becomes commercially available. These include the test being perceived as a good value for the information provided, its ability to provide relatively quick and simple data collection (for both patient and clinician), and its safety for patients. Important aspects of a test's performance are its validity, its discriminative ability, and its repeatability. These qualities are often described in the research literature, and they are used by manufacturers to advertise a test's usefulness, so they need to be understood.

A test is valid if it measures what it purports to measure. This is often indicated by how closely the results match those from a "gold-standard" measurement. For example, the validity of new tonometers is often determined by how close the results are to the gold-standard Goldmann tonometer, and the validity of autorefractors is determined by how close results are to subjective refraction. The gold standard used depends on what aspect of the test is being assessed. For example, when testing whether CS measures real-world vision, a gold-standard measure of real-world vision would be used, such as a visual disability questionnaire.⁶⁸ The relationship between the test and the gold standard is usually described by the correlation coefficient between the two tests.

Discriminative ability indicates how well a test discriminates between normal and abnormal eyes. In published results of clinical trials, it is often indicated that a highly significant difference was found between a group of patients with ocular abnormality and a control group. It should be noted that this only indicates that, *on average*, there is a difference between the groups; it does not indicate how well the test predicts whether an *individual patient* has the abnormality or not. If a test is used to discriminate between normal and abnormal, there are four possible outcomes. The test could indicate that an eye is abnormal and be correct (true positive or hit) or incorrect (false positive or false alarm); a false alarm results in a patient being treated or referred for further testing for no reason. The test could also indicate that an eye is normal and be correct (true negative or correct reject) or incorrect (false negative or

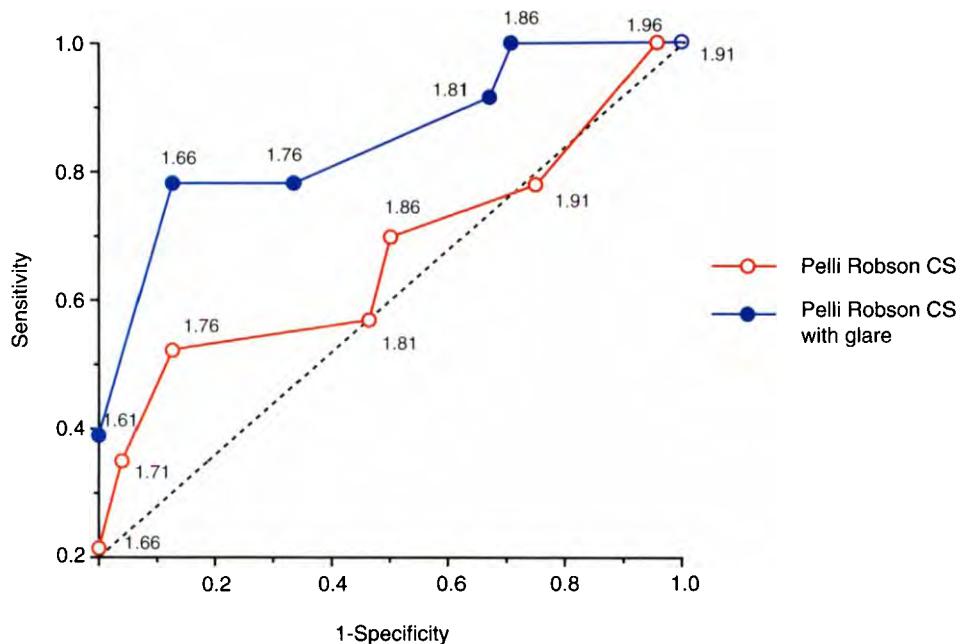
miss); a miss results in a patient who should have been treated or referred not being properly addressed. These results are usually described in terms of a test's sensitivity and specificity. *Sensitivity* (hit rate) is the proportion of abnormal eyes correctly identified, and *specificity* (correct reject rate) is the proportion of normal eyes correctly identified. In mathematical terms, it could be described as follows:

$$\text{Sensitivity} = \frac{\text{true positives or hits}}{\text{number of actual abnormal eyes}}$$

$$\text{Specificity} = \frac{\text{true negatives or correct rejects}}{\text{number of actual normal eyes}}$$

A test's sensitivity and specificity depend on the cutoff score chosen to differentiate normal and abnormal. For example, the sensitivity and specificity of VA would change depending on whether the chosen cutoff point is 20/15 (6/4.5) or 20/25 (6/7.5). Using 20/15 (6/4.5) would give the best sensitivity, because those abnormalities with 20/20 (6/6) or 20/25 (6/7.5) would be correctly identified as abnormal. However, specificity would be poor, because individuals with normal vision with acuity of 20/20 (6/6) or 20/25 (6/7.5) would be classified as abnormal. Alternatively, sensitivity would be poorer and specificity better using 20/25 (6/7.5) as the cutoff point. Plotting a graph of sensitivity (which corresponds to the hit rate) against 1-specificity (false-alarm rate) helps to visualize the tradeoff between specificity and sensitivity for different cutoff points and to determine where the optimal cutoff point lies; this plot is called the *receiver-operating characteristic (ROC) curve* (Figure 8-7). Although the ROC curve can indicate the most efficient cutoff point, this is not always the most appropriate. For example, if a test was being heavily relied on to determine whether a patient had a sight- or life-threatening disease that could be successfully treated, it would be desired that the test provide its best sensitivity. Although this would mean an increase in the number of false alarms, at least as few patients as possible with the disease would be missed. If early detection of the abnormality under consideration has little effect on the prognosis, a cutoff point providing relatively poor sensitivity may be chosen, because a relatively high miss rate would not be cause for alarm. This would then improve specificity, and relatively few individuals with normal vision would be wrongly treated or referred.

The end result of how well a test discriminates between normal vision and a particular disease also depends on the prevalence of the disease. It is unfortunate for clinical tests that ocular diseases are rare.⁶⁹ To improve the situation, tests are particularly used when there are other suspicious symptoms or signs of a particular disease, because the prevalence of the disease in this subpopulation is much greater than in the population as

**Figure 8-7**

Receiver-operating characteristic (ROC) curves indicating the ability of the Pelli-Robson chart with and without the Brightness Acuity Tester to discriminate between a group of 24 young subjects with normal vision and 23 older subjects with normal vision. The dashed diagonal line indicates the zero discrimination level. The closer the ROC curve is to the top left corner, the better the discrimination. CS, Contrast sensitivity.

a whole. For example, possible glaucomatous fields would not be looked for in a 20-year-old patient, but they would certainly be an issue to address if the patient were 75 years old with a family history of the disease. In addition, treatment or referral is not based on the findings of one test but rather on a battery of tests.

Studies tend to refer to reliability and repeatability as the same thing: the variability of results from test to retest. However, correlation coefficients, which are traditionally used to calculate reliability, provide information about between-subject variability as well as between-test variability (repeatability). When a test with the same repeatability is used, correlation coefficients can be low if subjects all have normal vision. The coefficients are higher with a larger spread of results when vision is subnormal, and it is then difficult to discriminate between subjects.⁷⁰ A correlation coefficient of 0.90 or higher has been suggested as a minimum required reliability for a clinical test.⁵⁵

Other more recent assessments that only give information about repeatability include the coefficient of repeatability (repeatability value) and concordance values. The coefficient of repeatability is calculated as 1.96 times the standard deviation of the differences between the test and retest scores,⁷⁰ and it provides the 95% confidence limits for a change in score. The mean of retest minus test scores should be close to zero if there is no significant training or fatigue effect between test

and retest. Because this value does not range between zero and one, this score should not really be called a coefficient, and it is probably better called the *repeatability value*. For measures that use a continuous score, the repeatability value provides a criterion for statistically significant change; any change in score above that figure is significant and sets specificity at 5%. For tests that do not measure on a continuous scale, the criterion for change falls at the next contrast level above the repeatability value (i.e., if the repeatability value was ± 0.23 log CS but the chart used step sizes of 0.10 log CS, the criterion for change would be ± 0.30 log CS, or ± 3 steps). In such cases, the change criteria are often best obtained by direct viewing of the data.⁷¹ Repeatability values cannot be used to compare the repeatability of tests that measure in different units, such as CS and VA.

Concordance values (the percentage of patients getting exactly the same score on test and retest) have also been used to indicate that a test is reliable. However, the fact that patients often obtain exactly the same score during follow-up visits indicates that the step sizes on the test are too big rather than that the test is somehow discriminative or reliable.⁶⁷ For example, a VA chart containing only a 20/20 (6/6) and a 20/120 (6/36) line would provide very high concordance, but it would be of little value.

Reliability appears to be the most important quality of a test, because it influences the others.⁶⁴ For example, if a

test has poor reliability and test results correlate poorly with retest results, it is unlikely that results from the test will correlate highly with a gold-standard measure. Therefore, its validity is poor. If a test cannot predict itself very well, how can it predict something else? Similarly, an unreliable test is likely to discriminate poorly. Because of the reliance of the correlation coefficient on the range of scores used, repeatability values should always be given with correlation coefficient results during test-retest studies.^{70,72} Repeatability values also indicate the amount of clinically significant change required when monitoring a patient's CS. Companies should be encouraged to provide reliability and repeatability data with any newly available test (preferably from independent researchers) so that it can be compared with current tests.

Gratings or Letters?

The arguments for gratings and against letter targets were propounded in a letter by Leguire⁷³ and responded to by Regan⁷³ and Pelli and Robson.⁷⁴ These are well written and thought-provoking letters, and they make for excellent reading. Several minor points that Leguire raises against letter charts, such as changes in mean luminance over the charts and increased variability due to differences in letter legibility, are red herrings. Leguire is correct in stating that letter tests measure an identification threshold, whereas traditional CS with gratings measures a detection threshold; it is unlikely that this is of any advantage to gratings.⁷³ He is also correct in stating that letters do not measure the traditional CSF (i.e., the CSF for a static sine-wave grating). The fundamental research of Campbell and colleagues and subsequent clinical research using gratings suggested to Leguire that CS measurements using sine-wave grating targets at several spatial frequencies should be used to assess the various visual channels; this view is also held by Ginsburg,⁷⁵ among others. Letters have a broad spatial frequency spectrum with spatial frequency information of different orientations, and they could be said to use a shotgun approach.⁷³ The most important frequency of a letter is approximately 1.5 to 2 cycles per letter width.^{76,77} For the Pelli-Robson chart, when measured at the standard 1 meter, this is about 0.50 to 1.0 c/deg; for Rabin's 20/50 (6/15) letter CS chart, it is about 7 to 10 c/deg. Despite all the theoretical arguments of letters versus gratings, in the end, it is a matter of which types of chart "work": which charts best discriminate normal from diseased eyes and which best predict functional vision loss. At present, letter charts appear to have an edge, because they use very good psychophysical test design features (i.e., smaller step sizes, a larger number of decisions at each level, and a large number of forced-choice alternatives) and provide reliable results, unlike the grating charts (see later sections). As stated earlier, the reliability of a test to a reasonable degree determines other properties of that test, such

as its validity and discriminative ability. Letter CS tests have also been shown to discriminate better than grating tests between different intraocular lens implants⁷⁸ and to best document improvement in CS due to Nd:YAG capsulotomy.⁷⁹

What About Mesopic CS?

The number of patients with symptoms of vision problems at night—and particularly with difficulty driving at night—has increased since the introduction of refractive surgery.⁸⁰ Photorefractive keratectomy can induce significant reductions in CS under mesopic conditions even though photopic CS is normal⁸¹; this was attributed to increased postsurgical optical aberrations for the larger pupil sizes found under mesopic conditions. Mesopic CS may therefore be a useful assessment of vision after refractive surgery. Mesopic CS measurements may also be useful in pre- and postoperative cataract patients who complain of poor vision in dim illumination. The backscattered light from cataracts (i.e., the light scatter seen when using the slit-lamp) does not reach the retina, and, in combination with increases in light absorption in patients with nuclear cataract, it can cause significant problems for cataract patients in dim illumination.⁴⁷ It is also likely that increases in pupil diameter at night and in dim illumination would affect the quality of vision achieved by patients with multifocal intraocular lenses.

Clinical Contrast Sensitivity Tests

The Earliest Clinical Contrast-Sensitivity Tests

An extensive and fascinating review of the history of CS testing is given by Robson.²⁷ CS measurements were first made as far back as 1845 by the physicist Masson using a rotating disk.²⁷ Even low-contrast letter charts are antique. Bjerrum made low-contrast letter charts of 9%, 20%, 30%, and 40% contrast in Copenhagen in 1884. The British ophthalmologist George Berry published papers about such measurements in patients with tobacco amblyopia and retrobulbar neuritis in the 1880s. The first commercially available test seems to have been George Young's CS test apparatus, which was available from J. Weiss of London in the 1920s. However, none of these tests found any widespread use. A piece of work not included in Robson's review but that is deserving of mention is that of Fortuin.⁸² His measurement of "visual power" included VA and letter CS at various illumination levels. Given the level of understanding of the visual system at that time, these tests must have been developed on the basis of a clinical intuition that Snellen VA was not providing adequate information for patients with some eye diseases. The reemergence of clinical CS measurements came after the studies of Campbell and colleagues that were mentioned above. Their work implied that, theoretically,

some patients with eye disease could have losses in CS with normal VA. Although this was first shown by Berry in the 1880s, these results were not widely known. Based on the understanding of the CSF provided by the Cambridge group, the clinical utility of CS was first reported using grating CS by Bodis-Wollner in 1972.⁴¹

The Arden gratings were first produced in 1978.⁸³ They consisted of six testing plates and a trial plate, each of which contained a printed sine-wave grating at a particular spatial frequency (0.2, 0.4, 0.8, 1.6, 3.2, and 6.4 c/deg). The contrast of the gratings varied from zero at the top of the plate to maximum contrast at the bottom, changing gradually on a logarithmic scale. A linear scale of 1 to 20 units was used to represent the contrast levels. The plates were stored in a folder and, during measurement, were slowly withdrawn by the practitioner. The patient was asked to scan the edge of the folder and indicate when he or she could first just see the grating. The unit on the scale at which the patient indicated that he or she could see the grating was recorded. The method used was criterion dependent, and the test suffered from differences in the speed of removal and interpretation of the testing method by different practitioners,⁸⁴ so test-retest repeatability was poor.⁸⁴ The Cambridge low-contrast gratings were developed in the 1980s, and they were presented in a 28 × 22-cm booklet that was spiral bound along the shorter edge.⁸⁵ The pages were presented in pairs, one above the other or side by side, at a viewing distance of 6 m. One page in each pair was a uniform gray; the other contained a square-wave grating of the same mean luminance. The grating on the first page was of very high contrast and easy to see, but the contrast of the 10 subsequent pairs became progressively lower. The patient was asked to state the position of the grating (e.g., "upper" or "lower"), and the practitioner noted the pair number on which an error first appeared. The CS level was calculated from a total of four runs using the score conversion system provided. The test therefore used a 2AFC method with essentially four decisions at each contrast level. The chart was designed to measure CS at 4 c/deg only. The system was externally illuminated, and, although no light meter was provided with the system, a technique of light-level measurement using a single-lens-reflex camera was suggested and explained. Repeatability values have been given for the Cambridge gratings, but these were in CS rather than log CS units, and so they could not be easily compared. However, Jones and colleagues⁸⁶ found a repeatability value of ±10.4 test units, which spans one third of the subject's performance range, and they suggested caution if one is using these charts to monitor CS.

The Pelli-Robson Letter CS Chart

The Pelli-Robson CS chart (Figure 8-8) is an 86 × 63-cm chart that is hung on the wall at 1 m from the

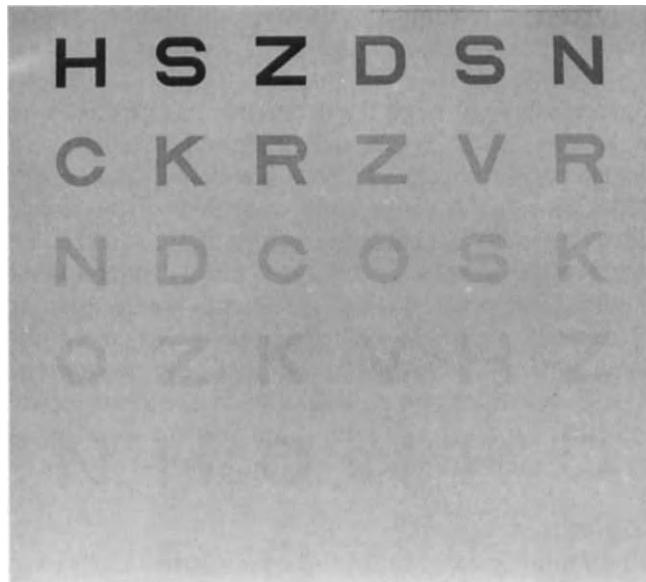


Figure 8-8

The Pelli-Robson letter contrast sensitivity chart.
(Courtesy of Dr. Denis Pelli.)

patient's eye. The chart consists of 16 triplets of 4.9 cm (2.8 degrees at 1 m) letters, and it assesses CS around 0.50 to 1 c/deg. Within each triplet, the letters have the same contrast, and the contrast in each successive triplet decreases by a factor of 0.15 log units. The test therefore uses a 26AFC (or 10AFC if the patient is aware that a selection of only 10 letters are used) method with three decisions at each contrast level. The measurement procedure is explained later. The system is externally illuminated, and scores change little over a wide range of luminance levels.⁷⁴ Measurements are quick, simple, and easy for the patient to understand. Reliability of the Pelli-Robson CS chart is good, and it is much better than that of the grating CS tests currently available (mean $r = 0.84$)^{43,64,87} (mean repeatability value = ± 0.29 log CS).^{53,64,87} Discriminative ability and validity is similarly good.^{1,9,12,64,68} The chart only measures CS at low spatial frequencies when measured at 1 m so that it is insensitive to high spatial frequency CS loss⁸⁸, and it is designed to complement traditional high contrast VA measurements. It is ideal when determining functional vision loss in patients with low vision¹⁴ and moderate and dense cataract^{9,89,90}; when screening for low spatial frequency loss in, for example, patients with optic neuritis, multiple sclerosis,¹⁸ or visual pathway lesions; and when examining diabetics with little or no background retinopathy,^{19,20} patients with Parkinson's disease, and patients with Alzheimer's disease. One disadvantage is that a variable endpoint can be gained depending on how long the patient is left to stare at the letters near threshold. The Pelli-Robson chart and other "large-letter" CS tests can be used with longer working dis-

tances so that higher spatial frequencies are assessed. For example, when used at 3 m, the chart measures CS at about 1.5 to 2 c/deg. The Pelli-Robson chart may be best used at 3 m when measuring CS in patients with cataracts, because it is insensitive to early cataract when used at 1 m.⁹¹ The charts are available from any Haag-Streit authorized dealer (e.g., Clement-Clarke, Lombart). VectorVision also produces a chart similar to the Pelli-Robson (CSV-1000LV), with a letter size of 20/630 at 1 m. However, the test has only eight contrast levels and step sizes of 0.15 and 0.30 log units as compared with the Pelli-Robson's 16 levels and constant 0.15 log unit steps. The CSV-1000LV has the advantage of being internally illuminated with a self-calibrating system, and it is available from www.vectorvision.com. Similar tests are also available on computer-based systems that use flat-panel liquid crystal displays at www.thomson-software-solutions.com.

Small-Letter CS

Rabin⁹² showed that the steepness of the CSF near the spatial frequency cutoff provided a relatively larger blur-induced loss of CS as compared with VA. Rabin compared the effect of optical defocus and luminance effects on VA and a small-letter CS test (assessing high spatial frequency CS), and he found that both affected the CS test much more than VA (approximately a 16-fold reduction in small-letter CS as compared with a three-fold reduction in VA). After correction for the greater variability in CS measurement, the test still provided a more sensitive (1.75-fold) index of optical defocus and decreasing luminance than VA. Recent studies have also shown that the small-letter CS test is more sensitive than VA to several clinical conditions, such as early cataract and contact lens edema.^{88,93} It should be ideal when attempting to measure subtle losses of vision that may be resistant to high-contrast VA, such as after refractive surgery^{94,95} and in patients with very early cataract, such as those participating in anticataract drug trials.⁸⁸ CS of very small letters, such as 20/30, correlates very highly with VA,⁸⁸ and the ideal size for a small-letter test may be about 20/50.⁹⁴ Letters that are 20/50 measure CS over a range of spatial frequencies of around 7 to 10 c/degree. The 20/50 letter charts use good test design features (Table 8-1), and repeatability has been shown to be good (repeatability value ± 0.20 log CS),⁹⁴ and the charts are available from www.precision-vision.com.

Low-Contrast VA

Letter CS charts like the Pelli-Robson measure the contrast threshold of letters of a fixed size. Low-contrast VA charts like the Regan and Bailey-Lovie charts, however, measure the smallest letter that can be resolved at a fixed contrast. Low-contrast acuity charts do not measure CS (Figure 8-9). It is difficult to state which spatial frequencies the low-contrast letter charts are measuring,

because this depends on the VA threshold. If only the large letters at the top of the chart can be seen, the score gives an indication of CS at intermediate spatial frequencies. If a patient can see the small letters at the bottom of the chart, the score gives an indication of higher spatial frequencies. Low-contrast VA scores are believed to indicate the slope of the high-frequency end of the CSF. It has been suggested that they can be used to indicate the CSF when used in combination with a low-frequency or peak CS measure (e.g., the Pelli-Robson or Cambridge charts) and a high-contrast VA measurement.⁹⁶ The lower the contrast of the acuity charts, the more sensitive they become to subtle vision loss. For example, for detecting subtle vision losses in aviators or subtle changes after refractive surgery, it has been suggested that the 11% or 4% Regan charts be used.⁹⁷ For greater losses in vision (e.g., cataract), these very-low-contrast charts cannot be seen by some patients, and a higher-contrast chart is necessary.

Bailey-Lovie VA charts (Figure 8-10) include a 10% contrast chart (this is Michelson contrast and corresponds with 18% in Weber contrast) in addition to the traditional high-contrast VA chart. These charts have several advantages over the traditional Snellen, including the same number of letters (five) on every line and a logarithmic progression in size from one line to the next (this provides equal perceptual steps). The test, therefore, uses a 26AFC method with five decisions at each acuity level, and a by-letter scoring system can be used. Low-contrast VA charts typically provide very reliable results as compared with grating CS tests (mean $r = 0.92$; mean repeatability value = ± 0.13 logMAR VA).^{18,64,87} The Bailey-Lovie charts are commercially available from MultiMedia at the University of California, Berkeley. In addition, "Mr. Happy tests" of CS are also available for testing infants and preschool children⁹⁶ and developmentally delayed or multihandicapped patients.⁹⁸ Regan VA charts are also available in a range of contrast levels (Weber contrast of 96%, 50%, 25%, 11%, and 4%). The Regan charts are similar in design to the Bailey-Lovie chart, but they differ slightly in font configuration and number of letters, and they produce slightly better VA thresholds than the Bailey-Lovie or Early Treatment of Diabetic Retinopathy Study (ETDRS) acuity charts.⁹⁹ The Holliday Contrast Acuity Test uses a flip-chart format for near testing with letters at 100%, 50%, 25%, 12.5% and 6.25% contrast (www.stereo-optical.com). Low-contrast acuity charts are also available from VectorVision in several ETDRS formats (similar to the Bailey-Lovie design; see Chapter 7) and with several contrast levels (from 6% to 95%), with three letters per row and a range from 20/100 to 20/16 acuity. All VectorVision tests have the advantage of being internally illuminated with a self-calibrating system, and they are available from www.vectorvision.com. A large array of low-contrast

TABLE 8-1 A Summary of the Target Stimuli, Range of Values, Step Sizes, Psychophysical Methods of Measurement of Contrast Sensitivity (CS), and Low-Contrast Visual-Acuity Tests

Test	Target	Range/Steps	Psychophysics
Contrast Sensitivity Tests			
Cambridge	Square-wave gratings	~1.50 log CS range	2 AFC
Gratings	Variable contrast	~0.17 log steps	4 decisions per level
CSV-1000E	Sine-wave gratings	~1.38 log CS range	Criterion-dependent/2 AFC
	Variable contrast	~0.16 log steps	1 decision per level
FACT	Sine-wave gratings	~1.20 log CS range	3 AFC
	Variable contrast	0.15 log steps	1 decision per level
Melbourne edge test	Edges	~2.30 log CS range	4 AFC
Miller-Nadler	1.7° Landolt rings	0.20 and 0.10 log steps	1 decision per level
	Variable contrast	1.5 log CS range 5% and 2.5% contrast steps	4 AFC 1 decision per level
Pelli-Robson	2.8° letters	2.25 log CS range	10 or 26 AFC
	Variable contrast	0.15 log steps	3 decisions per level
Vistech	Sine-wave gratings	~1.80 log CS range	Criterion-dependent/3 AFC
	Variable contrast	~0.25 log steps	1 decision per level
Low-Contrast Visual-Acuity Charts			
Bailey-Lovie	18% contrast letters	~1.30 logMAR range	10 or 26 AFC
	Variable size	0.10 log steps	5 decisions per level
Regan Charts	25%, 11%, and 4% contrast letters	~1.10 logMAR range	10 or 26 AFC
	Variable size	0.10 log steps	8 decisions per level
SKILL chart	Low luminance, low contrast chart	~1.30 logMAR range 0.10 log steps	10 or 26 AFC 5 decisions per level

AFC, Alternative forced choice.

acuity charts for children and adults are also available from www.precision-vision.com.

SKILL Low-Luminance Low-Contrast VA

The Smith-Kettlewell Institute Low Luminance (SKILL) chart consists of two near VA charts mounted back to back.⁴ One side has a chart with black letters on a dark gray background that is designed to simulate reduced contrast and luminance conditions. The other side has a high-contrast, black-on-white letter chart. The chart provides a measure of low-luminance low-contrast VA and also a difference score (i.e., the difference in VA between the two chart sides). The difference score may not be as useful a measure as the low-luminance low-contrast VA, at least for patients with optic neuritis.¹⁰⁰ Repeatability is as good as that of standard VA,⁴ and the test has been shown to predict future high-contrast VA loss.¹⁰¹

Melbourne Edge Test

The Melbourne edge test is a 30 × 25-cm chart that contains 20 discs of 25-mm diameter arranged in four rows.

Each disc has an edge that decreases in contrast from the top to the bottom of the chart. The patient must indicate whether the edge on each disc is vertical, horizontal, or oblique (45 or 135 degrees). Edge detection is considered to be related to the peak of the CS curve. Contrast levels are from 1 to 24 dB (approximately 0.10 to 2.40 log CS), with step sizes being 2 dB for the top row of five discs and 1 dB for the remaining three lines. The test has been shown to be reliable,¹⁰² and it correlates well with laboratory measures of activities of daily living¹¹ and the likelihood of falls.⁵ A new version of the chart holds 15 discs on three rows, with the first two rows having a step size of 2 dB; it is available from the National Vision Research Institute of Australia.

CSV-1000 Charts

VectorVision supplies a range of CS tests, with the most popular being the CSV-1000E (Figure 8-11). This test measures CS using sine-wave gratings at four spatial frequencies (3, 6, 12, and 18 c/deg). For each spatial frequency, there are two rows of eight columns of circular

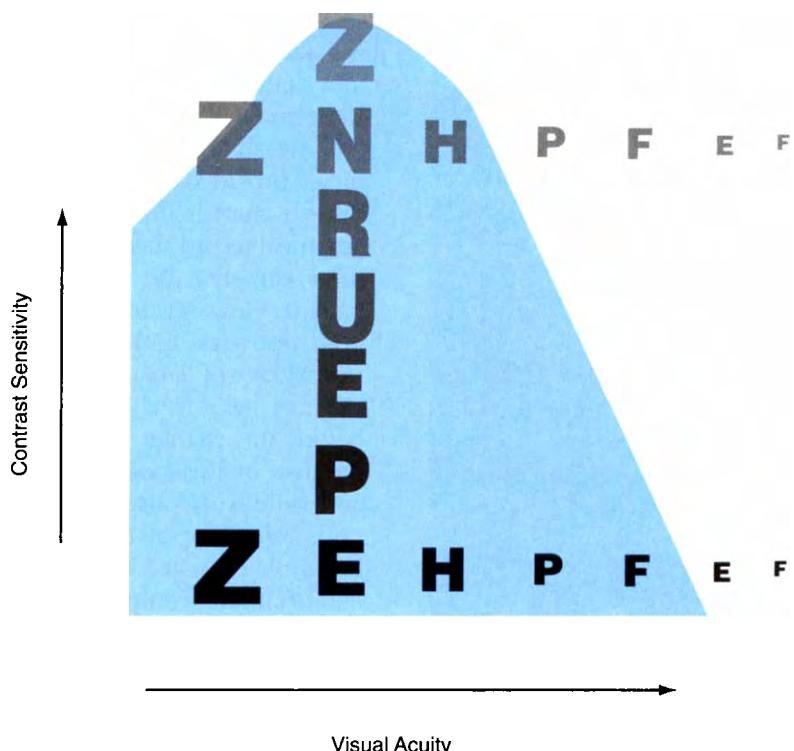


Figure 8-9

A schematic representation of the difference between letter contrast sensitivity measurements (e.g., Pelli-Robson charts) and low-contrast acuity measurements (e.g., Bailey-Lovie and Regan charts). The shaded area represents a typical contrast sensitivity function.

plates. One of each pair of plates contains a sine-wave grating, and the other contains a blank plate of equivalent mean luminance. The contrast of the plate with the grating decreases across the columns, with an irregular step size of about 0.16 log units. The patient is asked to indicate whether the top or bottom row contains the grating, but the patient can respond "I can't see a grating in either." The plate furthest along the row that has a position that is correctly identified provides a CS score for that spatial frequency. The chart therefore partly uses a criterion-dependent method and partly a forced-choice one. It is criterion dependent because the patient is allowed to decide at what point he or she cannot see a grating (cautious observers may give slightly low CS values), but there is a 2AFC check on risk takers, because they must indicate the position of the plate with the grating. VectorVision also supplies the CSV-1000S, which assesses CS in the same way at two spatial frequencies (6 and 12 c/deg). The reported good repeatability value of the CSV-1000E (± 0.19 log CS)¹⁰³ is surprising given its test design features, which are similar to the Vistech and FACT tests (see Table 8-1); further evaluation is required. These tests have the advantage of being internally illuminated with a self-calibrating system, and they are available from www.vectorvision.com.

Vistech and FACT CS Charts

The Vistech CS chart (renamed the Sine Wave Contrast Test, Figure 8-12) was first introduced by Dr. Art Ginsburg in 1984. It can be used in either a distance (VCTS 6500) or near format (VCTS 6000). The distance system is a 93 × 68-cm chart that is hung on the wall at 3 m. The chart contains circular photographic plates arranged in five rows and nine columns. Each plate contains a sine-wave grating, and each row has a different spatial frequency, with the contrast decreasing across the columns. The step sizes are irregular, but the average step size is about 0.25 log units. The gratings are either vertical or tilted 15 degrees to the right or left. The patient is asked to look along each row in turn, indicating the orientation of each grating, and he or she is instructed to respond "blank" if nothing is seen. This test partly uses a criterion-dependent method and partly a forced-choice one. It is criterion dependent because the patient is allowed to decide at what point he or she cannot see a grating (cautious observers may give slightly low CS values), but there is a 3AFC check on risk takers, because they must indicate the orientation of the gratings. The plate furthest along the row that has an orientation that is correctly identified provides a CS score for that spatial frequency. The spatial frequencies are 1.5, 3, 6, 12, and 18 c/deg. The near system uses a



Figure 8-10

The Bailey-Lovie 10% (Michaelson) contrast charts for logarithm of the minimum angle of resolution (logMAR). VAR, Vision acuity rating. (Courtesy of Dr. Ian Bailey.)

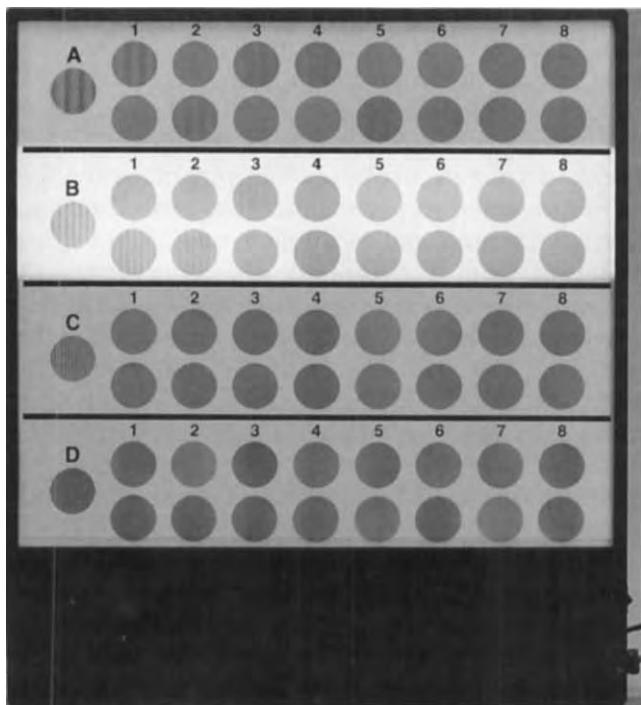


Figure 8-11

The CSV-1000. (Courtesy of VectorVision.)

smaller 17.5×14 -cm chart in the same format, which is placed 40 cm from the patient's eye in a unit similar to a Maddox wing or Holmes stereoscope. The system is externally illuminated, and the luminance levels used for measurement can be checked using the light meter provided with the system. An advantage of the Vistech chart is that the results can be plotted on the graphical record sheet, with its shaded normal area, and these can then be shown to and discussed with the patient. However, the Vistech charts consistently show poor test-retest correlations of between 0.25 and 0.61 (an average of 0.48),^{43,55,64,72,104,105} and reliability is poor. CS may have to change by more than 0.56 log units before the change can be considered significant.^{64,105} Because of this poor reliability, discriminative ability and validity are also poor.^{64,106} However, note that the poor reliability of the original Vistech charts is only known because it has been the most rigorously assessed and that the comparative reliability of other charts (e.g., FACT, CSV-1000) should only be claimed after they have been similarly assessed. The system is still commercially available from Stereo Optical (www.stereooptical.com).

The Functional Acuity Contrast Test (FACT) is a second-generation Vistech chart⁷⁵ (Figure 8-13). It differs from the original Vistech in that it has "blurred" grating patch edges, with the gratings smoothed into a gray background; it has a larger patch size so that an increased number of cycles is presented at low spatial frequency; and it uses equal 0.15 log CS step sizes, which are smaller than those of the original (average 0.25 log). This last chart design feature was made to improve the reliability over the original Vistech, and the FACT does provide slightly more repeatable results.⁷² However, because of the reduction in step size (while retaining the same number of steps as the Vistech chart), the range of contrast levels is reduced (see Table 8-1), and the chart suffers from significant floor and ceiling effects.⁷² This limits its usefulness when assessing CS at near-normal levels, such as after refractive surgery.⁷² The ceiling effect is increased by the use of a strict 3AFC technique (as recommended by the manufacturer), and it is probably better to allow a "blank" or "I can't see anything" option, as with the original Vistech, so that the testing method becomes partly criterion-dependent but with a 3AFC check on risk takers. The FACT is also available as one of several tabletop Optec vision testers, and it is available from www.contrastsensitivity.net and www.stereooptical.com.

How Many Measurements of CS Are Needed?

Given the channel theory of Campbell and colleagues, it is not surprising that the clinical CS research conducted subsequent to this fundamental research used sine-wave grating targets to measure the CSF at several spatial frequencies. It was hoped that different eye

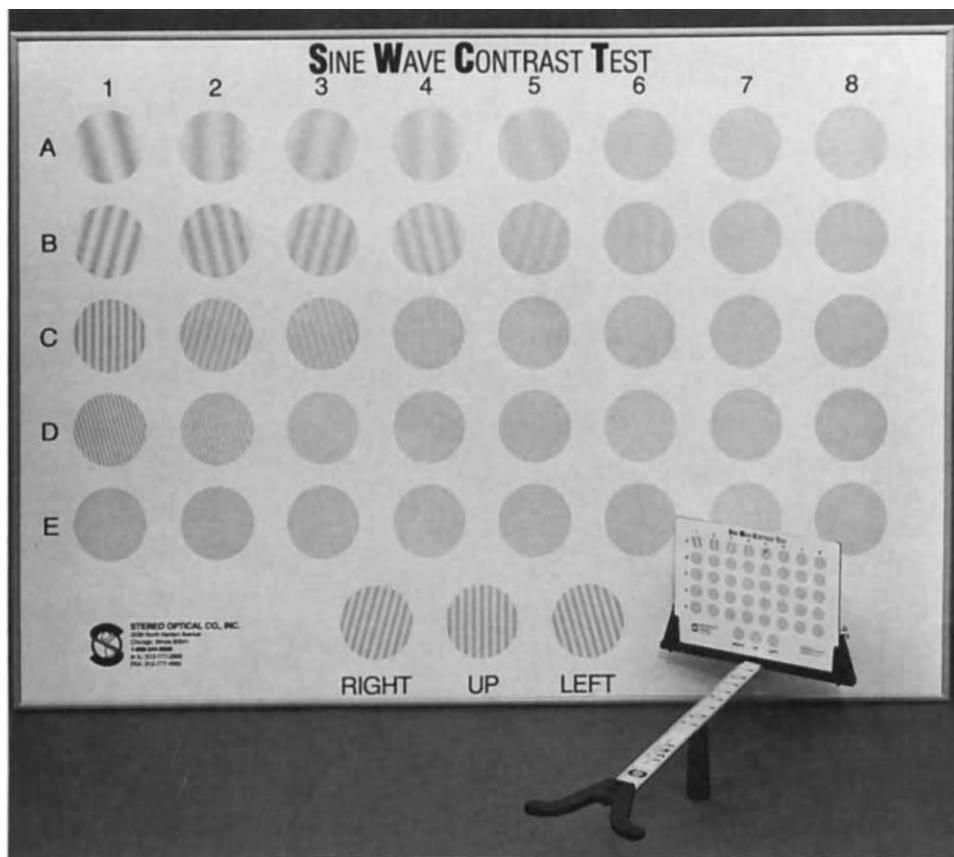
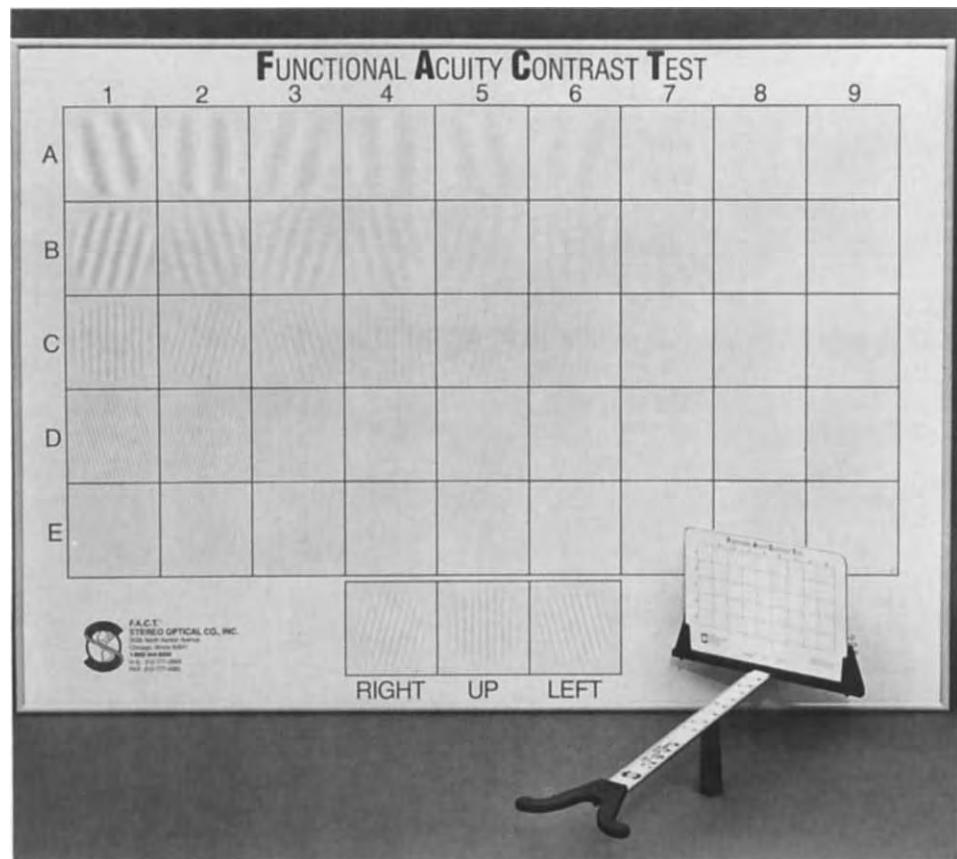


Figure 8-12

The Sine Wave Contrast Test distance and near charts. (Courtesy of Stereo Optical Company.)

diseases could perhaps have their own pattern of CS loss—an individual signature. However, this did not materialize, and the argument favoring measuring each of the four to six contrast threshold channels within the CSF does not appear to be valid. Pelli and associates¹⁰⁷ reported that the widely differing CS functions of a group of low-vision and normal subjects could be fitted reasonably accurately by the same parabolic curve shifted horizontally and vertically and could be predicted from measurements of CS of three degree letters and VA alone; this formed the rationale behind the chart design for the Pelli-Robson¹⁰⁸ and Cambridge gratings.⁸⁵ However, on another group of elderly subjects, Rohaly and Owsley¹⁰⁸ were unable to replicate their findings, and they suggested that a combination of the Pelli-Robson chart and VA was insufficient to assess the whole CSF. Other researchers have taken a different approach and looked at the information provided by a commonly used grating CS test, the Vistech, which measures CS at several spatial frequencies.^{66,72,104} Correlation coefficients between neighboring spatial frequency results were high, with an average r of 0.67 (compare this with the test-retest results of the Vistech of between 0.25 and 0.61; see Evaluating Clinical Con-

trast Sensitivity Tests); this suggests that much of the information provided by neighboring spatial frequencies is superfluous. Further statistical analysis indicated that the five measurements of the Vistech could safely be summarized by two scores—one at low spatial frequency and the other at high—and that the high-spatial-frequency factor was highly correlated with VA.^{66,72,104} These assessments of the results from the Vistech and FACT charts suggest that the rationale behind the design of the Pelli-Robson chart is a reasonable one. If VA measurements provide sufficiently sensitive information about the high-spatial-frequency end of the CS curve, only one other measurement is needed at a low to medium spatial frequency. Arguments made against the Pelli-Robson chart—that it only makes one measurement of the CSF,^{39,75} that it misses some type 1 CS losses,³⁹ and that it cannot reflect real-world vision⁷⁵—ignore the fact that the chart was designed to *supplement* high-contrast VA measurements. The chart cannot be judged on its own but only in conjunction with high-contrast VA. The combination of a Pelli-Robson CS and a high-contrast VA measurement measures *two* points on the CSF. For example, a type 1 CS loss would show a reduced VA and normal Pelli-Robson CS, a type 2 CS



hand of the patient is being used to occlude the other eye, care should be taken to use the palm; otherwise the patient might look through a gap between the fingers. It is traditional to measure the right eye first, but the left eye might occasionally be measured first if it is known that the patient has poorer vision in that eye. In some situations, it is useful to measure binocular CS. With the letter charts, ask the patient to read the lowest letters that they can see, and encourage them to guess. Encouragement appears to be particularly important for the letter CS tests. Generally, if a patient is given sufficient time and encouragement when looking at threshold, at least one more triplet of letters becomes visible on the Pelli-Robson chart. The letter C is often misread as an O on this chart; to balance the legibility of the letters, this should be counted as a correct response.¹¹¹

Letter charts such as the Pelli-Robson and low-contrast acuity charts, which have a regular logarithmic progression of step size and the same number of letters per step, can be scored per letter. So, if the progression is 0.15 log CS units and there are three letters per step (as there are on the Pelli-Robson chart), each letter is worth 0.05 log units.⁷ Each triplet of letters on the Pelli-Robson has a log CS level printed next to it on the score sheet. Take the CS score of the lowest triplet in which a letter was correctly read, and subtract 0.05 log units from this value for each letter incorrectly read at and before this triplet to calculate the final score. For the low-contrast letter charts, the progression tends to be 0.10 logMAR units, so each of the five letters of the Bailey-Lovie charts is worth 0.02 logMAR units, and each of the eight letters of the Regan charts is worth 0.0125 logMAR units. The letter charts can be scored per line, as Snellen charts are (score according to the lowest line in which a majority of letters are called correctly), but reliability is reduced.^{67,71}

The most common errors made when measuring the letter CS or low-contrast VA charts are using inappropriate illumination (generally too low or not uniform), using an occluder inappropriately (e.g., the patient can see the chart binocularly), not pushing the patient to guess, and not giving them time to look at letters near threshold.

Interpretation of Test Results

Different Contrast-Sensitivity Values from Different Tests

CS charts vary considerably in their test design and target parameters, and CS results from different tests do not compare well with others. For example, even when CS tests are very similar (e.g., the FACT and its predecessor, the Vistech chart), different CS values are produced. The FACT test provides higher CS results at low spatial frequencies as a result of the larger area (and greater number of gratings) at low spatial frequencies.¹⁰⁶

However, at higher spatial frequencies, the FACT chart gives lower CS results, because the data are truncated as a result of the reduced range. Woods and Thompson¹¹² also showed that the psychometric method and presentation procedure affected not only the absolute values of the CSF but also its shape. CS values also differ depending on whether Weber (Pelli-Robson) or Michelson contrast (all grating charts) is used.

The Development of Contrast Sensitivity

Infant CS has been examined using a variety of techniques, including visually evoked potentials, preferential looking, and oculokinetic nystagmus.¹¹³ The overall CSF increases throughout the first year of life, and peak sensitivity shifts toward higher spatial frequencies. The highest spatial frequencies reach adult levels later than the lower frequencies do,¹¹³ and this may be part of the reason that amblyopia preferentially affects higher spatial frequencies.¹¹⁴ There is little consensus as to when the CSF reaches adult level, with figures varying from over 3 to 10 years of age.¹¹³

Contrast-Sensitivity Changes Throughout Adulthood

Most recent reports using stationary gratings indicate that CS at medium and high spatial frequencies decrease in normal, healthy, aging eyes.¹¹⁵ The cause or causes of this loss in CS with age can be broadly categorized into optical and neural changes. Optical changes include reduced retinal illuminance (as a result of age-related miosis and increased lenticular absorption) and increased intraocular light scatter. A 20-year-old eye transmits about three times as much light as a 60-year-old eye. Neural changes include neural cell loss and degeneration, neurotransmitter changes, and lipofuscin accumulation, and these changes have been shown to occur from retina to cortex.¹¹⁵ The majority of CS loss with age appears to be the result of neural changes,^{115,116} with optical factors only having a significant effect at high spatial frequencies.¹¹⁷ Any reduction in CS caused by reduced retinal illumination appears to be offset by an improvement in CS caused by the smaller age-related pupil reducing aberrations.^{115,118} Most clinical CS tests also show an age-related decline in score throughout adulthood,^{66,85} and some charts provide age-related normative data⁸⁵; no normative data is currently provided with the Pelli-Robson chart. Using the scoring rules mentioned earlier, the 95% lower limits of normal scores (mean – 1.96 × SD) are found to be 1.65 log units for patients between 20 and 50 years old and 1.50 log units for patients more than 50 years old,⁶⁶ but it is better to obtain personal normative data (see the next section). The Vistech chart provides normative data, which are used for all age groups; this is because, in his original evaluation, Ginsburg³⁶ found no significant age change in results of the test, although other reports indi-

cate significant reductions in CS with age at medium and high spatial frequencies on the Vistech chart.⁶⁶

Collecting Personal Normative Data

Clinicians differ in the values they obtain using any chart that requires subjective responses, including CS charts⁶⁶; this is perhaps because of differences in the amount of encouragement given to patients to read letters or gratings near threshold so that cautious observers are allowed to determine their own threshold. Normative data provided with a chart may not compare with the data clinicians would obtain themselves from patients with normal vision. It is therefore advisable for clinicians to obtain their own normative data; this provides good experience with the equipment and the scores that are obtained. Because of probable age changes in score, measurements should be collected from patients of each decade of life. Taking a mean for each decade gives an average score, and the mean \pm (2 \times SD) or the mean \pm (3 \times SD) provides the outer limits of normal scores and sets specificity at 95% or 99%, respectively.

What Is an Abnormal Score When Measurements Are Obtained from Several Spatial Frequencies?

The answer to this question seems simple. With the Vistech, for example, any point that falls below the normal gray area on the record card is presumably abnormal. If this analysis was used, many abnormal points would be obtained. This is because the gray area represents the 90% limits of normal so that, for any single spatial frequency, 5% of observations might be expected to fall below the lower limit of normal (and 5% above). There is a $1 - (0.95)^5$ probability (23%) that one of the five will be reduced below normal because of chance alone. When using the Vistech (or FACT), it is probably not necessary to measure CS at the highest spatial frequencies, because this information is more reliably provided by a Bailey-Lovie type VA chart.^{66,72} Take the score on the Vistech or FACT to be abnormal if two of the three low-frequency scores are outside of the gray area. $1 - (0.95)^3 = 14.3\%$, so there is a 2% probability that two of the three values will be below the gray area as a result of chance. This seems to be an acceptable level of false-positive results in many situations, because most charts give the 2.5th percentile as the lower limit of normal.

Clinical Uses (and Abuses)

The Cambridge gratings manual suggests that the test can detect deficits, whereas more conventional tests fail to do so in patients with cataract, retinal pathology, glaucoma, retrobulbar neuritis, multiple sclerosis, and diabetes. The Vistech manual claims that the Vistech CS

test can provide useful clinical information in patients with cataract, glaucoma, amblyopia, contact lenses, low vision, visual pathway disorders, refractive surgery, and refraction. Is this all true? How much is the manufacturer's hype (or hope)? In the following sections, the usefulness of CS is listed in order of the amount of agreement in the literature; the sections listed first are those with the most agreement, whereas those listed last appear to have the least agreement. The opinion of the usefulness of CS testing suggested here is more optimistic than some¹¹⁹ yet more pessimistic than others.^{75,120} The use of CS as part of a glare test is discussed in later sections about glare testing.

Clinical Trials

CS measurements are now widely used in clinical trials of ocular drugs and ophthalmic treatments and instruments. For example, CS tests have been widely used in the assessment of refractive surgery^{21,22} and in clinical trials of treatments for cataract,²³ age-related macular degeneration,²⁴ and optic neuritis.^{25,26}

After Refractive Surgery

After refractive surgery, patients can suffer from visual problems that are identified by CS and low-contrast VA but not by traditional high-contrast VA.^{15,121} A significant number of patients complain of difficulty seeing at night, particularly when driving.^{80,121} These reductions in CS are caused by a combination of increased optical aberrations and increased forward light scatter. Tanabe and colleagues¹²² reported that the deterioration of low-contrast VA after photorefractive keratectomy is mainly attributable to increases in wave front aberration and not to light scatter (or "corneal haze"). If this is the case, then postoperative reductions in vision will be best measured using CS and/or low-contrast VA rather than disability glare measurements, which only assess the effects of light scatter. It has been suggested that CS should be measured under mesopic conditions⁸¹ or with a pharmacologically dilated pupil¹²¹ to best identify the effect of aberrations after photorefractive keratectomy, because the aberrations increase dramatically with a dilated pupil. The U.S. Department of Treasury and the U.S. Customs Service currently assess all applicants to federal agencies (e.g., the FBI) who have had refractive surgery using the ETDRS VA chart, the Pelli-Robson CS test, and the brightness acuity test under dilated and undilated conditions.⁸⁰ The effects of glare, star bursts, and halos after refractive surgery are discussed in later sections.

Low-Vision Examinations

CS appears to be a better predictor of reading speed than VA,¹²³ and reduced CS can explain a poor response to an optical aid by a low-vision patient and suggest

the need for a contrast-enhancing closed-circuit television. Indeed, Whitaker and Lovie-Kitchin¹²⁴ suggest that poor CS measurements are a useful indicator that a low-vision patient will not benefit from optical devices. CS can indicate what everyday tasks the patient is likely to have trouble with and help the practitioner with determining what additional rehabilitative strategies are needed.¹²⁵ It can be more useful to provide a monocular optical aid to the eye with the best CS rather than the eye with the best VA.¹²⁶ In addition, the presence of binocular CS summation may suggest the need for a binocular low-vision aid rather than a monocular one.¹²⁷ Patients with reasonable VA but reduced CS could be advised about various daily living tasks that could be improved by adaptations to increase the task's contrast. For example, the contrast of electrical wall outlets and light switches can be increased by changing the color to contrast with that of the wall; "whitish" food (e.g., potatoes, chicken, fish) can be more easily seen on a dark plate, which can be more easily seen on a white place mat or tablecloth, and so on.

Justifying Cataract Referral in a Patient with Good Acuity

The decision of when to refer patients for cataract surgery should not be based on a particular VA score but rather on when their reduced vision is affecting their desired lifestyle.¹²⁸ CS measurements can help by providing useful additional information about the patient's real-world vision.^{89,90} This includes driving performance⁸; CS losses in cataract patients appear to be highly indicative of crash involvement when driving, whereas VA and disability glare are not.⁹ Drivers with a history of crash involvement were eight times more likely to have a serious contrast sensitivity deficit in the worse eye (defined as a Pelli-Robson score of 1.25 or less) than those who had not been involved in a crash. Crash-involved drivers were six times more likely to have severe contrast sensitivity impairment in both eyes than were crash-free drivers. A reduced low-frequency CS in a patient with reasonable VA (better than 20/50 or 6/15) who is experiencing significant problems can help to justify referral. Consider the following example:

Example 1: A 68-year-old homemaker had extensive cortical cataract in both eyes and symptoms of great difficulty recognizing friends, reading, and knitting, with much worse vision in bright sunlight. VAs of OD: 20/20⁻² (6/6⁻²) and OS: 20/25⁻¹ (6/7.5⁻¹) were excellent, but reduced Pelli-Robson scores of OD: 1.05 and OS: 1.35 log CS provided justification for surgery. The right cataract was extracted (i.e., the eye with the worse CS rather than the one with the worse VA), and this significantly improved visual ability.

A normal low-frequency CS in a patient with poor VA (perhaps 6/18 or worse) can also explain why a patient

is not experiencing serious problems and need not necessarily be referred.¹²⁹

Explaining Symptoms of Reduced Vision in a Patient with Good VA

CS at low and intermediate spatial frequencies can be reduced when VA is normal in patients with visual pathway disorders, diabetic retinopathy, or glaucoma (see Various Types of Contrast-Sensitivity Loss in Patients). It can also provide important information about real-world vision (see Assessment of Real-World Vision) so that it can be used to explain symptoms of poor vision in patients with good acuity. This can be important to patients who have been told that their vision is fine (because of good VA) when they know that it is not. The following is an example from an optometric practice study¹²⁹:

Example 2: A 45-year-old female was diagnosed with multiple sclerosis 3 years prior to her visit. She has complained of reduced distance vision for the last 2 years. During this period, the patient had been examined by two optometrists and one ophthalmologist and had been told that her vision was fine. Her spectacles had not been altered. There were no significant ophthalmic findings. LogMAR VA in the right eye was -0.06 (20/17 or 6/5), and Pelli-Robson CS was 1.50 log units and abnormal for age (lower limit of 1.80 log CS; this is a type 3 CS loss, see Figure 8-5). Although the patient's vision could not be improved, she was delighted that someone finally agreed that her vision was not normal and was able to explain the situation to her. The patient was counseled that her vision would probably be slightly worse in low-contrast (and probably low-luminance) situations, such as at dawn, at dusk, in fog, or in heavy rain.

Should Contrast Sensitivity Be Measured in Diabetics?

CS can be reduced in diabetics without visible retinopathy²⁰ or maculopathy¹⁹ as compared with age-matched patients with normal vision, and CS can help with identifying early ischemic diabetic maculopathy.¹³⁰ Some studies have found significant correlations between some diabetic metabolic indices and CS loss,^{131,132} and CS has been shown to improve in diabetic patients with little or no diabetic retinopathy after improved metabolic control.¹³³ CS may therefore be a useful measurement for diabetics in that reduced CS without visible eye disease may indicate subclinical diabetic eye disease requiring careful monitoring^{19,20} and/or a need to improve metabolic control. Further research is required in this area. Determining CS loss as a result of diabetic changes in the elderly is complicated by CS losses due to such common eye diseases such as cataract and ARM.

Part of a Battery of Tests to Screen for Visual-Pathway Disorders

In many cases of visual-pathway disorders, VA and ophthalmoscopy can be normal, whereas CS at low frequencies is reduced. Although some of this research suffers from lenient definitions of "normal" VA (i.e., 20/20 or 6/6 or worse, when normal VA in young patients is typically much better than that; see Chapter 7), other studies have shown excellent levels of VA with CS loss. Normal VA and reduced CS have been found in patients with optic neuritis and multiple sclerosis, Parkinson's disease, papilledema, and compressive lesion of the visual pathways.⁴¹ In the optic neuritis treatment trial, Pelli-Robson CS proved to be a more practical (simple, quick, and reliable) and sensitive indicator of visual dysfunction than Humphrey VFs and the Farnsworth-Munsell 100-Hue Test.²⁶ CS was still abnormal in 46% of patients with normal VA and recovered optic neuritis. Because of the low incidence of such diseases and the fact that CS need not always be reduced, it is debatable whether routine CS screening by clinicians is worthwhile. However, because of the speed and ease of CS measurement and the relative inexpensiveness of CS charts—in combination with the possible drastic outcome of certain visual pathway disorders—some clinicians may feel that using CS as part of a battery of tests in routine screening is worthwhile. An example of the usefulness of CS in this regard follows¹²⁹:

Example 3: A 21-year-old female had headaches that seemed to be associated with close work. There were no significant ophthalmic findings and no significant refractive error. Snellen VA was 20/20⁺³ (6/6⁺³) OD and OS, but letter-chart CS was 25% of normal value, and the Vistech chart showed reduced CS at 1.5, 3, 6, and 12 c/deg and normal CS at 18 c/deg, which is consistent with normal VA. Central VFs revealed a bitemporal field defect and a possible chiasmal lesion.

Providing Additional Information About a Patient's Visual Function

CS levels at low spatial frequencies or at the peak of the CSF provide useful additional information beyond VA about real-world vision. Two patients could have the same VA (e.g., 20/40 [6/12]), yet one patient could have CS loss only at high spatial frequencies (hence the VA loss) and the other could have CS loss at all spatial frequencies. Despite having the same VA, the second patient is much more likely to suffer from (and complain about) visual problems. In patients with reduced VA, CS measurements can therefore be used to help explain symptoms of poor or deteriorating vision. One of several examples from Elliott and Whitaker¹²⁹ follows:

Example 4: An 85-year-old woman with multiple sclerosis was complaining about significant reduc-

tions in her distance and near vision since her last examination 2 years ago. She now had difficulty reading. Extensive cortical cataracts were found in both eyes. Her spectacle prescription was unchanged, and her logMAR VAs were as follows: OD, 0.28 and OS, 0.30 (20/40⁺¹ or 6/12⁺¹, and 20/40 or 6/12). Her Cambridge CS in the right eye was 24 as compared with a lower limit of normal for her age of 121. At her examination 2 years previously, the VAs were also 20/40 (6/12) in both eyes. Unfortunately, no CS measurements were made at that time, and so a comparison of CS scores was not possible. However, the low CS scores at this visit could account for her subjective decrease in vision. Woo has presented a similar case history.¹³⁴

Screening and Monitoring Glaucoma Patients

One of the earliest suggested clinical roles for CS was that of screening for POAG.⁸³ The value of currently available CS tests for POAG management is still to be determined¹⁰³; it may help to fully describe a glaucoma patient's visual disability,¹ but it does not appear to have a major role in screening.¹³⁵ This is particularly true in the elderly population, in whom CS losses could be due to cataract, diabetes, or age-related macular degeneration, as well as POAG. The Vistech¹³⁶ and Pelli-Robson charts¹³⁷ reveal no significant difference among POAG, ocular hypertensive, and control patients. Clinical CS tests seem to be about as sensitive (and specific) to glaucoma as traditional high-contrast VA.¹³⁵

Are Contrast-Sensitivity Measurements of Value in Contact Lens Wearers?

Claims have been made for the value of CS measurement in contact lens wearers.¹²⁰ There may be truth in these claims, but this area of research is another sufferer of the use of 20/20 (6/6) and worse as an indication of normal VA. Vision loss in hydrophilic lens wearers may be the result of subtle corneal edema, and Pelli-Robson CS and VA testing (even in the presence of glare) can only detect edema of greater than 9%.¹³⁸

Can CS Predict Future Vision Loss?

Schneck and colleagues¹⁰¹ recently reported that the low-contrast low-luminance SKILL chart was a significant predictor of subsequent high-contrast VA loss in individuals who had fairly good initial VA. They found that 55% of those in the worst category of low-contrast low-luminance VA at baseline subsequently had significant high-contrast VA loss several years later as compared with none of those with good initial low-contrast low-luminance VA. They suggested that this was because low-contrast tests are more sensitive to subclinical pathology present at a first test and that, at subsequent visits, this became frank pathology that even affected high-contrast VA. These findings suggest that CS or low-

contrast VA tests can be used to help identify patients at high risk for vision loss and who should perhaps be examined more thoroughly and recalled sooner for repeat examinations.

Is Contrast-Sensitivity Measurement of Any Value in Amblyopia Patients?

Although CS has proved invaluable for determining the underlying etiology and development of amblyopia, it does not seem to have accrued any clinical role at present. For example, although widely cited in the "basic aspects" chapters of Cuiffreda and colleagues' 1991 amblyopia textbook, it is not mentioned in any of the clinical chapters. The oft-cited categorization of CS loss in amblyopia by Hess and Howell¹³⁹ may be anomalous.¹¹⁴ Some authors¹²⁰ have suggested that monitoring CS in patients undergoing therapy can be useful, because a patient may gain improved CS with no improvement in VA.

Contrast-Sensitivity Measurement in Sports Vision

A large volume of sports vision research is dedicated to identifying visual skills related to sports performance and to determining if the visual skills of athletes are better than those of nonathletes. Many studies have lacked proper research design and techniques.¹⁴⁰ Athletic success depends on many factors other than vision, such as size, speed, coaching, level of experience, and ability to "read the game." These confounding factors make it difficult to evaluate an athlete's potential using the results of visual tests alone.¹⁴⁰ Furthermore, although some authors conclude that athletes have certain visual skills that are superior to those of nonathletes, others disagree with this conclusion. Also, those authors who have found relationships between visual abilities and athletic achievement do not report strong correlations.¹⁴⁰ Use of varying measures and instrumentation and a lack of consistency in the classification of athletes versus nonathletes among researchers may contribute to the problem. Standardization is required. In particular, criterion-dependent CS measurements must not be used to evaluate the efficacy of visual training, because improvement in CS could arise as a result of a shift in the subjects' decision criteria rather than a physiological change in "sensitivity."¹⁴¹ In particular, such changes in decision criteria could occur with the positive mood changes that have been found to occur after exercise.¹⁴¹ A further problem is the rationale for the choice of tests. CS may be of predictive value for whether a baseball player will be able to see (and subsequently catch) a fly ball against a dull or cloudy sky. However, why should it predict how good a hitter he or she will be? To hit the ball, the batter must predict the instant (to within a few milliseconds) that the ball will pass through a volume of space perhaps only 5 cm in

diameter¹⁴²; no visually guided modification to the swing is possible during the later stages of the ball's flight (possibly as long as 200 msec before the moment of contact between bat and ball).¹⁴³ It may be that testing a batter's ability to use the visual correlates of the ball's time of arrival and direction of motion might be more predictive of performance than some of the visual measures that have previously been used. A case in point is that interindividual differences in the flying performance of pilots for a variety of flying tasks correlated more closely with the results of visual tests that are closely related to the task (e.g., sensitivity to retinal image expansion) than with the results of more general tests of visual function, such as Snellen acuity, CS, and stereoaucuity.¹⁴⁴

Can Contrast Sensitivity Be Used During the Subjective Refraction?

Although some manufacturers have claimed that high-spatial-frequency CS can be used to measure refractive error as accurately and quickly as with VA charts, they do not appear to be used by eye-care practitioners. VA measurements appear to be perfectly designed for use in determining refractive error, as will be noted in Chapter 20. For example, they have been shown to be more sensitive to refractive blur than high-frequency Vistech CS results.¹¹⁰

GLARE TESTING

Background Information

Definitions

Lucretius provided a description of glare nearly 2000 years ago: "Bright things the eyes eschew and shun to look upon; the sun even blinds them, if you persist in turning towards it, because its power is great and idols are borne through the clear air with great downward force, and strike the eyes and disorder their fastenings." A good definition of glare is "a strong unpleasant light." Glare sources can be direct, such as the sun and lamps, or indirect, such as surfaces that are too bright. The latter includes reflections of primary sources in glossy materials or off of water (i.e., veiling reflections).

There are three main types of glare: (1) disability glare, (2) discomfort glare, and (3) light-adaptation glare. *Disability glare* is the loss of visual function that occurs because of a peripheral glare source, and it is the most commonly used clinical measure of glare; a common example is the loss of vision that occurs when light hits a dirty windshield. *Discomfort glare* is the feeling of discomfort in some bright-light situations. This could be the result of looking into a steady high-luminance field, such as reading a book in bright sunlight or being in an environment in which bright,

nonshielded light sources are within your field of view. This discomfort can cause a loss of vision by an evasive action, such as when looking away from undimmed car headlights when driving.¹⁴⁵ The discomfort is probably a result of spasm of the iris sphincter,¹⁴⁶ and it is dramatically illustrated in a patient with iritis. Although it is of great importance when driving and with regard to ergonomics, discomfort glare will not be discussed further, because it is difficult to quantify, and there are no commercially available clinical tests. *Light-adaptation glare* is the reduction in vision caused by the afterimage of a glare source producing a central scotoma after directly looking at a bright light. Light adaptation glare therefore can remain even when the glare source has gone (unlike disability glare). This glare is due to the light adaptation of the photoreceptors, and it can become significantly disabling in patients with macular problems (see Light-Adaptation Glare).

Most of the remaining text concentrates on disability glare, because it is the type of glare that is most commonly measured clinically.

Neural vs. Optical Etiology for Disability Glare

The arguments regarding whether disability glare has an optical or neural etiology go back as far as Goethe in 1810 (neural) and Purkinje in 1823 (optical). Disability glare is the result of either the strong peripheral light causing lateral inhibitory effects in the retina or of intraocular light scatter (straylight) causing a veiling luminance and reducing the retinal image contrast. Disability glare was first evaluated using the technique of measuring "equivalent luminance" or "equivalent veil."¹⁴⁷ The technique involved imitating the masking effect of a peripheral glare source on a target using a uniform veiling light. The illumination at the observer's eye as a result of the glare source (E_{gl}) was varied, and the equivalent luminance (Leq) needed to produce a similar loss of detectability of the target was measured. If there was a simple linear relationship between E_{gl} and Leq , it was argued that the cause of the disability glare could only be intraocular straylight: with increasing amounts of illuminance from the glare source at the eye, there would be a corresponding increase in the amount of straylight and therefore in the amount of reduction in retinal image contrast. This would not be the case if disability glare were caused by neural inhibitory effects. Linearity between E_{gl} and Leq was found, and it was determined that disability glare was due totally to the effects of straylight (at least for glare angles greater than 1 degree).¹⁴⁷ More recent results have questioned whether there is a neural component in some clinical glare tests.^{148,149} This question has arisen as a result of an article that found a nonlinearity of E_{gl} and Leq with a more clinical disability glare measurement¹⁵⁰ and other reports of an improvement in CS with some glare tests (a negative disability glare). However, if an appropriate test design is used (see Ideal Design Features for

Clinical Glare Tests), linearity between E_{gl} and Leq can be obtained with a clinical disability glare setup,^{151,152} and such glare tests should only be assessing the effects of light scatter.

Ocular Media Transparency

Maurice's¹⁵³ lattice theory suggests that, because the collagen fibers of the cornea are parallel, equal in diameter, and have their axes disposed in a regular lattice arrangement, each row of fibers acts like a diffraction grating. Interaction occurs between scattered light from individual fibers in the form of destructive interference, with two out-of-phase light waves from neighboring fibers canceling each other out (see the beginning of Chapter 26). This takes place in all directions (except that of the incident beam), producing a high-intensity maximum, surrounded by a small number of very-weak-intensity, lower-order maximums. If the spacings between the fibers are decreased, the angles through which the lower-order maximums are deviated increases, and fewer are formed. When the spacing is equivalent to the wavelength of light, only the central maximum remains, and the "grating" appears transparent. The cortex of the lens also retains its transparency as a result of a regular lens fiber lattice arrangement that compensates for the light scattering caused by differences in refractive index between fiber membranes and cytoplasm.¹⁵⁴ In the lens nucleus, there are only minor differences in refractive index between fiber membranes and fiber cytoplasm so that there is minimal scattering and no need for a regular lattice arrangement.¹⁵⁴

Miller and Nadler¹⁵⁵ used special inverse holograms to provide a clever illustration that ocular media opacification is caused by light scatter rather than the "stopping" or absorption of light. The holograms collected the scattered light from a cataract and recreated a sharp image. Light scatter occurs when the spacing between elements of different refractive indices become comparable with or greater than the wavelength of light. In this way, "lakes" in corneal edema and water clefts and vacuoles in the lens scatter light after they become comparable in size with the wavelength of light. Diffraction halos can be seen by patients with corneal edema caused by contact lenses and acute glaucoma¹⁵⁶ and after refractive surgery,¹⁵⁷ and the faint-colored halo that can be seen around streetlights and candles in dark surroundings is caused by lenticular diffraction.¹⁵⁸ Large-particle (or, more correctly, large-refractive-index fluctuation) light scatter also occurs when the elements themselves become comparable in size with the wavelength of light. There are few mitochondria and other large intracellular organelles in the lens, and those present are hidden behind the iris so as not to scatter light.¹⁵⁹ Light scatter in cataract probably occurs with changes in refractive index at the boundary of the lens fibers,¹⁶⁰ multiple scattering by

aggregations of protein molecules in the lens nucleus,¹⁶¹ Mie scattering by multilamellar bodies in nuclear cataract,¹⁶² and the posterior migration of epithelial cells containing many large organelles in posterior subcapsular cataract.

Backward vs. Forward Light Scatter

The amount of light scattered by the ocular media can be assessed clinically by slit-lamp biomicroscope examination. This provides an assessment of backward light scatter (i.e., the amount of light scattered back from the eye toward the light source) (Figure 8-14). This is distinct from the light scatter that causes reduced vision, which is the light scattered forward onto the retina. Glare tests aim to provide an indication of the amount of forward light scatter. Backward-light-scatter measurements have the advantage of not requiring subjective responses from the patient, and they are independent of neural function. Unfortunately, although *in vitro* measurements of forward and backward light scatter have been shown to be well correlated,¹⁶³ this is generally not true of measurements in patients. A variety of

methods, including modified slit-lamp examination and the Interzeag Opacity Lensmeter, have shown a poor correlation between backward and forward light scatter in patients with cataract, particularly for subcapsular and cortical cataract.^{148,164} This lack of correlation may be partially due to "backscatter" measurements consisting of both real backscatter and specularly reflected light.^{165,166} Lohmann and colleagues¹⁶⁵ found that, when specularly reflected light was removed, backscatter correlated better with forward scatter. Interestingly, the contribution of specularly reflected light to backscatter is much less for nuclear cataract,¹⁶⁶ which may explain why backscatter measurements from both slit-lamp examination and the Opacity Lensmeter 701 correlate reasonably well with forward scatter in patients with nuclear cataract.^{148,166}

Light Scatter Changes with Glare Angle

Optical imperfections in the ocular media mean that a localized point source of light within the VF does not produce an equally well-defined point image on the retina. Instead, a proportion of light is scattered, and this forms the point-spread function (PSF). It is important to differentiate between the PSF for the optical media and the functional PSF that is relevant for vision and to consider the Stiles-Crawford effect, which limits the effect of wide-angle light scatter on foveal function.¹⁴⁹ Beyond a degree or so of visual angle from its center, the functional PSF declines in amplitude in approximately inverse proportion to the square of the visual angle.¹⁴⁷ This is called the Stiles-Holladay relationship:

$$\text{PSF}(\phi) = K/\phi^n \text{ for } \phi \text{ between 1 and 90 degrees}$$

where $K = 10$ and $n = 2$. Several other angular-dependency formulas have been suggested, with slightly different values of K and n .^{147,167} With increasing age and cataract, there is a relative increase in the amount of light scatter from the lens so that K increases, although the angular dependency remains similar.^{148,167} Within a degree of glare angle, the PSF is much steeper.¹⁴⁷

What parts of the eye scatter light? Slit-lamp biomicroscopy and ophthalmoscopy indicate that the cornea, lens, and fundus scatter the most light and that the aqueous and vitreous scatter little, if any. In a series of clever experiments, Vos¹⁴⁷ showed that, in young subjects, this intraocular straylight is produced in approximately equal proportions by the cornea, the lens, and the fundus. At small glare angles, light scatter in the cornea and lens should be held responsible, whereas, at very large angles, scattering by the fundus and possibly light leakage through the ocular wall in light-colored eyes may play a greater role.¹⁶⁸

The relationship of glare angle to light scatter is important clinically, because it raises the importance of this angle in glare tests. For example, imagine using a



Figure 8-14

A Scheimpflug slit-image photograph of an early nuclear cataract. The photograph is produced by the light scattered back out of the eye. (Courtesy of Dr. Mark Hurst.)

penlight as a glare source at a standard glare angle of 20 degrees. A clinician is unlikely to be precise at standardizing to this value, and it may be 10 degrees for one measurement and 30 degrees for another. Because light scatter is inversely proportional to the square of the glare angle, the first measurement should give nine times the amount of light scatter as the second ($1/10^2$ divided by $1/30^2$). Because the amount of illuminance from the glare source reaching the eye also depends on an inverse square law, the distance of the glare source from the eye is also critical.

How Does Light Scatter Affect Vision?

In normal, healthy eyes with little light scatter, straylight can be modeled using a scattering theory based on individual particles (called *Mie scatter*), which have dramatically different properties depending on whether the particles are large or small.¹⁶⁹ Small-particle scattering includes Rayleigh scattering, which preferentially scatters blue light (it scatters light in proportion to λ^4), and it scatters light a similar amount in all directions. Rayleigh light scattering by very small particles in the earth's atmosphere is responsible for the sky appearing blue. Sunsets are caused by the much greater distance that sunlight has to travel in the earth's atmosphere to reach the eye when the sun is close to or below the horizon so that large amounts of blue light are scattered, and the remaining direct, comparatively unscattered light is dimmed but enriched in reds and yellows. Large-particle scattering is wavelength independent so that light scattered by large water droplets in the sky (clouds, mist, fog) appears white. Large-particle scattering scatters far more in the forward direction than in the backward direction. In the eye, this would mean more light scattered toward the retina and less light scattered back out of the eye. As the number of light scattering particles increases (i.e., as it does in patients with more dense cataract or corneal edema), a single-scattering Mie model breaks down, and multiple scattering must be considered.¹⁶⁹ It is important from a clinical viewpoint to know how much light scatter in the eye is caused by Rayleigh scatter. Significant amounts of Rayleigh scatter would mean that blue-absorbing tints would be extremely useful to prevent disability glare. Rayleigh scatter would also give similar amounts of forward and backward light scatter, which would suggest that slit-lamp examinations could be used to indirectly evaluate forward light scatter and therefore disability glare. The cornea certainly scatters more blue than red light,¹⁷⁰ and it has a blue tinge under slit-lamp examination. Protein aggregates that are small enough to produce Rayleigh scatter accumulate in the lens, and it has been claimed that these are a major source of intraocular scatter.¹⁶¹ However, the vast majority of forward straylight is caused by larger scattering structures, such as refractive index fluctuations at lens fiber intersections¹⁷¹ and

multilamellar bodies in nuclear cataract.¹⁶² Certainly, the light scatter caused by cataract appears white under slit-lamp examination (the yellow-brown appearance of nuclear cataracts shown in Figure 8-14 is caused by pigments that absorb blue light; the light scatter in nuclear cataracts appears white). The angular dependency of straylight is also untenable with a significant amount of Rayleigh scatter,¹⁴⁷ and recent studies indicate that there are negligible amounts of wavelength-dependent scatter in normal and cataractous eyes.^{168,172}

One special form of light—or more precisely, radiation—scatter that is wavelength dependent is ultraviolet (UV) fluorescence. Specific wavelengths outside of the visible spectrum can produce the fluorescence of chromophore-containing proteins within the human lens. These "fluorophores" have activation wavelengths of approximately 350 nm and 430 nm, the latter resulting in emission wavelengths well within the visible spectrum, between 500 and 520 nm.¹⁷³ The visible light scatter produced by this fluorescence can act in the same way as a veiling luminance arising from a glare source. UV-induced ocular fluorescence has been shown to have a significant effect on visual function.^{173,174,175} Autofluorescence has been shown to increase with age and in patients with diabetes and nuclear cataract.^{176,177}

Scattered light reduces vision in two ways. First, light from the object itself is scattered and reduces the contrast of its retinal image. This vision loss is unrelated to what patients would likely call glare. Glare symptoms are generally caused by wide-angle light scatter from a peripheral glare source; this produces a veiling luminance on the retina and further reduces the contrast of the retinal image. Disability glare also has a drastic effect on chromatic sensitivity. The veiling luminance produced by scattered light from a glare source has the effect of desaturating colors so that they appear to be washed out.¹⁷⁸

Halos and Sunbursts

Halos and star bursts accompany "glare" as relatively common symptoms after refractive surgery,^{157,179,180} and they can cause serious problems for night driving.¹⁸¹ Wound healing after excimer laser treatment leads to corneal edema, disturbing the well-organized pattern of collagen fibrils that ensure transparency and lead to increased light scatter and diffraction halos. Similar diffraction halos have been reported for many years in patients with corneal edema caused by contact lenses and acute glaucoma.¹⁵⁶ They are also similar to lenticular halos, which are the faint-colored halos that can be seen around streetlights and candles in dark surroundings and which are caused by lenticular diffraction.¹⁵⁸ These halos show the typical range of spectral colors of a (first-order) diffraction image, with blue on the inner side and red on the outside. Lenticular halos increase with age and probably also with cataract formation.¹⁵⁸

Another type of (noncolored) halo has also been reported after refractive surgery, when optic zones are small as compared with mesopic pupil size.¹⁸⁰ These are simply myopic blur circles, and patients with these symptoms have been treated with either negative lens overcorrection or miotics at night.¹⁸⁰ Starbursts are numerous thin lines of light radiating from the glare source, and these increase after refractive surgery¹⁵⁷ and cataract.¹⁸² They are also called ciliary corona, although their exact etiology is uncertain.^{158,182}

Can Traditional Visual-Acuity Measurements Assess Glare?

Normal Snellen VA measurements are performed in office lighting, which can be much lower than daylight conditions, let alone glare situations. International standard luminance levels for measuring VA are between 80 and 320 cd/m². With uncleaned and old charts or bulbs, the luminance can easily fall below this lower limit. In comparison, the luminance of the Commission Internationale de l'Eclairage standard overcast sky with sunlight obscured by a cloud, for example, is 2050 cd/m². The increase in luminance from office to daylight does not particularly affect the vision of most patients, but it can cause a substantial loss of vision to patients who are susceptible to glare, such as those discussed in later sections. When working or driving in sunny conditions or driving against oncoming headlights at night, such patients can suffer from glare that is potentially dangerous, yet they can have normal or near-normal VA in the practitioner's office. Cataract patients, for example, can lose over five lines of Snellen acuity when it is measured outdoors in daylight as compared with when it is measured in the office.^{183,184} So, the answer to the title question is definitely negative.

Measuring Disability Glare

Research Methods

Wide-angle forward light scatter can be measured directly using the van den Berg Straylightmeter^{148,167} (Figure 8-15). This device has several advantages over alternative techniques:

1. It provides a direct measure of forward light scatter.
2. It provides measures of light scatter at different glare angles.
3. Results are claimed to be free from neuronal interference.
4. Scores are repeatable and sensitive. For example, the test has been able to show differences in forward light scatter among normal subjects with different eye pigmentation.¹⁶⁸
5. The amount of contrast loss caused by the light scatter can be calculated.
6. The results are more sensitive to subtle changes in posterior capsular opacification and corneal edema

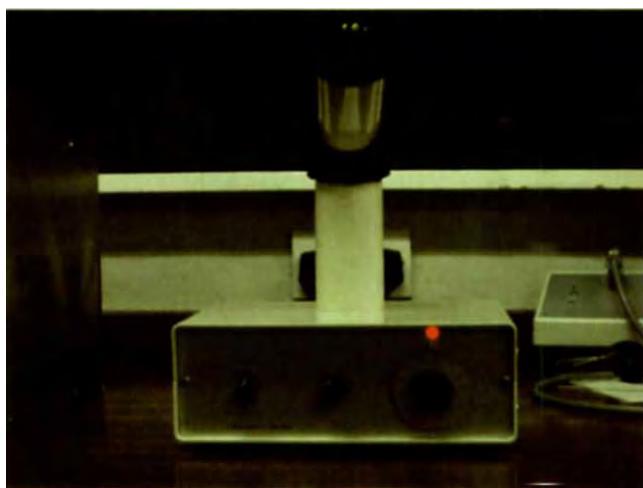


Figure 8-15

The portable version of the van den Berg Straylightmeter.

as compared with VA and other glare tests.^{138,185}

For example, the straylightmeter could detect a 1% decline in posterior capsular opacification,¹⁸⁵ and it was much more sensitive than contrast sensitivity (~45%) and VA (78%).

A clinical version of van den Berg's laboratory apparatus allows the measurement of forward light scatter at 3.5, 10, and 28 degrees¹⁸⁶ (see Figure 8-15). The 28-degree Straylightmeter score is slightly less affected by early media opacity, and it is less discriminative and reliable than the other straylight scores.⁶⁴ Because the 3.5- and 10-degree light scatter scores are highly correlated⁶⁴ and because initial results suggest that all types of cataract scatter light in a similar angular fashion,¹⁴⁸ cataracts can be assessed using either of these measurements (or the mean of both to improve reliability). The test, however, poses a somewhat difficult task for some subjects,¹⁸⁷ and a new, more "patient-friendly" straylightmeter is being developed.¹⁸⁸

Light scatter has also been assessed indirectly by measuring CS under glare and nonglare conditions. From the veiling luminance studies of Cobb and others,¹⁴⁷ the light-scattering factor (LSF) of the eye can be defined as $LSF = Leq/E$, where Leq is the equivalent veiling luminance and E is the illuminance of the glare source at the eye. The veiling luminance reduces stimulus contrast by a factor of $L/(L + Leq)$, where L represents mean stimulus luminance. Therefore, the ratio of contrast thresholds measured with and without the presence of a glare source (c_2 and c_1 , respectively) is given by the following equation:

$$c_2/c_1 = (L + Leq)/L = 1 + Leq/L, \text{ and}$$

$$Leq = [(c_2/c_1) - 1]L$$

Substituting into the LSF equation above, the following is obtained:

$$\text{LSF} = (L/E) \times (c_2/c_1 - 1)$$

This method and calculation was devised by Paulsson and Sjöstrand.¹⁸⁹ The equation allows an intrinsic LSF to be determined for any given glare angle. In addition, the LSF calculated in this way should remain independent of the precise stimulus conditions used for its determination, because variations in L and E should be counteracted by corresponding variations in contrast thresholds (however, see Ideal Design Features for Clinical Glare Tests).

How Is Disability Glare Measured Clinically?

Disability glare tends to be determined by remeasuring CS or VA after placing a peripheral glare source within the patient's field of view. Disability glare is determined as the reduction in log CS (or logMAR VA) caused by the glare source (i.e., log CS – log CS glare). This is therefore a simplification of the Paulsson and Sjöstrand¹⁸⁹ equation.

Some glare tests are scored using just the level of CS or VA under glare conditions and not the reduction caused by the glare source; they do not measure disability glare in its true sense, because they reflect any initial reduction in vision plus the further effect of the glare source. True disability glare scores should only reflect the effect of a glare source. Unless there is a substantial drop in vision as a result of a glare source, patients are likely to complain of poor vision rather than glare. When measuring visual function in patients with media opacity and an abnormal retinal/neural system (e.g., ARM), disability glare *must* be measured as the differences in CS or VA caused by the glare source. Imagine a patient with cataract and ARM whose Pelli-Robson CS is 1.30 log units without glare and 1.20 log units with glare. The 1.20 log CS level under glare conditions suggests a substantial loss in vision, but the small drop in CS caused by the glare source indicates the relatively small effect of the cataract and suggests that ARM is the major cause of vision loss. Such a patient is unlikely to complain about glare problems. By measuring the reduction in CS caused by a glare source, the scores are relatively independent of the neural system and more closely indicative of the level of intraocular light scatter.

Measuring the level of CS or VA under glare conditions can be a reasonable assessment of disability glare in a patient with media opacity and normal neural function. For example, in patients with cataract and normal neural function, measurements of CS or VA under glare conditions has been shown to be highly correlated with straylight.⁶⁴ This may be because the initial reduction in CS is also caused by straylight.

Ideal Design Features for Clinical Glare Tests

Glare tests that use VA and CS measurements should use the chart design recommendations suggested by the

American Academy of Ophthalmology⁶³ (see Ideal Contrast-Sensitivity Test Design Features). The reliability of disability glare scores is not surprisingly similar to the reliability of whatever CS or VA tests are used as part of the glare test.⁶⁴ Therefore, only CS and VA tests that use good psychophysical test-design features (i.e., small step sizes, a larger number of decisions at each level, and a large number of forced-choice alternatives) should be used. If the underlying CS test does not use good test-design principles, even ideal geometry and standardization of the glare source will not help.

One very important design principle that is often overlooked with disability glare tests is that the CS or VA test must have relatively high chart luminance.^{152,190} This is necessary to avoid increasing CS by increasing retinal illuminance. The veiling luminance model only holds within the region of Weber's law, whereas contrast thresholds are independent of luminance. At luminances that are below the region of Weber's law, veiling glare increases retinal illuminance and subsequently CS; this is very likely the reason that some studies^{150,191} have found negative disability glare scores (i.e., CS with glare scores that are better than CS without glare scores). For this reason, if a high chart luminance is not possible, the target should be of low spatial frequency content,^{189,190} because such stimuli become dependent on retinal illuminance at lower levels than do higher frequencies¹⁹² and VA. In addition, the target surround should have a luminance similar to that of the target, because CS is reduced by borders and dark edges.¹⁹³ It has been suggested that, if the without-glare CS is measured with a target surrounded by a dark border, the subsequent addition of a veiling luminance on the retina caused by a glare source could improve CS by brightening the border of the target.¹⁴⁸

The illuminance of the glare source should be bright enough to cause a decrease in CS or VA in patients with small amounts of intraocular light scatter, such as in younger patients after refractive surgery. For this reason, a glare source with a small glare angle may be required; otherwise, an extremely bright glare source is needed. In patients with a large amount of intraocular light scatter (e.g., patients with cataract), a less-bright glare source may be needed to ensure that the patient can see some of the letters on the chart. The geometry and position of the glare source should attempt to ensure that all targets are at the same distance from the glare source. For example, in a glare test that uses targets (letters or grating plates) in rows with peripherally placed glare sources, the central letters or plates will have a much bigger glare angle, will receive less veiling glare as a result of light scatter, and will be easier to see. Finally, central glare sources can reduce reliability, because they are very position sensitive, and they may cause light-adaptation glare as well as disability glare.^{63,64}

Should High-Contrast Visual-Acuity or Contrast-Sensitivity Targets Be Used with Glare Tests?

Low-contrast charts (either CS or low-contrast VA) used in glare tests provide a more sensitive measure of disability glare than do glare tests using high-contrast VA charts.^{64,194,195}

This is probably because typical Snellen VA luminance levels tend to be below the Weber region so that a glare source increases VA by increasing retinal illumination, and this could offset any loss in VA caused by veiling glare.¹⁵² An alternative explanation is that the steepness of the CSF curve as it cuts the spatial-frequency axis results in a reduction in contrast that is associated with a relatively small reduction in high-contrast VA.¹⁴⁵ However, measuring traditional high-contrast VA with a glare source has the advantage that the score is universally understood. This is well illustrated by the scoring systems of both the Miller–Nadler and the Vistech tests; they provide charts that convert the disability glare scores measured using CS into equivalent outdoor Snellen VA values. Given the insensitivity of high-contrast VA to veiling glare, glare tests using such measurements should only be used when the amount of straylight is relatively large (e.g., in patients with cataract), and a particularly sensitive test may not be necessary. When increased straylight is subtle (e.g., in patients with early corneal edema and after refractive surgery), it would appear futile to measure glare with high-contrast VA charts.

Evaluating Disability Glare Tests

Important qualities of a glare test's usefulness are its validity, its discriminative ability, and its reliability, with reliability being the most important of the three, as described earlier. The validity of glare tests has been determined by how well they correlate with outdoor VA,^{183,184} glare symptoms,¹⁹⁶ perceived visual disability,⁶⁸ and straylight.^{64,148}

Clinical Glare Tests

Early Glare Tests

In 1852, it appears that Helmholtz was the first to mention the effect of glare on vision, and Depène's work in 1890 is probably the first documented result of this effect. Depène showed that a glare source (a candle!) had lessening effects on acuity when the angle between the glare source and the acuity chart was increased. Holladay's work in 1926 represents the first classic study of glare. Before 1983, when the Miller–Nadler Glare Tester was commercially released, clinical methods of measuring disability glare were limited. They involved measuring VA under glare conditions, such as when the chart was placed in front of a window against the incoming light or while directing a penlight into the patient's eye. These tests are simple, quick, and inexpensive, and they con-

tinued to be used.¹⁹⁷ David Miller and Ernst Wolf produced the first commercially available glare tester during the early 1970s.¹⁹⁸ When only three were sold, test production was discontinued.¹⁹⁹ Subsequently, Princeton Nadler's enthusiasm for glare testing led to the production of a simpler, less-expensive version of the test, the Miller–Nadler Glare Tester, which was much more popular. It consists of a modified slide projector; the slides present a series of randomly orientated Landolt rings of progressively reduced contrast (80% to 2.5%). They are surrounded by a broad glare source of constant luminance. The working distance is 40 cm. The endpoint of the test is recorded as the last correctly identified slide. The Miller–Nadler Glare Tester has reasonable test-retest reliability, but it is not sensitive to subtle changes in disability glare because of its large step sizes (in log CS terms) at low-contrast levels⁶⁴ (see Ideal Contrast-Sensitivity Test Design Features). The lowest contrast threshold levels are 2.5% and 5% contrast (1.60 and 1.30 log CS), which is a huge perceptual difference. This is probably why the Miller–Nadler tester was unable to detect any increase in disability glare in the prospective evaluation of radial keratotomy (PERK) study.²⁰⁰

Convenient Glare Sources and Standard VA

Although only 12% of ophthalmologists in a UK survey indicated that they used glare tests when assessing visual function in patients with cataract; those that did used convenient glare sources, such as ophthalmoscopes or penlights with a Snellen chart.²⁰¹ The obvious advantage of these tests is that they are simple, quick, and inexpensive, although they are only of any value when a patient suffers from a lot of light scatter, such as is seen in those with posterior subcapsular cataract or moderate cortical and nuclear cataract. Care must be taken to ensure that the glare source is at the correct position in relation to the eye. The square power laws relating disability glare with the glare angle and distance of the glare source from the eye mean that the positioning of the glare source can be important.

Brightness Acuity Tester (BAT)

A survey of US eye-care practitioners¹⁹⁶ indicated that the most commonly used disability glare tests at the time were the now obsolete Miller–Nadler Glare Tester and the Brightness Acuity Tester (BAT). The BAT is a handheld instrument that consists of a white, ice-cream-scoop-shaped hemispherical bowl situated on top of a 16-cm handle (Figure 8-16).²⁰² The hemisphere is held over the eye and diffusely illuminated by a hidden light source, which illuminates the entire peripheral field. The glare source subtends a visual angle of 8 to 70 degrees at a vertex distance of 12 mm. The patient is asked to read a chart through a small aperture in the bowl. It can be used with CS and low-contrast acuity charts and with traditional high-contrast VA charts. The

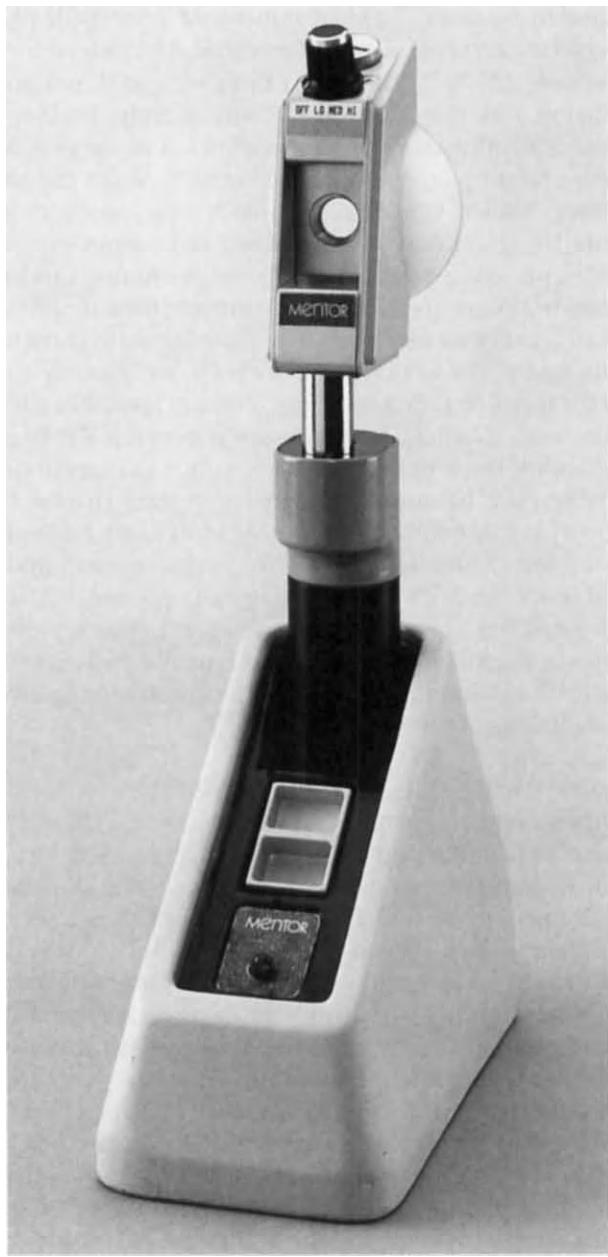


Figure 8-16

The Brightness Acuity Tester (BAT). (*Courtesy of Mentor O & O.*)

low-intensity setting is not recommended, because it has little effect. The BAT has been shown to provide reliable and discriminative measurements of disability glare when used with logMAR VA charts and the Pelli-Robson CS chart⁶⁴; it has also been shown to be a good predictor of outdoor VA.¹⁸³ The medium-intensity setting is preferred when measuring disability glare in patients with cataract, because the high-intensity setting has been reported to give poor predictions of VA measured outdoors,²⁰³ and it reduces contrast beyond a chart's limits for some patients with early cataract.⁹⁷ The



Figure 8-17

The Optec 3000. (*Courtesy of Stereo Optical Company.*)

high-intensity setting may be of value when measuring subtle amounts of disability glare, such as in patients with contact lenses and after refractive surgery. As discussed earlier, high-contrast VA charts should only be used with the BAT when the amount of straylight is relatively large (e.g., in patients with moderate to dense cataract); otherwise, low-contrast VA charts or CS charts should be employed. Of the Regan low-contrast charts, the 25% contrast chart seems to be the most appropriate for assessing disability glare in patients with early cataract.^{64,97} The BAT is commercially available from www.marco.com/classic/acuity_tester.html.

Grating CS-Based Tests

Several glare tests are available that measure grating CS under glare conditions. The CSV-1000 Halogen Glare Test includes the CSV-1000 CS test plus two peripheral glare tests, and it is available from www.vectorvision.com. The Optec system (Figure 8-17) is a range of automated tabletop instruments that provides CS and disability glare testing in addition to a range of other tests. The disability glare test uses the same targets and measurement method as the FACT CS test and the Holliday Contrast Acuity Test (see Currently Available Contrast-Sensitivity and Low-Contrast Visual-Acuity Tests), and it uses a radial glare source. A console controls target presentation and glare-source calibration. The instrument is available from www.contrastsensitivity.net.

It is likely that these grating CS-based tests will be limited by the poor reliability of the underlying CS test. For example, the Vistech MCT8000, which was an automated tabletop instrument incorporating a Vistech CS chart with radial glare sources, was found to provide unreliable results that were similar to those that occurred with the underlying Vistech CS test⁶⁴ (see Evaluating Clinical Contrast-Sensitivity Tests); it also had poor discriminative ability and correlated poorly with straylight.^{64,148}

Mesotest and Nyktotest

These tests are used in Europe to measure mesopic CS under glare conditions, and patients have to adapt to darkness for 10 minutes before measurement begins. The tests have been designed to detect visual losses that would cause night driving problems, and they have been shown to correlate with perceived driving disability.²⁰⁴ There is little literature about their usefulness, although they appear to be purely screening tests, and they attempt to place patients into a normal or abnormal category. The Mesotest has only four steps, and the Nyktotest has eight; they reputedly provide large ceiling and floor effects.

Recommended Glare Tests

For a gross estimate of disability glare in patients with cataract (particularly posterior subcapsular cataract), a remeasurement of traditional VA with a bright glare light is probably sufficient. For detecting and monitoring more subtle light scatter, such as after refractive surgery, a CS or low-contrast VA chart should be used. Letter CS tests rather than gratings are preferred because of their better reliability. The CS or VA chart must be at a reasonable luminance (preferably above the Pelli-Robson chart's recommended level of 85 cd/m²), and the glare source should be bright (use the brightest setting on the BAT). The glare source should be at a fixed angle and distance from the eye. Given the excellent reliability and sensitivity of the research-oriented van den Berg straylightmeter, it is hoped that the clinical version that is being developed is easy for patients to use.¹⁸⁸

Measurement Procedure for a Glare Test

Monocular disability glare is tested with one eye viewing the chart while an occluder is placed before the other eye. If the hand of the patient or the clinician is being used to occlude the other eye, care should be taken to use the palm, because otherwise the patient might look through a gap between the fingers. It is traditional to measure the right eye first, but the left eye might occasionally be measured first if it is known that the patient has more glare problems in that eye. Measurements should be made with the patient's own distance-vision spectacles or contact lenses, because the reduced aperture of phoropters or some trial case lenses

can obstruct the glare light getting into the eye. Check whether any spectacles are badly scratched, because this may increase disability glare scores. The test should generally be performed without dilating the pupils so that the normal pupillary constriction from bright light occurs. Most glare tests involve remeasuring CS or VA under glare conditions, so the only difference in the measurement procedure from that explained elsewhere is that care must be taken to ensure that the glare source is at the correct position in relationship to the eye. The square power laws relating disability glare with the glare angle and distance of the glare source from the eye mean that the positioning of the glare source can be critical. With the BAT, the patient should attempt to position his or her eye so that it is in the middle of the BAT aperture. The most common errors include incorrect positioning of the glare source, using the BAT when it is not fully charged up and providing too low a glare source, using a target luminance that is too low and below the Weber region, and using the glare test with a phoropter or reduced-aperture lenses in a trial frame.

Interpretation of Test Results

Influence of Refraction on Glare Tests

Glare-induced pupillary miosis could provide a pinhole effect in patients who are not optimally corrected, and it could improve VA or CS caused by uncorrected refractive error in the glare condition. This could produce negative disability glare scores among young patients.

Different Glare-Test Values from Different Tests

Theoretically, disability glare and straylight measurements can be compared using the Paulsson and Sjöstrand equation¹⁸⁹: LSF = (L/E) × (M₂/M₁ - 1). Obviously, this is only true for glare tests that measure the drop in CS caused by a glare source. Average straylight values in patients with cataract have been found to be around 40, with maximums of about 80.^{64,148} Straylight predictions based on contrast thresholds with and without glare also vary somewhat. Paulsson and Sjöstrand¹⁸⁹ used only posterior subcapsular cataracts, some of which demonstrated enormous straylight values despite relatively good acuities. de Waard and colleagues¹⁴⁸ found rather poor agreement between straylightmeter values and CS loss (measured with a Vistech chart) under glare conditions. For patients with early cataracts, contrast loss produced by a glare source significantly underpredicted straylight, whereas, for patients with more advanced cataracts, contrast loss far exceeded that predicted on the basis of straylight measurement. Among a very small sample, similar contrast loss and straylight among patients with early cataract and much greater contrast loss as compared with straylight in patients with more advanced cataract was found.¹⁷²

Further research is required to determine why these differences occur.

Differences in terminology can be found regarding disability glare scores measured using low-contrast VA, which can be confusing. It is not valid to calculate disability glare scores measured using VA with the Paulsson and Sjöstrand equation, because this uses contrast thresholds and not acuity. Regan and colleagues use the term "glare susceptibility ratio," and they calculate the ratio between decimal VA with and without the glare source.^{35,73,97} Bailey and Bullimore¹⁹⁴ use "the disability glare index," which is calculated as the number of letters lost as a result of the glare source.

Changes with Age and Ocular Pigmentation

In 1960, Wolf first showed significant increases in disability glare with age, and these findings have subsequently been confirmed.^{35,64,73,194} Forward light scatter has also been shown to increase several times over with age in healthy eyes¹⁶⁷; this is thought to be caused primarily by changes in the lens,^{149,167} with negligible changes in the cornea.¹⁷⁰ Ocular pigmentation has also been shown to significantly affect forward light scatter, with blue-eyed Caucasians having significantly higher straylight than brown-eyed Caucasians and especially non-Caucasians, particularly at wide angles of about 25 degrees.¹⁶⁸ This wide-angle light scatter is probably caused by light transmission through the iris and reflectance from the fundus. Backscatter also increases with age, especially from the lens. The lenticular cortex shows a small, steady increase in backscatter with age, and this is thought to be the growth of this region and the specular reflections from the ever-increasing zones of discontinuity.¹⁶⁶

Collecting Personal Normative Data

It is advisable for clinicians to collect their own disability glare test norms.

Clinical Uses

Patients may use the word "glare" (or "blinding lights," "dazzling," and so on) to describe a whole host of situations. Thus, when a patient complains about glare problems, further questions are required to determine to what exactly they are referring. Many glare situations are normal and simply require confirmation of this fact (e.g., the glare when reading a book in full direct sunlight or when leaving a darkened cinema on a sunny day). In addition, clinicians need to differentiate between disability glare and light-adaptation glare, because the latter needs to be assessed using photostress recovery time (see Light-Adaptation Glare) rather than a disability-glare test.

The most common reason for using a glare test is for preoperative and postoperative evaluation of patients

undergoing cataract surgery and Nd:YAG capsulotomy. In a national survey of practitioners, 23% stated that they frequently or always used glare testing during pre-operative evaluations for cataract surgery.²⁰⁵ The usefulness of disability glare testing has been listed in order of the amount of agreement in the literature: the sections listed first are those with most agreement, whereas those listed later appear to have the least agreement. The clinical usefulness of assessing light-adaptation glare is discussed in its own section at the end of the chapter.

Determining When to Refer for Cataract Surgery and Nd:YAG Capsulotomy

Guidelines by the Agency for Health Care Policy and Research (AHCPR)¹²⁸ have suggested the following indications for cataract surgery:

1. The patient's ability to function in his or her desired lifestyle is reduced because of poor vision.
2. VA is 20/50 (6/15) or worse and is solely caused by cataract.
3. The patient decides that the expected improvement in function outweighs the potential risk, cost, and inconvenience of surgery after being given appropriate information.

However, anecdotal evidence and case reports have suggested that using a VA of 20/50 (6/15) as a guideline for cataract extraction can be inadequate. Many clinicians have reported seeing patients who retain better VA than 20/50 (6/15) yet who report significant visual problems. The AHCPR report recognizes that such patients exist, and guidelines are provided for patients with 20/40 (6/12) or better VA. However, the report merely suggests a more careful documentation of the patient's symptoms, although the report does suggest the use of glare tests for patients who complain of glare yet who have reasonable VA. Research is still required to determine whether glare tests provide any significant information about functional vision beyond acuity,¹²⁸ but several case reports strongly suggest that these tests do for some patients. An example is the case reported by Rubin in 1972. A healthy 45-year-old prison guard complained of a gradual decrease of vision over the previous year. The vision loss particularly occurred in bright sunlight, such as when guarding prisoners working outside. The loss of vision had recently become so great as to allow two convicts to escape! His VAs were measured to be 20/20 (6/6) in both eyes. However, careful examination found small posterior subcapsular cataracts and VAs of 20/400 (6/120) in bright light levels. Posterior subcapsular cataracts give much greater levels of disability glare than other morphological cataract types^{148,189,206}; this is probably because of the dramatic effect on vision with these centrally positioned cataracts caused by pupillary constriction in bright light levels²⁰⁶ and/or a greater amount of forward light scatter as compared with backward scatter as seen at the slit-

lamp. The latter is supported by the finding that patients with choroideremia but without clinically visible PSC cataracts still have increased forward light scatter.²⁰⁷ Surprisingly, the angular distribution of forward light scatter has been found to be similar for the different morphological types of cataract.¹⁴⁸ There are no definitive levels of glare scores at which a patient should be referred. Referral should be based primarily on patient symptoms and the presence of cataract or posterior capsular remnants,¹²⁸ with high glare scores providing justification for referral, particularly in a patient with good or reasonable acuity.

After uncomplicated surgery, disability glare in pseudophakic subjects compares well with that of healthy, age-matched, phakic patients with normal vision.²⁰⁸ Capsular opacification in pseudophakic patients commonly causes increased disability glare,^{185,191} and this can be resolved by Nd:YAG capsulotomy.^{191,209} As with central opacities, capsular opacification can cause significant disability glare while leaving Snellen VA relatively normal, and glare tests can be used to justify referral in symptomatic patients.

Patients with Symptoms of Glare

Disability glare scores in patients with glare symptoms can be used to indicate whether such symptoms are caused by significant disability glare (as opposed to, for example, discomfort glare or light-adaptation glare) and to quantify the degree of any problem for subsequent monitoring. Glare symptoms and increased disability glare have been found in a wide range of patients, including those with cataract and posterior capsular opacification (see Determining When to Refer for Cataract Surgery and Nd:YAG Capsulotomy), those with corneal edema and contact lens wear (see the next section), those with hereditary corneal dystrophies,²¹⁰ those with diaphony, diabetic patients after panretinal photocoagulation,²¹¹ and those with nephropathic cystinosis.²¹²

Clinical Trials

Disability glare tests have been used in the clinical trials of anticataract formulations²³ and refractive surgery techniques.²⁰⁰

Monitoring the Effects of Refractive Surgery

The success of refractive surgery has been somewhat limited by the "corneal haze" (a description of backward light scatter as seen at the slit-lamp) and associated symptoms of glare after the procedure. Early studies did not adequately investigate the functional consequences of corneal haze, mainly because of the difficulty of assessing the loss of corneal transparency. As indicated by Lohmann and colleagues,¹⁶⁵ studies primarily focused on assessing backscatter and relating this to VA. As discussed earlier, backscatter is not a good indicator of vision loss, because it does not necessarily indicate

the forward light scatter that causes loss of vision (see Backward vs. Forward Light Scatter), and VA provides a limited assessment of disability glare (see Can Traditional Visual-Acuity Measurements Assess Glare?). In addition, the sensitivity of available glare tests to these subtle corneal changes has been questioned.^{187,200} This lack of sensitivity could be the result of a variety of causes. First, it is important that the glare test uses good test-design features. For example, the Miller-Nadler glare test used by Waring and colleagues²⁰⁰ was insensitive to subtle changes in glare as a result of its very large step sizes at low contrast levels.⁶⁴ In addition, all glare tests should use a CS or VA chart of high luminance; otherwise, their addition of a glare source will merely lead to retinal adaptation and negative disability glare scores (i.e., better CS with the glare source). Using a high chart luminance is obviously not possible for mesopic glare testers, and it may not be possible to measure the glare problems associated with night driving with any of the disability glare charts currently available. In this respect, it has been suggested that glare tests should use a transient glare source rather than a steady source of glare, because transient glare sources are more disabling and more accurately reflect the glare problems that occur during night driving.²¹³ All commercially available tests currently use steady sources of glare. Finally, the deterioration in vision at night is caused by a combination of increased optical aberrations and increased forward light scatter,¹²² so postoperative reductions in vision may be best measured using CS and/or low-contrast VA rather than disability glare measurements, which only assess the effects of light scatter. The US Department of Treasury and the US Customs Service currently assess all applicants to federal agencies (e.g., the FBI) who have had refractive surgery using the ETDRS VA chart, the Pelli-Robson CS test, and BAT under dilated and undilated conditions.⁸⁰

Patients with Contact-Lens-Induced Corneal Edema

Several vision tests have been proposed to provide useful quantitative assessments of edema, including disability glare.²¹³ Epithelial edema is characterized by large increases in forward light scatter,²¹⁴ and it is considered to be more visually disabling than stromal edema.²¹⁴ Epithelial edema is common during the early stages of rigid contact lens wear. The increased lacrimation caused by the lens discomfort produces an osmotic imbalance between the tear film and the epithelium, thereby leading to the edema.²¹⁵ As adaptation to the contact lens occurs, this potential epithelial osmotic stress is reduced. Edematous changes in adapted lens wearers are more likely to be found in the corneal stroma and to be hypoxic in origin.²¹⁵ However, the data from various studies have found little increase in the stromal scattering of light until stromal thickness

increases significantly.²¹³ It has been suggested that most increases in light scatter as a result of hypoxia are from small epithelial changes.²¹⁶ Disability glare tests have been found to be much more sensitive to osmotically induced epithelial edema (1% to 2% edema) than to hypoxia-induced stromal edema (about 8%).²¹³

Can Disability Glare Tests Help When Prescribing Tints?

Glare symptoms can often lead to the prescribing of tints and filters. Filters act by varying the amount and spectral distribution of transmitted light, and they are often prescribed by clinicians under the assumption that they provide some improvement in visual performance, especially in the presence of glare. The choice of filter type is usually somewhat arbitrary, which is perhaps not surprising, because the topic of visual performance and tinted lenses is complex and often controversial. The use of a filter does not directly reduce disability glare, although it will reduce the veiling luminance caused by a glare source; it also reduces the target luminance by the same proportion, and disability glare would be expected to remain unchanged.²¹⁷ Filters may, of course, help alleviate discomfort glare. The optimal way to alleviate disability glare is to reduce straylight reaching the eye from a glare source without affecting the object of interest. This is the logic behind the use of horizontally louvered spectacles, pinhole glasses, or honeycombs, but their limited field of view restricts their clinical application. A similar effect is achieved by the use of visors, broad-brimmed hats, and squinting in bright sunlight, although these tactics are often considered purely as methods to combat discomfort glare. Graduated tints, which selectively block glare from above, work along the same principles, but they are usually prescribed on a cosmetic rather than a functional basis. Another example of selective attenuation of glare is in the use of polarizing filters that preferentially absorb light that has been polarized by surface reflection. Given the lack of any significant Rayleigh light scattering in the eye, there seems to be little rationale for prescribing blue-absorbing tints. This is particularly true for patients with nuclear cataract, because they already have a built-in blue-absorbing filter. Several studies have shown that yellow and luminance-matched neutral lenses have similar effects on contrast thresholds.^{218,219}

Clark surveyed nearly 100 studies of tinted lenses and concluded that there was no advantage of any colored lens relative to neutral tints. Despite this, several studies report increased contrast performance and increased VA in the presence of filters.²¹⁷ One reason for these observations may lie in the efficacy of certain tints to absorb UV light and to reduce disability glare caused by fluorescence.¹⁷⁵ Filters that demonstrate negligible transmission at the activation wavelengths of lens fluorophores may result in increased visual performance in environments that contain relatively high levels of

UV light. Such filters may particularly help reduce disability glare in patients with nuclear cataract and in diabetics, because they contain significant lenticular autofluorescence.^{176,177}

Disability glare scores may help to determine whether to prescribe a tint to patients with centrally placed subcapsular opacities. In these patients, a tint may help alleviate disability glare by reducing pupil constriction.

Light-Adaptation Glare

Light-adaptation glare has a neural or, to be more precise, retinal etiology. A central scotoma is perceived when a bright light bleaches the foveal cone photopigments, thereby causing a temporary state of retinal insensitivity. The time required to regain spatial resolution depends on the photochemical capability of the macula. Brindley²²⁰ has shown that long-lasting afterimages (longer than 15 seconds) produced by brief light flashes (less than about 5 seconds) are caused by photochemical changes in the receptors. He suggested that other neural effects contribute to the first 15 seconds of an afterimage. The influence of optical factors (e.g., pupil size, lenticular absorption) is minimal and due only to their influence on the amount of light reaching the retina. The basic dynamics of cone-pigment regeneration are similar to those of rhodopsin, except bleached cone-pigment molecules return to their regenerated state more quickly. The light sensitivity of the visual pigments is caused by a chromophore, vitamin A aldehyde (retinal), which is bound to the visual pigment protein (opsin). Light absorption by rhodopsin leads to the separation of the retinal chromophore from opsin. This process is called *bleaching*, because it results in the loss of rhodopsin's purple color.

There are several possible causes of prolonged light adaptation or recovery time.²²¹ The retinal pigment epithelium ingests and destroys membranes shed by receptor cells as well as storing and transporting vitamin A. Therefore, any interference between the retinal-pigment-epithelium-receptor complex (e.g., ARM, angiofibrous streaks, choroideremia, serous retinal detachment, pigment epithelium retinopathy) disturbs these processes and slows the regeneration of photopigments.^{221,222} Also, the receptors' high metabolic activity depends on the integrity of the underlying choriocapillaris. Disruption of this metabolic activity, such as in patient with hypoxia²²³ or in patients with impaired retinal vascular supply (e.g., diabetics, hypertensives), can lead to longer recovery times.²²²

Light-adaptation glare is most commonly measured clinically as a photostress recovery time (PSRT). This is measured by timing a patient's recovery to within one line of preadaptation VA after a 10-second exposure to a bright light source. Abnormal values tend to be suggested as being longer than 50 seconds. Textbooks

suggest slight differences in technique, and standardization would provide a more sensitive test. This is best achieved by each clinician obtaining his or her own normative data. Given the variability of light output from penlights and the low PSRT scores using the BAT, a direct ophthalmoscope or transilluminator may be the best light source to use. From Brindley's²²⁰ work, the duration of exposure should not be much more than 5 seconds, but it should be sufficient to give a PSRT time that is greater than 15 seconds. It seems advisable to have the postphotostress task involve reading a line that is larger in size than the one read during prestress task, given that VA measurements are not exactly reproducible and that retest measurements can be about a line worse than test measurements.

PSRT testing is probably most useful clinically for differentiating macular from optic nerve disease. The cause of central vision loss can occasionally be difficult to diagnose, because optic nerve disorders and subtle maculopathies can give inconclusive funduscopic findings. Because optic nerve disorders such as optic neuritis and ischemic optic neuropathy (and other abnormalities such as amblyopia) do not affect the photochemical processes in the photoreceptors, recovery times remain normal.²²⁴ A long recovery time suggests a macular problem. PSRT may also aid in the monitoring of the recovery or progression of maculopathies, such as early cystoid macular edema, idiopathic central serous chorioretinopathy, and chloroquine or solar burn effects on the macula.

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9

Color Vision

Paul L. Pease

A great deal has been written about color vision and color blindness, and there is a fairly extensive understanding of normal color vision and its variations as a result of inheritance and disease. This chapter is about the testing of color vision. The goals are to provide the fundamentals for understanding normal color vision and color vision deficiencies and to provide the essentials for testing, diagnosis of the type of color defect, and management of patients. Background information about color terminology, color mixture, and systems of color notation is included to enhance the understanding of the different types of tests and how they work. Some useful resources include the book *Congenital and Acquired Color Vision Defects*, edited by Pokorny and colleagues¹; a chapter titled "Color Deficiency" by Adams and Haegerstrom-Portnoy² in *Diagnosis and Management in Vision Care*; and the biennial *Proceedings of the International Colour Vision Society* (previously known as the International Research Group on Colour Vision Deficiencies), available through <http://orlab.optom.unsw.edu.au/ICVS/>.

HISTORY

It is likely that defective color vision, or color blindness, as it is commonly called, has been known for a long time^{3–5}; however, the topic did not receive any appreciable attention until relatively recent times.^{6,7} Scientific interest began in earnest after chemist John Dalton published his description of his own color blindness in 1794. Dalton believed his color defect was the result of a blue coloration of his vitreous humor, and he requested that, upon his death (July 27, 1844), his eyes be dissected to test this hypothesis. Postmortem examination proved him incorrect. One hundred and fifty years later, tissue from his eyes was subjected to DNA analysis, and it was determined that Dalton had been a deutanope.⁸ One appreciates Dalton's prominence in the field when one considers that the French, Spanish, and Russian words for "color blindness" are derived

from "daltonism." Complete background information about John Dalton's color vision in his own words and an account of his phenotype and genotype assessed with modern tools can be found in the proceedings called *John Dalton's Colour Vision Legacy*, edited by Dickinson and colleagues.⁹

THE TWO TYPES OF COLOR DEFECT

There are two principal types of color defect: inherited and acquired. *Inherited color defects* are congenital, genetically inherited, and without other associated abnormality. These defects are not physically debilitating, but they can have a major impact on one's life. A person may not see the red cardinal perched on a branch in the green shrubbery. A child may be reprimanded by his father for not picking up the red toy truck on the front lawn. Others may not even be aware of their deficiency until a peer makes fun of their choice of color in art class, an experience that could have a negative impact on their outlook on school and learning. All of these individuals have an inherited color defect with which they must learn to cope. Many who have a color deficiency learn of it only after they fail a color vision test, and some who are informed that they are so afflicted deny it.¹⁰ Some color-defective individuals are defensive and understandably insolent when forced to deal with the consequences of their deficiency, which can deny them employment in certain occupations. It is unfortunate that many individuals learn of their color deficiency when they take a color vision test as part of a physical examination for employment and then are disqualified for the position after years of planning. Eye care practitioners should ensure that color testing is done at a young age for the purpose of providing good baseline data and to lessen the adverse impact that ignorance of the defect can create.

Acquired color defects are a different story. A change in color vision may be the prelude to serious ocular and systemic conditions, and testing may provide for an

early diagnosis. The status of color vision can be a sensitive indicator of the success of therapy, and it may facilitate a differential diagnosis; for example, a blue-yellow color defect is found in just one of the several types of hereditary optic atrophy (autosomal dominant optic atrophy). Color vision may be altered by drugs, medications, and the toxic effect of chemicals. Tracking changes in color vision allows the clinician to monitor a patient's condition.

COMMON MISUNDERSTANDINGS ABOUT COLOR BLINDNESS

People who have an inherited color defect are labeled "color blind," "color deficient," or perhaps even "color-challenged." They are different enough from the majority to invite curiosity. Color is of interest to just about every person, and everyone has some understanding—and perhaps some misunderstanding—about color blindness. Common misunderstandings include the beliefs that defective color vision is relatively rare, that color blindness only occurs in males, that color blindness is always hereditary, and that color-blind persons fail to see particular colors (e.g., the "green blind" do not see green, the "red-green blind" do not see red and green). Professionals generally use the term *color deficiency* instead of the misnomer *color blindness*. Patient counseling should include information to dispel the common misunderstandings and an explanation about the inappropriateness of the term *color blindness*, despite its common usage.

In fact, inherited color vision defects are very common, occurring in about 1 out of 12 (8%) males and 1 out of 200 (0.5%) females. Other, less-prevalent conditions with more serious outcomes are often perceived as more common. For example, the prevalence of blindness in North America is 0.2%,¹¹ and AIDS prevalence is estimated at 0.5%,¹² yet these two conditions are generally thought by the public to occur much more frequently than the figures indicate. The notion that color blindness in women is so rare that you are likely never to encounter a color-deficient woman no doubt results from the perception of the consequence of the condition; this perception is put into perspective by the question, "What if 1 out of 200 females had one eye and two noses instead of vice versa?"¹³

Although most people who have a color deficiency inherited their defect, there are other causes, including disease, trauma, the toxic effect of drugs, and aging. Common labels such as "red blind" and "green blind" wrongly convey the notion that color-blind people are not able to see a particular color. On the contrary, they see all the colors that people with normal color vision

see, but they see them differently. Most see colors as being more washed out or paler as compared with the way that those with normal color vision see them. It is unfortunate that some eye-care professionals do not test for color blindness, because they mistakenly believe that there is nothing that can be done for the color-blind person. Although there are no cures for an inherited color defect, counseling patients about their vision and how they inherited their color defect, advising about career decisions, and providing aids that may possibly help them discriminate colors better are important services for the welfare of the patient.

Because individuals with an inherited color deficiency are managed differently from those with an acquired color deficiency, it is important to differentiate the two types of deficiency. People with an inherited defect benefit from counseling and, if appropriate, the selection of a colored filter to improve color discrimination may be helpful as well. With acquired color defects, results of color testing may provide a sensitive indicator of the progression or regression of the condition, and, if the cause is known, appropriate intervention may restore normal color vision.

OCCUPATIONAL ASPECTS

Color vision testing is an integral part of the physical requirements for certain occupations, and eye-care professionals are asked to administer color tests as part of this requirement. The following excerpt helps demonstrate why this came about.

On the night of the 5th of July, 1875, there was a collision near Norfolk, Virginia, between the steam-tug Lumberman and the steam-ship Isaac Bell, the former vessel bound to, and the latter from, Norfolk. The accident occurred about nine P.M. on an ordinary clear night, under circumstances which until recently seemed more or less mysterious. The master of the steamer and all his officers made oath that at the time signals were made to the tug the latter was from 1 to 2 points on the steamer's starboard bow, and consequently the steamer's green light only was visible to the approaching tug. Yet the master of the tug, whose statement was unsupported by any other testimony, asserted that the steamer's red light was exhibited, and signaled accordingly. The discrepancy in the statement was so great that many persons uncharitably charged the master of the tug with being intoxicated, although no evidence was offered in support of the charge. By this accident ten persons lost their lives. Upon a visual examination of this officer under the rules during the past summer, and during which time there had been no questions as to sight by the Sergeant of the Marine Hospital at Norfolk, he was found to be color-blind, two examinations having been accorded him, with an interval of ten days between them.

The foregoing is the evidence given by T. H. Bickerton to Lord Rayleigh, chairman of the Committee on Colour Vision, and it was made part of the published report.¹⁴

Later that same year, on November 15, 1875, two Swedish express trains ran into each other, causing many injuries and nine deaths. The facts that came out of this accident led A. F. Holmgren, a celebrated Swedish physiologist and ophthalmologist, to develop a test for color blindness and to campaign tirelessly to encourage standards for the exclusion of color-blind persons from certain occupations in which faulty interpretation of color signals is dangerous. It is quite probable—although difficult to establish definitively—that the faulty judgment of color has been responsible for many accidents. The accident involving the Isaac Bell was conclusively established to be caused by a color-deficient individual; however, persons with normal color vision may also make errors of interpretation of color, especially when signals have to be identified under less-than-ideal viewing conditions, such as adverse weather. The transport industry has changed enormously since 1875; there are better standards for color signal lamps, and color is used redundantly with position, size, shape, and intensity to present information more effectively. For example, position clues are used for traffic lights. When the fairly standard vertical code, with red on top and green on the bottom, is changed to a lateral code with red on the left and green on the right, difficulty is created for the color-defective person who is accustomed to the vertical code. In some situations, a single flashing red or yellow is used, and hence no position clue is available. The color-defective person may have to rely more on what the other drivers are doing than on the color of the signal.

Although correctly identifying a color code is often a greater challenge for the color-defective person, some people with normal color vision may perform as poorly as those with a color defect, depending on the complexity of the code and individual ability. Moreover, in some situations, color-defective people may have an advantage. For example, individuals with a protan (red) color deficiency perceive the retinal blood vessels as much darker than do people with normal color vision and hence have the benefit that the latter can obtain only with the cyan filter in "red-free" ophthalmoscopy. Although persons with normal color vision can never fully appreciate what the person with defective color vision sees, they can gain some appreciation for the problems experienced by the protanope by viewing through a #370 "Italian blue" Roscolux filter. Interestingly, the mistaken notion that color-defective persons are better at detecting camouflage persisted for a long time after World War II. In fact, color-defective persons lack superior detection ability except in a very limited number of viewing conditions.^{15,16}

CLASSIFICATION OF COLOR VISION DEFICIENCIES

Color vision deficiencies are broadly classified into two groups based on the origin or primary cause: (1) congenital or inherited color vision defects and (2) acquired color defects. Although each of these two categories includes several subcategories that are distinguished on the basis of test results, the most important diagnosis to make when making an assessment of color vision is the distinction between an inherited and an acquired defect. As mentioned earlier, the inherited color defects are nonpathological and incurable conditions, and they do not change over time. The most common are the red-green defects, which are inherited as an X-chromosome-linked recessive trait (i.e., an alteration or absence of one of the receptor photopigments). Acquired color defects accompany another condition (e.g., disease, trauma), or they are caused by the side effects of certain drugs, medications, or exposure to chemical toxins. Acquired color vision defects may also be congenital or inherited, but the color deficiency is a result of the cause or condition. Whereas inherited color defects are the result of changes at the photopigment level, acquired color defects result from a change that could occur at any of several areas or levels of the visual system. The diagnosis of the specific type of acquired color defect can lead to clues about the site in the visual system at which the anomaly lies, and this may facilitate the differential diagnosis of the underlying disease or cause. Unlike inherited color defects, the acquired color defects ordinarily change over time, in both type and severity; thus, *any observed change in color vision status is a certain indicator of an acquired defect*. The absence of a change in color vision, however, is not always a certain indicator of an inherited defect. In some inherited conditions that produce an acquired color defect, such as the blue-yellow color defects associated with dominant inherited juvenile optic atrophy, color vision is stable, as is the condition. The major differences between inherited and acquired color defects are summarized in Table 9-1 and discussed in detail in the next two sections.

Inherited Color Vision Defects

The inherited color defects are classified on the basis of the number of primary colors that are used in a color mixture to match all colors that an individual can see. People with normal color vision, who are referred to as *normal trichromats*, are able to match all colors with mixtures of three primary colors, such as an additive mixture of red, green, and blue. People with an inherited color defect are classified as *anomalous trichromats*, *dichromats*, and *monochromats*. The *anomalous trichro-*

TABLE 9-1 Summary of the Major Differences Between Inherited and Acquired Color Vision Defects

Inherited	Acquired
The defect is the same in each eye with regard to both type and severity.	The severity of the defect may be greater in one eye than in the other, or one eye could be normal and the other not.
The defect is constant throughout life.	The defect changes with the progression or regression of the primary cause.
Test results are relatively stable with changes in testing conditions.	Test results are often strongly influenced with changes in test conditions, such as viewing time and light level.
The defect is almost always a red-green defect.	The defect is frequently a blue-yellow defect.
Colors of familiar objects are correctly named.	Changes occur in the color appearance of familiar objects.
Results of color tests are reliable, and it is easy to categorize the type of defect with common tests.	There are differences in test results from one test to another, and there are problems with categorization of the defect.
Often there are no other signs or symptoms.	The defect is always associated with disease (systemic or ocular), toxicity, or trauma.
Inherited defects are more prevalent in males than females.	Acquired defects are equally prevalent in males and females.

mat uses three primaries in a mixture to match any color but requires a different intensity of each primary as compared with the normal trichromat. The *dichromat* uses only two primaries, and the *monochromat* matches any color by adjusting the intensity of any other color. Characteristics of the different types of anomalous trichromats, dichromats, and monochromats are summarized in Box 9-1.

Before 1897, dichromats were referred to as either "red blind" or "green blind." This terminology was based on the primaries used in color matching; for instance, "red-blind" persons used only two primaries: green and blue and not red. Similarly, "green-blind" persons did not require the green primary to match colors. In 1897, J. von Kries¹⁷ introduced the Greek terms *protanopia* and *deutanopia* to replace "red blind" and "green blind," respectively. These labels were coined to avoid the false implication that "red-blind" persons lacked only the sensation of red and "green-blind" persons lacked only the sensation of green. The alterations of vision are really a bit more complex. It is now known that dichromacy occurs because there are only two retinal photopigments. The protanope lacks the L-cone photopigment; the deutanope lacks the M-cone photopigment; and a third type, the *tritanope*, is missing the S-cone photopigment. The letters L, M, and S designate cones that best respond to long (red), medium (green), and short (blue) wavelengths, respectively, in persons who have normal color vision. A fourth type of dichromacy, *tetartanopia*, is probably only a hypothetical defect. Even so, there are color plates in the American Optical-Hardy, Rand, Rittler (AO-HRR) that test for tetartanopia, and the score form for the Roth 28-Hue Test shows a tetartan confusion line.

In early color-matching experiments with subjects believed to have normal color vision, John William Strutt, who later became the third Lord Rayleigh, observed wide variations in the amounts of monochromatic red and green light subjects mixed together to match a monochromatic yellow.^{18,19} The mixture of red and green to match a standard yellow is now referred to as the *Rayleigh equation*. Color mixture values falling outside of the ranges for normal color vision in the Rayleigh equation can be used to distinguish normal trichromats from *anomalous trichromats*. Using terminology introduced by Nagel,²⁰ anomalous trichromats are classified as *protanomalous*, *deuteranomalous*, and *tritanomalous*, and these are commonly referred to as "red-weak," "green-weak," and "blue-weak," respectively. On the basis of the matching ranges for the Rayleigh equation, anomalous trichromats can be subdivided into the *extreme protanomalous* and *extreme deuteranomalous*.²¹ These individuals have very large matching ranges that include the mean value for normal color vision, whereas simple protanomalous and simple deuteranomalous individuals have small matching ranges and values that are clearly different than the mean for normal color vision.

In 1947, Farnsworth²² introduced the contractions *protan* for protanomalous and protanopic individuals, *deutan* for deuteranomalous and deuteranopic persons, and *tritan* for tritanomalous and tritanopic persons (Table 9-2). These terms are necessary, because the performance of some anomalous trichromats cannot be differentiated from that of dichromats on certain tests of color vision, including the Farnsworth-Munsell 100-Hue Test and the Farnsworth Dichotomous Test (Panel D-15). At times, protan and deutan defects are referred to in combination as *red-green defects*, and the tritan

Box 9-1 Classification of Color Vision Status Based on the Minimum Number of Primary Colors Used to Match Perceived Colors

- I. **Trichromatism:** Three primary colors, in appropriate proportions, will match any perceived color.
- Normal Trichromasy:** Maximum photopic luminosity is at 555 nm.
 - Anomalous Trichromasy**
 - Protanomaly:** Photopic sensitivity for red wavelengths is low. Maximum luminosity is at 540 nm. As compared with normal vision, protanomaly requires more red light in a color match of red and green to match a standard yellow.
 - Deuteranomaly:** Photopic spectral sensitivity is nearly normal. As compared with normal vision, deuteranomaly requires more green light in a color match of red and green to match a standard yellow.
 - Tritanomaly:** Photopic spectral sensitivity is normal. As compared with normal vision, tritanomaly requires more blue light in a mixture of blue and green to match a standard cyan.
- II. **Dichromatism:** Two primary colors, in appropriate proportions, will match any perceived color.
- Protanopia:** Photopic sensitivity to long (red) wavelengths is decreased, and hence reds are often confused with blacks. Luminosity peaks at 540 nm; the neutral point is at 494 nm.* Wavelengths longer

than the neutral point may all appear to be the same or to differ in saturation and brightness. Reds, oranges, yellows, and greens are frequently confused. Wavelengths shorter than the neutral point are seen as blue.

- Deutanopia:** Photopic sensitivity is nearly normal, with a luminosity peak at 560 nm; the neutral point is at 499 nm. Wavelengths longer than the neutral point may all appear to be the same or to differ in saturation, but they will appear to have the same brightness as the normal. Reds, oranges, yellows, and greens are frequently confused. Wavelengths shorter than the neutral point are seen as blue.
 - Tritanopia:** Photopic spectral sensitivity is normal; the neutral point is at 570 nm.
- III. **Monochromatism:** All portions of the visible spectrum are seen as grays of differing brightness. Vision is achromatic, and all perceived colors can be matched by adjusting the intensity of any single color.
- Rod Monochromasy (typical achromatopsia):** Signs include reduced visual acuity, no Purkinje shift, nystagmus, central scotoma, and aversion to bright lights.
 - Cone Monochromasy (atypical achromatopsia):** Visual acuity is normal, and none of the signs found in rod monochromasy are present.

* The neutral point is a wavelength that can be matched with white by a dichromat.

TABLE 9-2 Color Defects Encompassed by the Terms Protan, Deutan, and Tritan

	Protan	Deutan	Tritan
Anomalous trichromat	Protanomaly	Deuteranomaly	Tritanomaly
Dichromat	Protanopia	Deuteranopia	Tritanopia

defect is described as a *blue-yellow defect*. Other than differences in color matching and chromatic discrimination, the anomalous trichromats and dichromats are visually normal; there is no reason to expect any other unique visual abnormality.

There are two types of monochromats: typical rod monochromats and cone monochromats. Rod and cone monochromats are also classified as complete achromats, which means that they have no ability to discriminate chromaticity. *Rod monochromats* have poor visual acuity (typically 20/200 [6/60]) and an aversion to bright light that is sometimes described as photophobia, although there is no pain involved (as

might occur with photophobia resulting from other causes). Rod monochromats often exhibit a pendular nystagmus that usually disappears during adolescence, a central scotoma, and no Purkinje shift. Anatomical studies have shown that rod monochromats have both rods and cones, although the cones are fewer and abnormally shaped as compared with the cones in persons with normal color vision. An excellent resource for a review of achromatopsia and a personal account of complete achromatopsia with reduced acuity was published by Hess and colleagues.²³ Cone monochromats exhibit a Purkinje shift and have normal visual acuity. The defect in color vision may be caused by altered pho-

topigments in some of these individuals, and it may be postreceptoral in others. There are two categories of incomplete achromats: those with *X-chromosome-linked incomplete achromatopsia* (also called *blue monocone monochromacy or blue-cone monochromacy*) and those with *autosomal recessive incomplete achromatopsia*. The incomplete achromats are able to discriminate colors under some—but not all—viewing conditions and light levels. For example, the blue-cone monochromats have dichromatic vision at intermediate light levels (mediated by S cones and rods) and monochromatic vision at both high (S cones) and low light levels (rods).

Patterns of Inheritance

There are two patterns of inheritance for color vision characteristics: autosomal inheritance and X-chromosome-linked inheritance. (Humans have 23 pairs of chromosomes, 22 with chromosomes of a similar size and shape, which are called the *autosomes* or *autosomal chromosomes*, and one pair that determines sex. Females have two sex chromosomes of similar size designated XX; males have one X and a smaller Y chromosome [XY]. Daughters receive an X chromosome from each parent; sons receive a Y from their father and an X from their mother.) X-chromosome-linked inheritance refers to traits inherited on the pair of chromosomes that determines the sex of the individual, and autosomal inheritance is the result of genes that code information on the 22 other pairs of chromosomes.

The Red-Green Defects. The most common inherited color defects are the red-green defects, which are inherited as X-chromosome-linked recessive characteristics. Because of this mode of inheritance, there is a greater incidence of defective color vision in males than in females. For the defect to manifest itself in a female, defective genes must be inherited on both X chromosomes. A female who inherits one defective gene is a carrier and is heterozygous for the color defect. Heterozygotes pass most of the standard clinical tests of color vision and hence are often described as phenotypically normal, although they may have some color vision deficits, such as a larger-than-normal matching range on the Nagel anomaloscope.²⁴ The male who inherits the defective gene from his mother will always manifest the defect, because males have only one X chromosome. The pattern of inheritance of color defects by children of parents with a known phenotype is illustrated in Figure 9-1.

There are racial differences in the frequency of red-green color vision defects (Table 9-3). Among Caucasians in Europe, Great Britain, and the United States, they are seen in about 8% of males and about 0.5% of females. The lowest rate, which is about 2% of males, occurs in the aboriginal populations of Australia, North America, South America, and Fiji and in certain Asian Indian tribes. Among African-American males, the inci-

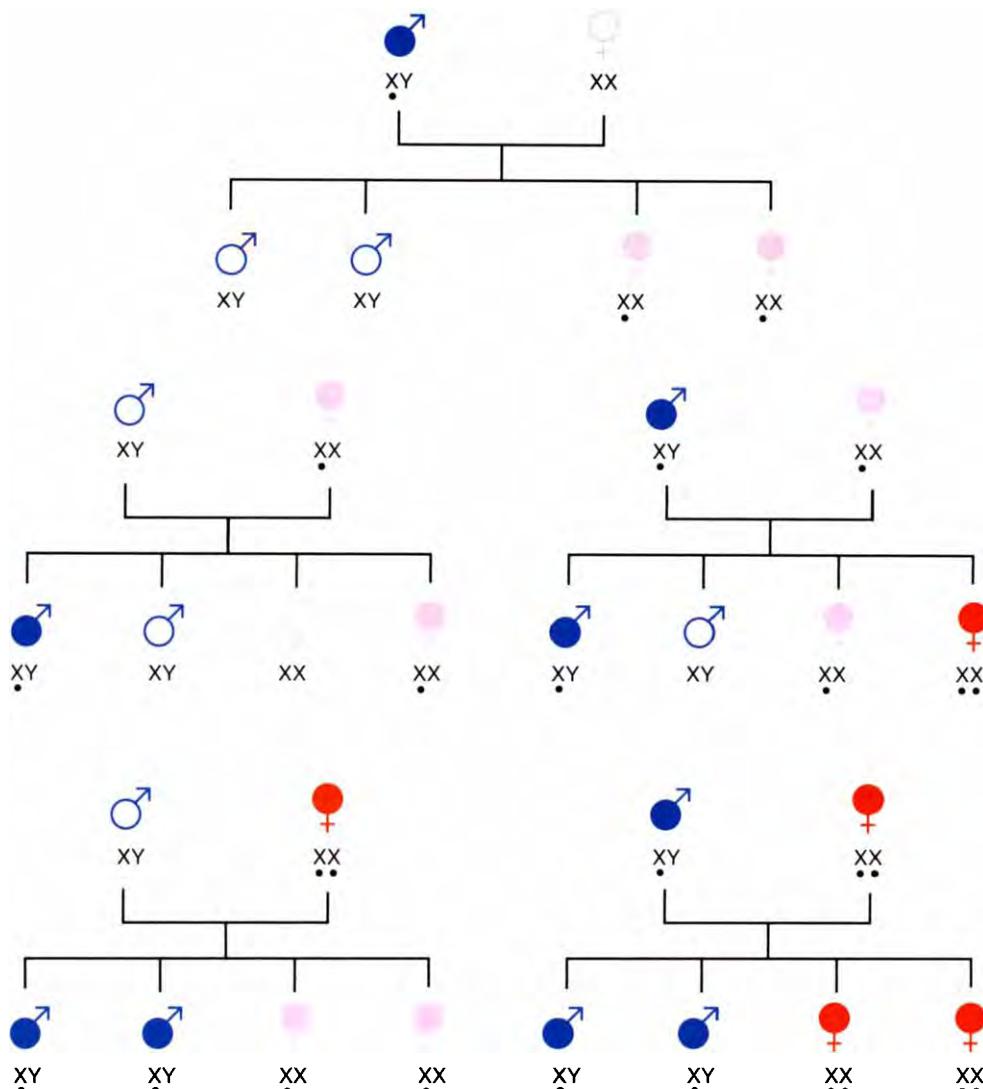
dence is about 3.6%. These differences in the prevalence of red-green color defects in males are based on a number of studies conducted between 1922 and 1962 using the Ishihara test as reported by Post.²⁵ Because proper conditions of testing may not always have been met, there is some reservation about the validity of these figures. Nonetheless, prevalence data are potentially useful for theoretical studies of genetics, and they are of clinical consequence should a significant departure from the expected occur.

The Blue-Yellow Defects. The blue-yellow defects are much rarer than the red-green defects. Tritanopia is inherited as an autosomal dominant defect, with a prevalence estimated at 0.002% to 0.008%,^{26,27} although it might be more common than 1 in 1000 or 0.1%.²⁸ Inherited tritanomaly is either extremely rare or, indeed, it may not even exist; in the past, it was mistaken for an acquired color defect.

The Achromatopsias. Complete achromatopsia with reduced visual acuity (rod monochromacy) has an incidence of about 0.0025% and is inherited as an autosomal recessive trait. Complete achromatopsia with normal visual acuity (cone monochromacy) was estimated by Pitt²⁹ to have an incidence of 0.000001%, or 1 in 100 million. This estimate was based on the premise that the cone monochromat is a double dichromat; in other words, he or she is a protanope who is also tritanopic, as Pitt's data for one subject indicated. Pitt calculated the probability of occurrence of double dichromacy by taking the product of the prevalences of tritanopia (0.0001%) and either protanopia (1.0%) or deutanopia (1.1%). For the prevalence of protanopia and deutanopia, he made the error (not uncommon) of using only the numbers for males and did not include females, so his estimate should be revised for the total population, making it 1 in 200 million. In addition, more recent figures indicate that tritanopia is more common than Pitt's estimate, occurring not in 0.0001% but rather in 0.002% to 0.007% of the population.²⁷ Hence, to the nearest million, the likelihood of a double dichromacy is 1 in 3 to 10 million. Recent evidence indicates that some cone monochromats have more than one cone type and thus are not double dichromats.³⁰⁻³² The mode of inheritance for complete achromatopsia with normal visual acuity and more than one cone type is not known. The two forms of incomplete achromatopsia are inherited as either an autosomal recessive or an X-linked recessive trait.

Molecular Biology/The Genes for the Photopigments

During the past few years, considerable progress has been made in the understanding of the molecular biology of the photopigments and the structure of the genes for the cone photopigments. The genes that encode the opsins for rhodopsin and for the three cone

**Figure 9-1**

Patterns for X-chromosome-linked recessive inheritance of the red-green color deficiencies. Dots indicate recessive defective genes. Solid blue and red circles represent individuals with a color deficiency, pink circles represent carriers, and open circles represent individuals with normal color vision.

photopigments (the L, M, and S pigments) have been determined: the rod pigment gene is on chromosome 3, the S-cone photopigment gene is on chromosome 7, and the L- and M-cone pigment genes form a head-to-tail arrangement on the X-chromosome.³³⁻³⁵

Protan and Deutan Color Vision Defects. The L and M photopigments exist in several forms, and the genes for these are found on the X chromosome, beginning with the L and followed by several M pigment genes. These genes are highly homologous, which has led to the formation of L/M hybrid genes that encode several anomalous pigments that are like the normal L-cone or M-cone pigments but with different wavelengths of maximal absorption (λ_{max}). There is also an amino-acid polymorphism at codon 180 of the

L-pigment gene that accounts for two forms of the L pigment, depending on whether the amino acid is alanine, L(ala¹⁸⁰), or serine, L(ser¹⁸⁰). The red-green dichromats have just two retinal photopigments, the S photopigment and either M in the protanope or L in the deutanope. Protanopes lack the normal L pigment, some have the normal M pigment, and others have an anomalous M pigment; deutanopes lack the normal M pigment, some have L(ala¹⁸⁰), others have L(ser¹⁸⁰), and still others have an anomalous M pigment that has a λ_{max} close to the λ_{max} of the L pigment. The red-green anomalous trichromats have three photopigments. The protanomalous have an anomalous L pigment and the normal M and S pigments; the deuteranomalous have an anomalous M pigment, either

TABLE 9-3 Prevalence and Inheritance of Color Vision Defects

Color Vision Status		Male	Female	PREVALENCE (%) Inheritance
Anomalous trichromacy				
Protanomaly		1.0	0.02	X-linked recessive
Deuteranomaly		5.0	0.38	X-linked recessive
Tritanomaly		?	?	?
Dichromacy				
Protanopia		1.0	0.02	X-linked recessive
Deutanopia		1.1	0.01	X-linked recessive
Tritanopia		0.002	0.001	Autosomal dominant
Monochromacy				
Rod monochromacy (complete achromatopsia with reduced visual acuity)		0.003	0.002	Autosomal recessive
Cone monochromacy (complete achromatopsia with normal visual acuity)		?	?	?

L(ala¹⁸⁰) or L(ser¹⁸⁰), and the S pigment. The severity of the anomalous trichromat's color defect is correlated with the magnitude of the separation of the peaks of the spectra of the pigments; a small separation of the peaks of the M and L pigments leads to a more severe defect than a large separation of peaks.

Tritan Color Defects. Tritanopia and incomplete tritanopia have an autosomal dominant inheritance and are caused by a complete or partial absence of S-cone function. Unlike the protan and deutan defects, there is no polymorphism, because the S-cone opsin gene exists as a single copy. Tritanopia results from gene mutations on chromosome 7. Tritanomaly as a true form of an inherited defect has not been well documented.

Blue-Cone Monochromacy. Blue-cone monochromacy is inherited as an X-linked recessive trait, and all reported cases so far have been in males (prevalence in females is exceedingly rare). The defect is the result of an absence or mutation of the L- and M-cone pigments. Blue-cone monochromats have reduced visual acuity and a small central scotoma (10' of arc) corresponding to the central area of the color-normal retina, in which there are no S cones (small field tritanopia).

Rod Monochromacy. Rod monochromacy, which is otherwise known as *complete achromatopsia with reduced visual acuity* or *typical monochromacy*, is inherited as an autosomal recessive trait and is characterized by a functional loss of all three cone types. The defect results from mutations in the genes encoding the cGMP gated cation channel in the cone photoreceptor, and it is the only color defect that is caused by a defect in the phototransduction pathway rather than the cone photopigment.

Acquired Color Deficiencies

Acquired color defects are frequently classified as red-green and blue-yellow. Red-green defects can be thought of as protan-like or deutan-like in the sense that color vision test results for individuals with acquired red-green defects are similar to those for individuals with an inherited color defect. Because of the rarity of inherited tritan defects, a tritan color defect is usually acquired. Achromatopsia may also be acquired; often the macula is involved, resulting in a reduction in visual acuity.³⁶ More rarely, visual acuity is normal in patients with acquired achromatopsia.³⁷

Verriest³⁸ classified acquired color defects as type I and II acquired red-green defects and type III acquired blue-yellow color defects; this classification was thoroughly described by Pinckers and colleagues.³⁹ The *type I acquired red-green defects* are progressive deteriorations that manifest themselves early by chromatic confusion along the red-green axis, a deficit in visual acuity, and a change in photopic luminosity that ultimately deteriorates to a rod or scotopic luminosity; this scotopization is likely a result of degeneration of the macular cones. With *type II acquired red-green color defects*, there is no change in luminosity, but there is a moderate to severe chromatic discrimination deficit along the red-green axis, with a milder blue-yellow loss. Type II defects are associated with optic nerve involvement (i.e., optic neuritis or optic atrophy). In patients with *type III acquired blue-yellow defects*, there is a chromatic discrimination loss along the blue-yellow axis, with a variable alteration of visual acuity. Type III defects may result from age-related changes in the ocular media (i.e., a

brunescence crystalline lens, changes in the choroid, age-related maculopathy, and glaucoma, among many other conditions).

Acquired color defects may obey a rule usually credited to Köllner⁴⁰ and hence called *Köllner's rule*: acquired blue-yellow color defects are the result of changes in the ocular media, choroid, and distal layers of the retina; acquired red-green defects are the result of changes in the optic nerve and more proximal parts of the visual pathway. The usual age changes in the crystalline lens produce an acquired blue-yellow color defect. Box 9-2

Box 9-2 Summary of the Ocular Diseases and Commonly Used Drugs Associated with Acquired Color Defects

Diseases

Red-Green Defects

- Optic neuritis
- Papillitis
- Leber's optic atrophy
- Toxic amblyopia
- Lesions of the optic nerve and pathway
- Dominant cystoid macular dystrophy*
- Hereditary juvenile macular degeneration (Stargardt's)*
- Fundus flavimaculatus*

Blue-Yellow Defects

- Glaucoma*
- Diabetes
- Retinal detachment
- Age-related maculopathy
- Chorioretinitis
- Central serous retinopathy
- Papilledema*
- Hereditary autosomal dominant optic atrophy*

Drugs

Red-Green Defects

- Antidiabetics (oral)
- Tuberculostatics

Blue-Yellow Defects

- Erythromycin
- Indomethacin
- Trimethadione
- Chloroquine derivatives
- Phenothiazine derivatives

Red-Green and/or Blue-Yellow Defects

- Ethanol
- Cardiac glycosides (Digitalis, digitoxin)
- Oral contraceptives

* Conditions that are exceptions to Köllner's rule.

shows a number of prevalent conditions and the color defects associated with them, along with some notable exceptions to Köllner's rule. The rule is useful for the early stages of a condition, but, because of the progressive nature of acquired color defects, it may not apply when a condition has advanced to a stage at which it becomes difficult to diagnose the type of color defect. Knowing the expected color defect for any given condition is helpful for a differential diagnosis. For example, it is sometimes difficult to distinguish papillitis from papilledema, but papillitis shows a red-green defect, and papilledema shows a blue-yellow defect. In addition, of all of the forms of optic atrophy, only one—hereditary dominant optic atrophy—presents a blue-yellow color defect, which is an exception to Köllner's rule. Lyle's reviews^{41,42} and the books by Fraunfelder,⁴³ Grant and Schuman,⁴⁴ and Pokorny and colleagues^{1,27} are excellent resources for information about color defects resulting from drugs and chemicals.

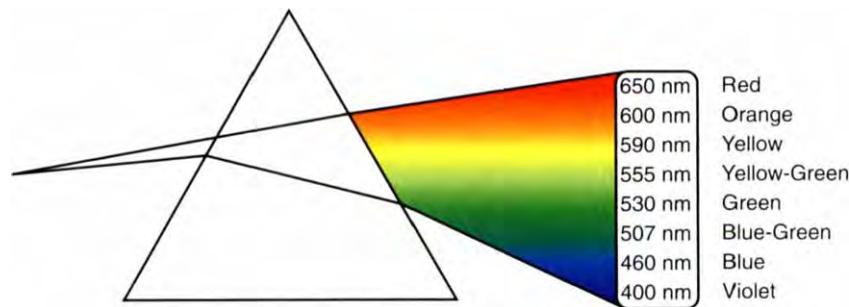
WHAT IS COLOR?

Newton used seven words to describe the spectrum produced by a prism, because repeated delineation of the spectrum fell into seven areas separated by distances equal to the numerical ratios of the frequencies of the musical chord of just intonation: the whole tone (9/8), a minor third (6/5), a fourth (4/3), a fifth (3/2), a major sixth (5/3), a minor seventh (16/9), and an octave (2/1). Of course, the spectrum is not limited to seven colors, as Newton was well aware:

The spectrum . . . did . . . appear tinged with this Series of Colours, violet, indigo, blue, green, yellow, orange, red, together with all their intermediate Degrees in a continual Succession perpetually varying. So that there appeared as many Degrees of Colours, as there were sorts of Rays differing in Refrangibility.⁴⁵

Because color terms are used to describe the sensation of a particular wavelength—saying, for example, “The brightest part of the spectrum is a yellow-green of 555 nm” and “The sky is blue and the grass is green”—it is instinctive to presume that wavelengths or objects are in fact colored. However, this is not the case. Color is a sensation and not a physical attribute of an object.⁴⁶ Color is what is seen by the eye, and it is the result of stimulation of the retina by radiant energy in a small band of wavelengths of the electromagnetic spectrum, usually considered to span about one octave (from 380 nm to 760 nm) (Figure 9-2). To relate the stimulus to the sensation requires methods for the measurement of each.

The electromagnetic radiation at any wavelength can be measured in a purely physical way in terms of its energy (joules), its radiant power (watts), or the number of quanta. These radiometric specifications provide no

**Figure 9-2**

The spectrum that results when white light is dispersed by a prism. Shown are the color names that are frequently associated with different wavelengths.

indication of how effective the energy is as a stimulus for vision. On the other hand, *photometric* quantities are based on the psychophysical measurement of the eye's spectral sensitivity, and they take into account the effectiveness of radiant energy as a stimulus for vision. Two standard spectral sensitivity curves were adopted for international use by the Commission Internationale de l'Eclairage (CIE): the 1924 standard relative luminous efficiency function, V_λ , for photopic measurements, and the 1951 standard relative scotopic luminous efficiency function, V'_λ , for scotopic measurements. These functions are shown in Figure 9-3, in which the ordinate values (or luminosity coefficients) are normalized to have a maximum value of 1.0 at 555 nm for V_λ and 507 nm for V'_λ . The luminosity coefficients are used to weight radiometric values by the spectral sensitivity of the eye and to allow for the conversion of radiometric to photometric quantities. For example, radiant intensity or flux can be converted to luminous flux using the following equation:

Equation 9-1

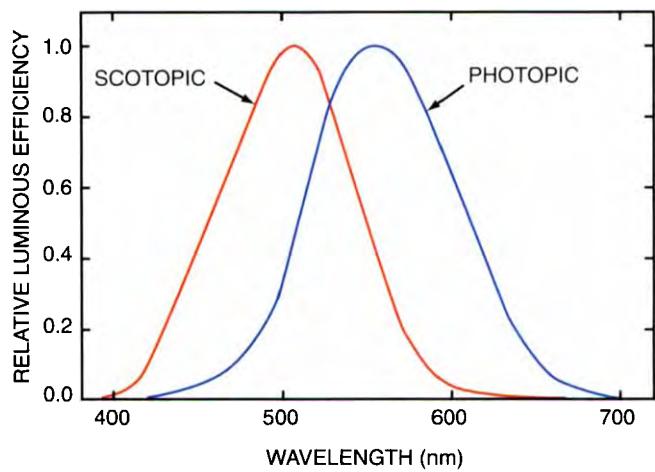
$$\Phi_v = K_m \int_{380}^{760} \Phi_{e\lambda} V_\lambda d_\lambda$$

where Φ_v is luminous intensity in lumens, K_m is a constant (683 lumens/watt), $\Phi_{e\lambda}$ is radiant flux in watts, V_λ is the relative photopic luminosity coefficient, and d_λ is the wavelength interval (usually 5 nm or 10 nm). For a scotopic specification, the constant is different, and the scotopic luminosity coefficients are used:

Equation 9-2

$$\Phi'_v = K'_m \int_{380}^{760} \Phi_{e\lambda} V'_\lambda d_\lambda$$

where Φ'_v is the luminous intensity in scotopic lumens, K'_m is 1700 scotopic lumens/watt, and V'_λ is the relative scotopic luminosity coefficient. In practice, photometric quantities are always understood to be photopic

**Figure 9-3**

The standard scotopic (V'_λ) and photopic (V_λ) luminous efficiency functions of the Commission Internationale de l'Eclairage.

unless they are specified to be scotopic. For example, the photometric unit of luminance is the cd/m^2 , which is understood to be a photopic unit unless it is written as *scotopic* cd/m^2 .

In addition to luminous intensity (Φ_v), two other quantities are necessary to specify color: dominant wavelength and purity. These two dimensions belong to the topic of colorimetry, which is discussed in the context of the principles of additive and subtractive color mixture in the next section. The many physical bases for color—incandescence, luminescence, refractive dispersion, and interference, to name a few—are fascinating to study, but they are beyond the scope of this discussion. Two excellent references are the classic *An Introduction to Color*, by Ralph M. Evans,⁴⁷ and *The Physics and Chemistry of Color: The Fifteen Causes of Color*, by Kurt Nassau.⁴⁸

Additive Color Mixtures

Physical Mixtures

Light from two or more colored sources can be added together by projecting light onto a white (neutral) screen. The result of adding partially overlapping discs of red, green, and blue light is shown in Figure 9-4. Red, green, and blue are *additive primaries*. A primary, by definition, is one member in a set of three colors that cannot be formed by a mixture of the other two. Any set

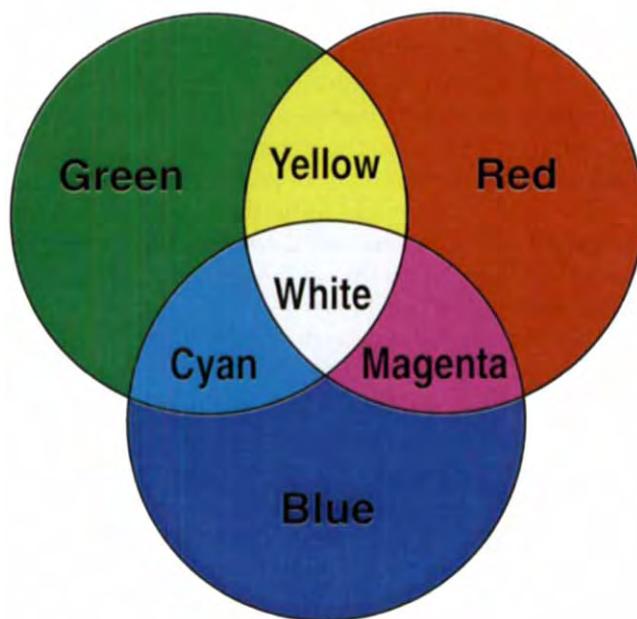


Figure 9-4

Colors that result when red, green, and blue lights are partially overlapped to give the mixture colors yellow, magenta, and cyan. When all three colors overlap with the correct intensities, white is the result.

of three colors could be considered primaries: these could be monochromatic lights or broadband spectra, as shown by the filter transmission curves in Figure 9-5.

Colorimetric equations show how the *mixture colors*—yellow (Y), cyan (CY), magenta (MG), and white (W)—are formed from the mixture of red (R), green (G), and blue (B). In these equations, the three-bars sign represents the word “produces” or “matches.”

Equation 9-3

$$R + G \equiv Y$$

$$G + B \equiv CY$$

$$B + R \equiv MG$$

$$R + G + B \equiv W$$

Psychological Mixtures

Additive mixture colors can also be produced through perceptual mechanisms or the optical properties of the eye: these could be called *visual additive mixtures*, because light is effectively mixed within the eye. One method is to place colored papers on a disc and spin the disc rapidly enough to produce a mixture color. The mixture color is perceived because the rate of rotation of the individual components is above the color fusion frequency and, if the disc is spinning fast enough, above the critical flicker fusion frequency, at which point the brightness of the component colors will fuse. Another method is to arrange small dots of differently colored ink spots close enough so that the blur circles in the retinal image overlap and hence physically mix within the eye. Color television works this way, because the spacing of the red, green, and blue phosphors on the screen is sufficiently close that the individual points cannot be resolved at the customary viewing distance or without magnification. Many well-known French Impressionists, such as Seurat, were pointillists who

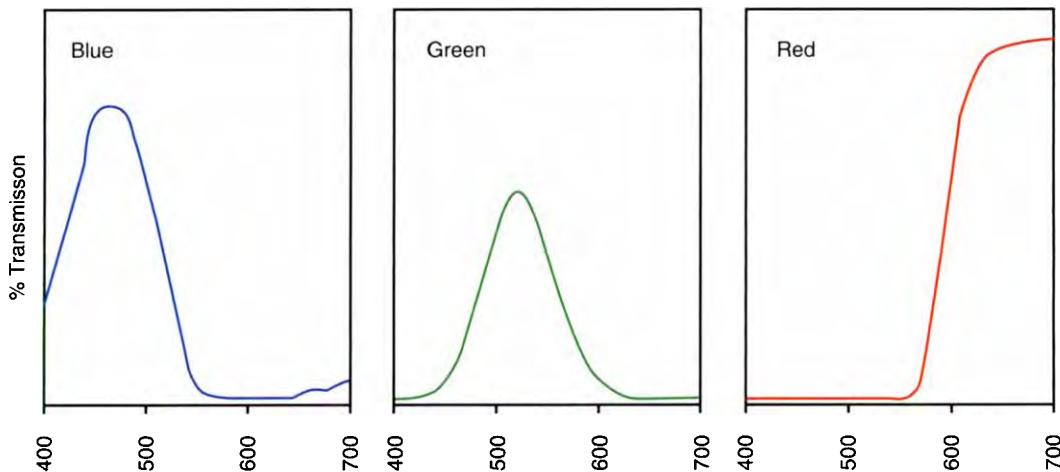


Figure 9-5

Spectral transmission curves for three filters representing blue, green, and red additive primaries.

created their paintings with small points of color that are fused by the eye to give the perception of the additive mixture. Half-tone color printing, used in most color illustrations, is done with dots of cyan, yellow, magenta, and black printed on white paper; however, the process is not quite as simple as pointillism, because some of the dots may overlap to some extent, resulting in a combination of both additive and subtractive mixing.

Subtractive Color Mixtures

Many materials selectively absorb wavelengths to produce color by subtraction. The principles involved in subtractive color mixture apply to those involved in mixing pigments, although these mixtures are a little more complex than the mere basics introduced here.⁴⁹ The spectra for a set of *subtractive primaries*—yellow, magenta, and cyan—are shown in Figure 9-6. As was shown for the mixture of all three additive primaries, white light can be thought of as being composed of red, green, and blue components. If white light is incident upon a yellow filter, the filter will selectively absorb or subtract the blue component and transmit the red and green components, which form yellow. In other words, the yellow filter can be thought of as a -B filter. A magenta filter is a -G filter, because it selectively absorbs or subtracts the green component of white, and a cyan filter is a -R, because it subtracts the red component of white. Subtractive mixing can be demonstrated by partially overlapping colored filters on a diffuse white source (Figure 9-7).

Newton's Circle

The relationship between the additive and subtractive primaries and the results of additive color mixture can

be summarized in the context of Newton's color circle, where the primaries are located on the circumference and white is located at the center, as shown in Figure 9-8. Mixture colors resulting from additive color mixture are represented along straight lines that connect the colors that are mixed. Red and green are mixed to produce yellow, which is located between the red and green, and cyan is placed between green and blue and results from their additive mixture. The three additive primaries are located on diameters opposite the three subtractive primaries. Because a diameter passes through the center—or white—point, an additive mixture of red and cyan, for example, produces white. Red and cyan are called *complementary colors*, because their additive mixture produces white. Colors on the opposite ends of any diameter are also complements: for example, yellow is the complement of blue, and magenta is the complement of green.

Two colors that appear to be the same but that are composed differently are called *metameres*. For example, yellow is produced by the additive mixture of red and green, but it also can be seen as the hue of a single monochromatic source, such as the sodium D line (589 nm). Metamerism shows that the way the visual system processes information is fundamentally different from how the auditory system processes information. If two notes are played on the piano, the two different notes are heard (with perhaps some harmonic overtones as well); however, if two different frequencies are viewed simultaneously, only one color is seen. Metamerism is central to any process involving the reproduction of color. A color photograph does not reproduce the spectral properties of the scene, but rather it provides colors that are metamerous to those in the scene. The degree to which the colors on the film are metamerous with the original is one measure of the

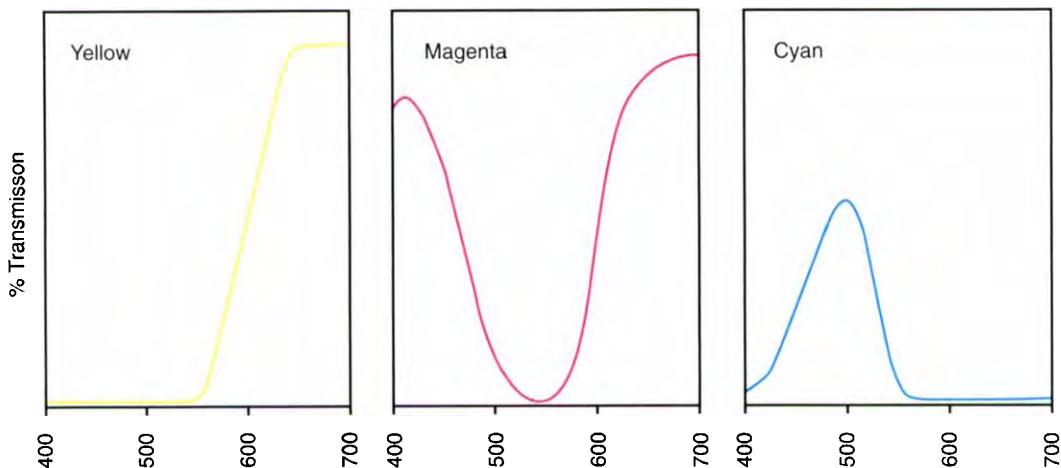


Figure 9-6

Spectral transmission curves for three filters representing yellow, magenta, and cyan subtractive primaries.

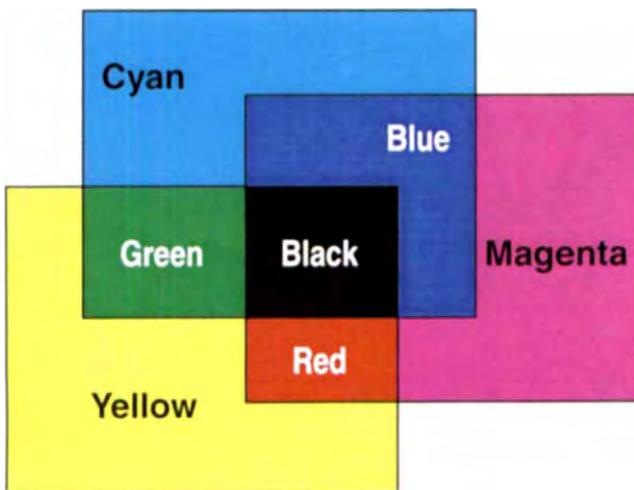


Figure 9-7

Subtractive color mixing occurs when yellow, magenta, and cyan filters are partially overlapped to produce red, green, and blue and when all three overlap to produce black.

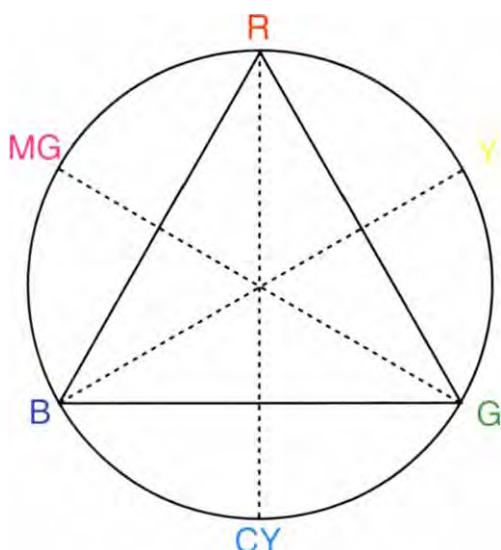


Figure 9-8

Newton's color circle. Shown are the three additive primaries—red (*R*), green (*G*), and blue (*B*)—on opposite ends of a diameter of the three subtractive primaries—yellow (*Y*), cyan (*CY*), and magenta (*MG*).

quality of film. The adequacy of any process of color reproduction—paints, photographs, television—obviously depends on there being a way to measure the limits of metamericism (i.e., a way to measure thresholds for differences in color perception). Such a measurement requires a way to measure and specify color.

The foundation for the specification of color was provided by Newton (1642–1727), and now there are

many systems in use. One of these is the CIE system of color specification, which is widely used and applicable to color vision tests. The CIE method for color notation is based on the *trichromicity* of vision, or the fact that any color can be matched by an appropriate mixture of three primaries. In Figure 9-9, A, the trichromatic principle is illustrated in the context of Newton's circle. The wavelengths of the natural spectrum are located on the circumference, which forms the boundary of all of the colors that can be seen; that is, all colors that can be seen are located on or within the circle. Between the short- and long-wavelength ends of the spectrum, there exists a range of nonspectral hues called *purples*. These purples result from the mixture of long-wavelength reds with short-wavelength blues (in fact, the circle should be flattened in the purple region). Shown on the circumference are the positions of three monochromatic primaries: red, green, and blue. The mixture of any two primaries produces a mixture color along a straight line connecting them; therefore, a triangle shows the gamut of colors that could be formed by an additive mixture of all three primaries. Now, if all colors that can be seen fall within the circle and all colors that can be matched by an additive mixture of the three primaries are within the triangle, it appears that it is not possible to match all colors by an additive mixture of the three primaries. This emerges as an apparent violation of the trichromatic principle; however, the colors located outside of the triangle but within the circle can be matched by adding one of the primaries to the color being matched. As shown in Figure 9-9, B, three primaries can be mixed to match the sample shown by the diamond (◆), but only when one primary—green, in this example—is added to the sample:

Equation 9-4

$$B + R \equiv \blacklozenge + G$$

This equation can be rearranged algebraically as follows:

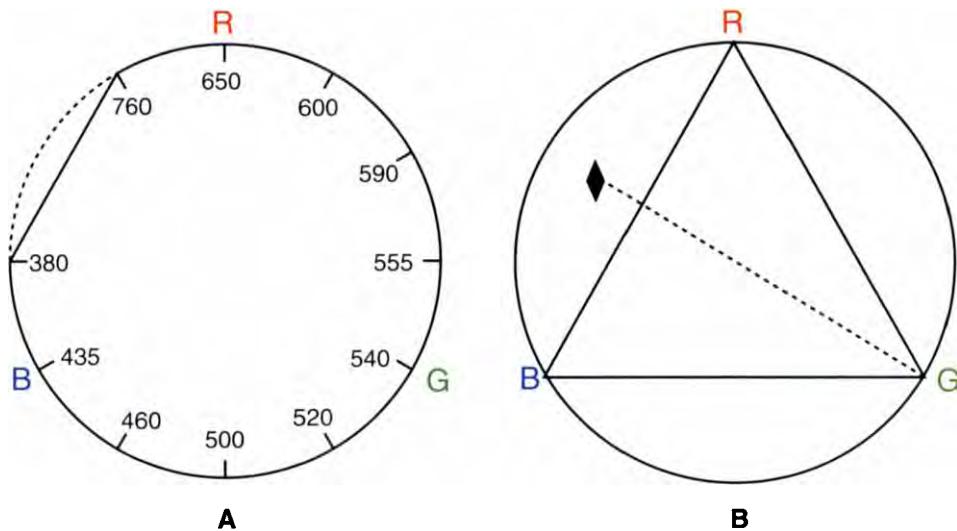
Equation 9-5

$$B + R - G \equiv \blacklozenge$$

A negative value in a colorimetric equation does not indicate a negative intensity of light (there is no such thing), but it simply means that the sample was changed by adding one of the primaries to it, as shown in Equation 4. These basics form the foundation for the CIE system of color specification.

The 1931 Commission Internationale de l'Eclairage Standard Observer

In the CIE system of color notation, the relative percentages of three primaries required to match a sample

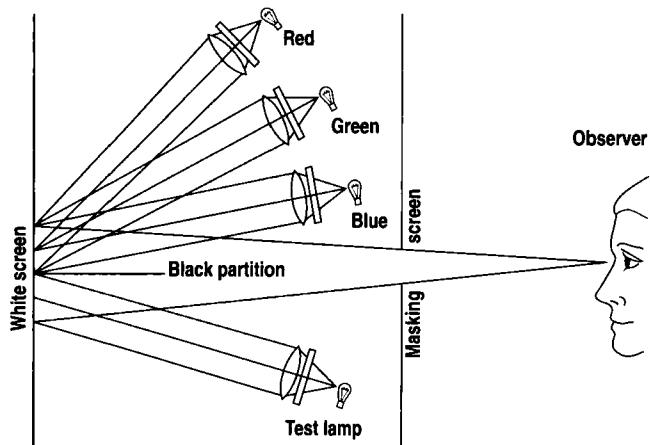
**Figure 9-9**

A, Newton's color circle showing the wavelengths of the visible spectrum and the locations of three primaries: red (R), green (G), and blue (B). B, The triangle shows the gamut of colors that can be produced by an additive mixture of the three primaries. The color located at the diamond cannot be matched by adding together R, G, and B, but it can be matched by adding G to it.

color are determined and mathematically represented on a chromaticity diagram as a point in a two-dimensional space. The locus of the color is specified by two coordinates: x and y . The CIE system of color notation and the associated chromaticity diagram are based on the results of color-matching experiments. Normal trichromatic observers adjust the relative intensities of three monochromatic primaries (red, green, and blue) to match each of the wavelengths of the visible spectrum. This is done by having any of the wavelengths to be matched on one side of a bipartite field and light from each of the primaries added to the other half of the field, as shown in Figure 9-10.

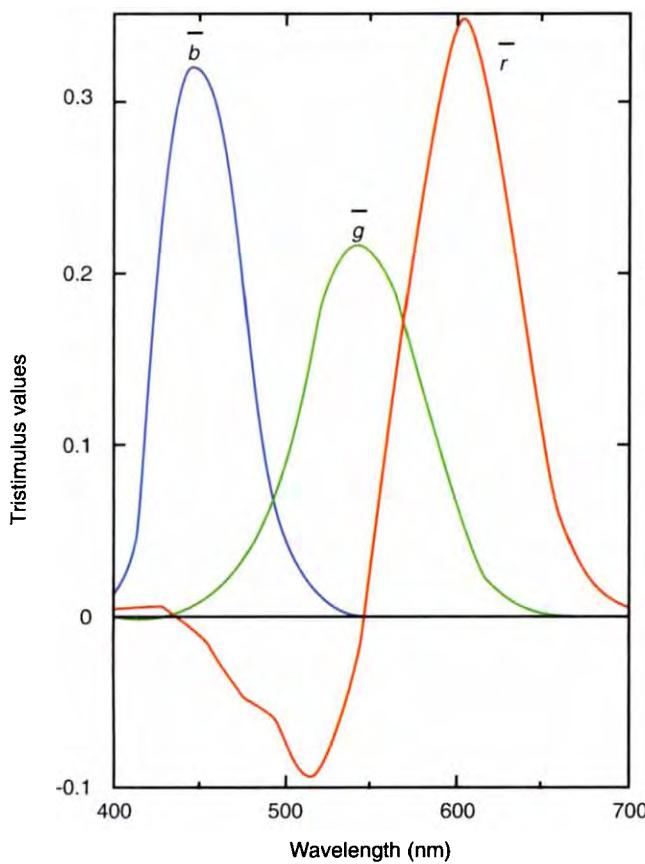
The intensity of each primary required to match all wavelengths in the visible spectrum is determined. The result is shown in Figure 9-11, in which the ordinate quantities are the tristimulus values for the spectrum, and they represent the relative intensities of the primaries required to match each wavelength. Notice that, for some wavelengths, the tristimulus values for the red primary are negative. Negative values indicate that the red primary was added to these wavelengths to achieve a match. This requirement for a negative value was illustrated earlier in the context of Newton's color circle. The CIE transformed the color-matching data so that there would be no negative values. The transformed tristimulus values are shown in Figure 9-12. These curves, which are now labeled \bar{x} , \bar{y} , and \bar{z} (read as "x bar," "y bar," and "z bar"), are the color-matching functions for the 1931 standard observer.

The CIE chose to make one set of the transformed values—those found for the green primary—to be

**Figure 9-10**

An arrangement for mixing three primary colors (red, green, and blue) to match a large number of colors provided by a test lamp. (Redrawn from Billmeyer FW Jr, Saltzman M. 1981. Principles of Color Technology, 2nd ed, p 39. New York: Wiley, with permission of John Wiley & Sons, Inc.)

equivalent to the 1924 CIE photopic luminosity coefficients (V_λ). Hence, the \bar{y} curve is equivalent to the luminosity of the spectrum. To specify the chromaticity of a color sample with the CIE system, one must first determine the reflectance spectrum for the sample. If the sample is transmitting light, as is the case with a color filter, the transmission spectrum is determined. As

**Figure 9-11**

Tristimulus values \bar{b} , \bar{g} , and \bar{r} for the mixture of three primaries to match equal-energy spectrum colors.

shown by Equations 9-6 through 9-8, the summation of the product of the reflection (ρ) or transmission (τ) coefficients at each wavelength; the tristimulus values for the spectrum \bar{x} , \bar{y} , \bar{z} ; and the radiant intensity ($\Phi_{e\lambda}$) of the source at (usually) 5- or 10-nm intervals (Δ_λ) give the tristimulus values X, Y, and Z. The Y tristimulus value is also the luminance of the color if one keeps track of the units for $\Phi_{e\lambda}$ and uses the conversion constant K_m (see Equation 9-1).

Equation 9-6

$$X = \sum_{380}^{760} \Phi_{e\lambda} \rho \bar{x} \Delta_\lambda$$

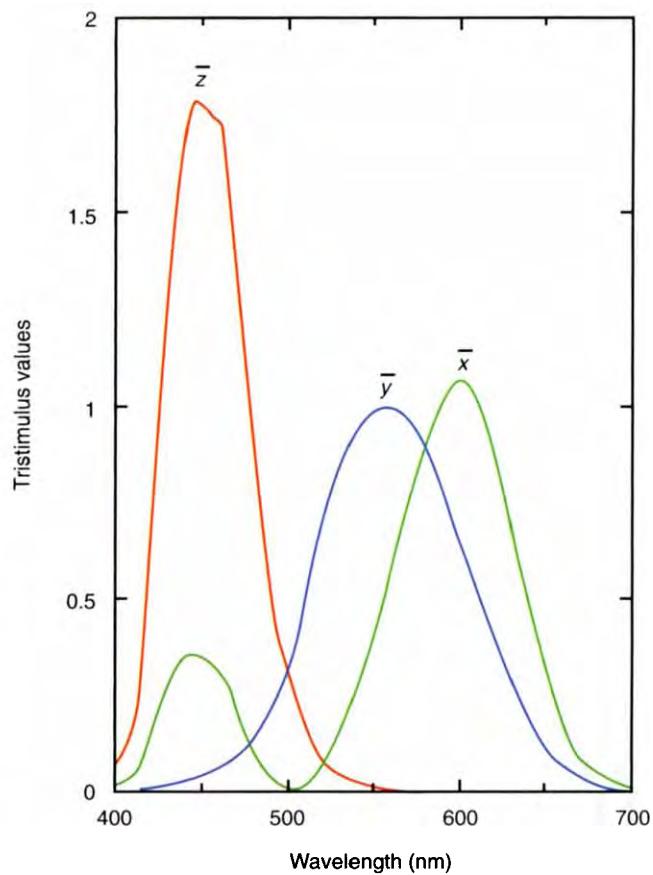
Equation 9-7

$$Y = \sum_{380}^{760} \Phi_{e\lambda} \rho \bar{y} \Delta_\lambda$$

Equation 9-8

$$Z = \sum_{380}^{760} \Phi_{e\lambda} \rho \bar{z} \Delta_\lambda$$

The tristimulus values are used to calculate the chromaticity coordinates of the sample using Equations 9-9

**Figure 9-12**

Tristimulus values for equal-energy spectrum colors for the 1931 Commission Internationale de l'Eclairage (CIE) Standard Observer. The curves \bar{x} , \bar{y} , and \bar{z} define the color-matching functions for the 1931 CIE Standard Observer.

through 9-11. Chromaticity coordinates are denoted with lowercase letters: x, y, and z.

Equation 9-9

$$x = \frac{X}{X + Y + Z}$$

Equation 9-10

$$y = \frac{Y}{X + Y + Z}$$

Equation 9-11

$$z = \frac{Z}{X + Y + Z}$$

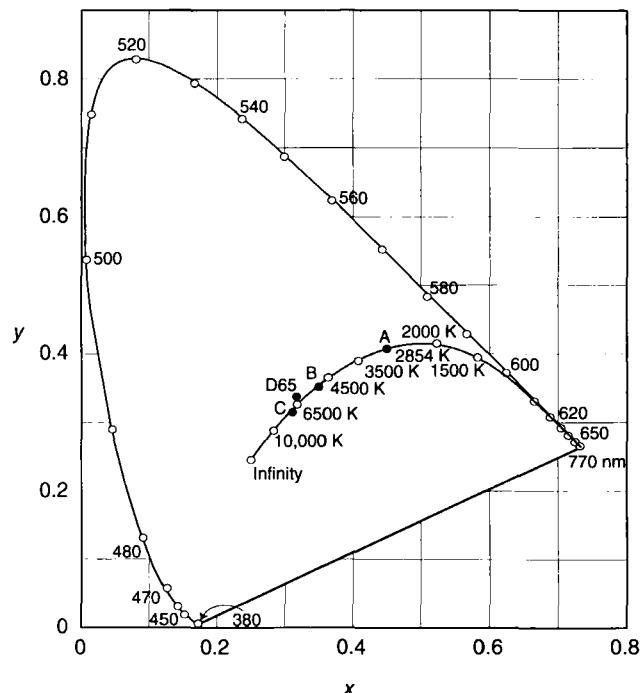
Because the chromaticity coordinates are fractional values (each representing the relative contribution of one primary in the total mixture), the sum of the coordinates equals 1.0.

Equation 9-12

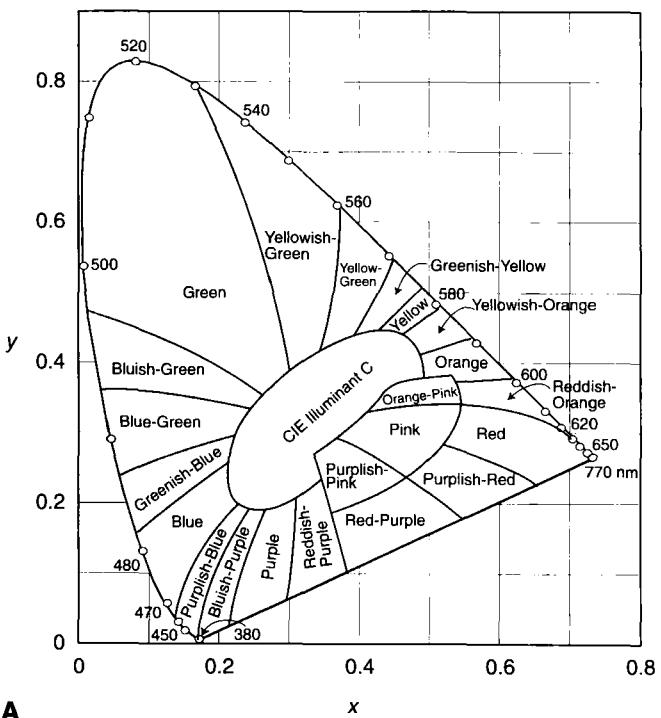
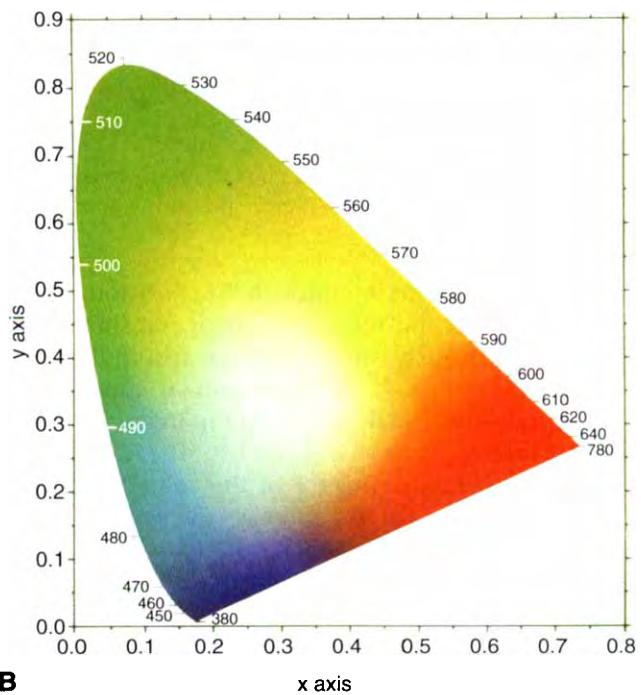
$$x + y + z = 1.0$$

The practical consequence of this last relationship is that only two coordinates (x and y) are needed to denote the chromaticity of the color numerically, and only two dimensions are needed for a chromaticity diagram (Figures 9-13 and 9-14). The third dimension can always be found by calculation.

From the chromaticity coordinates of the sample, it is straightforward to locate graphically the dominant wavelength and excitation purity of the sample relative to a particular illuminant or light source; this process is illustrated in Figure 9-15. For the purposes of colorimetry, the CIE has defined a series of standard illuminants that include standard illuminants A, B, C, and D. Each of these has an associated correlated color temperature, which is the temperature of a blackbody that matches the color of the illuminant. Standard illuminant A is an incandescent source operated at a color temperature of 2854 K. Sources B and C are prepared by allowing light from A to pass through liquid filters⁵⁰ that raise the color temperature of A; B has a color temperature of 4870 K, and C has a temperature of

**Figure 9-13**

The 1931 Commission Internationale de l'Eclairage (CIE) chromaticity diagram showing the spectrum locus with colors identified by their wavelengths, the purple boundary that joins the ends of the spectrum, and the blackbody locus with color temperatures indicated in Kelvin. Shown also are the locations for the CIE standard illuminants A, B, C, and D₆₅.

**A****B****Figure 9-14**

An approximation of the colors represented on the 1931 Commission Internationale de l'Eclairage (CIE) chromaticity diagram. A, A diagram noting the colors by name. B, A color rendition of the chromaticity diagram.

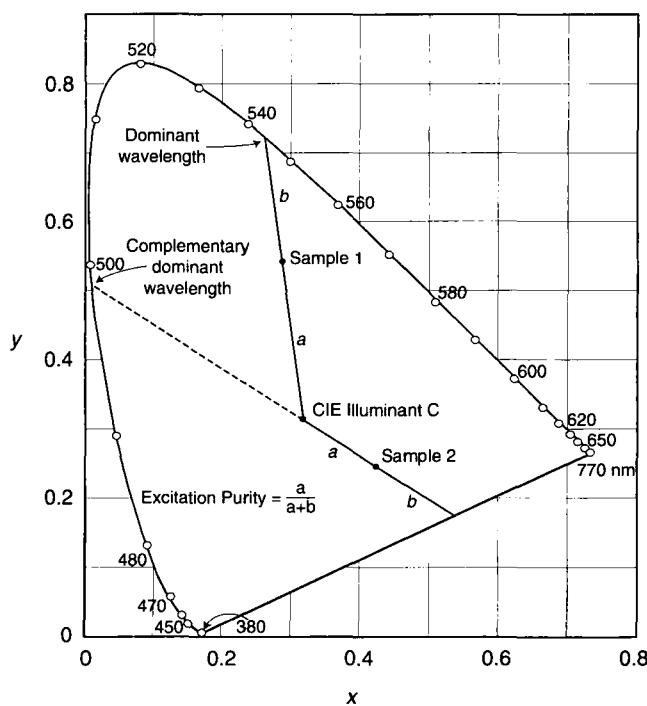


Figure 9-15

The location of the dominant wavelength and complementary dominant wavelength and a representation of excitation purity for two samples on the 1931 Commission Internationale de l'Eclairage (CIE) chromaticity diagram.

6740 K. Standard illuminant B simulates noon sunlight, and C simulates light from a completely overcast sky. The spectral outputs for these three standard illuminants are illustrated in Figure 9-16, where it can be seen that A has relatively more "red" energy and C has relatively more "blue" energy. Most color tests that use reflecting samples are standardized for standard illuminant C, which, in the past, was realized by using the Macbeth easel lamp, which is no longer commercially available. CIE Daylight D is a series of standard illuminants having different color temperatures; for example, D₆₅ is a standard illuminant having a color temperature of 6500 K. The chromaticity coordinates of the standard illuminants are shown in Figure 9-13.

There are some limitations to the application of the 1931 CIE system of color notation. The data for the 1931 Standard Observer were obtained with 2-degree foveal stimuli, and they are usually applied for stimuli that are no larger than 4 degrees. The color-matching functions for the 1964 Supplementary Observer were obtained with 10-degree targets, and, therefore, for targets larger than 4 degrees, the Supplementary Observer is used. The 1931 CIE chromaticity diagram does not represent the equal visual spacing of colors. For this purpose, either the CIELAB or CIELUV system⁵¹ or, alternatively, a

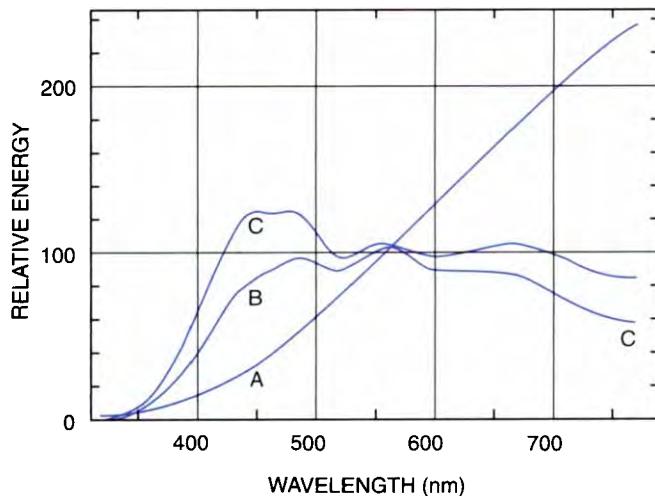


Figure 9-16

The relative energies for the Commission Internationale de l'Eclairage standard illuminants A, B, and C.

system of color notation based on equal visual steps (e.g., the Munsell system) is applied.

The Munsell System of Color Notation

The Munsell system of color notation is based on the perceptual scales of hue, saturation, and lightness, and its principal feature is the orderly arrangement of color samples by equal visual steps. To specify a color with the Munsell system requires making a visual match of the color to one in a set of standard samples that are identified by three quantities or attributes: hue, value, and chroma. These three quantities relate to the CIE dimensions of dominant wavelength, luminance, and purity, respectively.

The *Munsell hue* designation includes five principal hues with the names *red*, *yellow*, *green*, *blue*, and *purple* and five intermediate hues with the names *yellow-red*, *green-yellow*, *blue-green*, *purple-blue*, and *purple-red*. Each of the 10 hue scales is divided into 10 steps, resulting in 100 Munsell hues designated with a number and letter combination (1R, 2R, 3R... 10R). Each hue differs from an adjacent hue by the same number of just-noticeable differences (JNDs), which means that all hues are visually equally spaced. The *Munsell value* indicates the lightness or darkness of a color and neutral grays. The value scale ranges from 0 for pure black to 10 for white. A gray that is visually halfway between white and black has a value of 5. Any hue—for example, a red—has an associated value from dark red to light red. The *Munsell value* (V) is approximately equal to the square root of the percentage of luminous reflectance (R%) of a color sample; thus $V \approx \sqrt{R}$. The *Munsell chroma* scale corresponds to steps in perceived saturation, from gray to highly saturated. The chroma scale is designated with numbers from 0 to a maximum of

about 20, but the actual maximum number varies, depending on the availability of stable pigments used to produce the color samples. Munsell samples are perceived by persons with normal color vision as being equally spaced only under daylight conditions of illumination (CIE standard illuminant C). Although each dimension in the Munsell system represents colors that are visually equally spaced, the magnitude of visual spacing is different on each scale: one value step is approximately equal to two chroma steps, which are approximately equal to three hue steps.

The Munsell notation for a given color is written in the form of a pseudofraction in the following manner: hue value/chroma. An example is 5G 4/3, which is read as "five green, four slash three." When finer divisions are needed, decimals are used (e.g., 2.5G 4.5/2.2). The neutral grays are written with the letter N followed by a number for the value, for example, N9 or N5. One can appreciate the Munsell system in terms of a color solid or tree (Figure 9-17) in which the value scale is the vertical axis from black (N0) on the bottom to white (N10) at the top. Single hues are in vertical planes, and chroma increases as one moves away from the central axis. The method of specifying a color with Munsell notation is rather straightforward, and there are published

guidelines.⁵² The extensive numerical calculations needed with the CIE system are not needed with the Munsell system. The process allows one to find a visual match between the color one wishes to measure and one of the many color samples found in *The Munsell Book of Color*, which contains an orderly arrangement of some 1500 samples (available in glossy or matte). With a little practice, visual matching becomes fairly precise. Although there are several other systems of color notation (e.g., the Optical Society of America [OSA] Uniform Color Scales, the Inter Society Color Council–National Bureau of Standards [ISCC-NBS] method for designating colors, and the Pantone specification used by printers), the Munsell and CIE systems are applied to color vision tests and are commonly used by those who study vision.

ESSENTIALS OF THE PRINCIPAL THEORIES OF COLOR VISION

Trichromatic Theory

The most fundamental aspect of normal color vision is that it is trichromatic, which is evident from color mixture. Persons with normal color vision match any

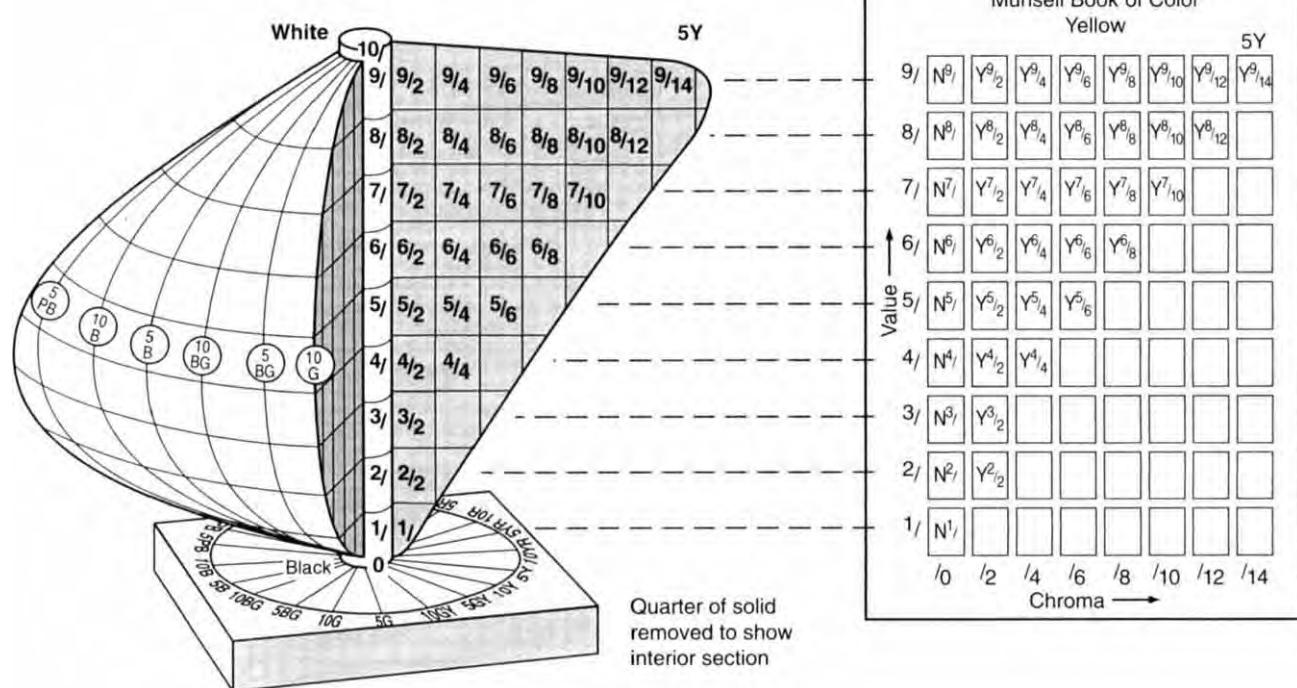


Figure 9-17

The Munsell system of color notation shown in three dimensions, with a representation of one page from the *Munsell Book of Color*. The book contains color chips, each of which is identified with by hue, value, and chroma. (From Burnham RW, Hanes RM, Bartleson CJ. 1963. Color: A Guide to Basic Facts and Concepts, p 168. New York: Wiley.)

color by a suitable mixture of three primary colors. Thomas Young⁵³ laid the foundation for a trichromatic theory of color vision. The stimulus for his theory was Newton's improbable explanation that color was encoded by visual neurons that resonated with the stimulating wavelengths. If that were the case, every point on the retina must have as many neurons as there are wavelengths that can be discriminated. According to Young:

Now, as it is almost impossible to conceive each sensitive point of the retina to contain an infinite number of particles, each capable of vibrating in perfect unison with every possible undulation, it becomes necessary to suppose the number limited; for instance to the three principal colours, red, yellow and blue.⁵³

A model for Young's theory was developed by Hermann von Helmholtz,⁵⁴ and the theory is now known as the Young–Helmholtz theory. Helmholtz envisioned three mechanisms, each having a peak response in a different spectral position. The response of each mechanism is proportional to the intensity of the stimulus, and color results from activity in all three mechanisms. One mechanism acting by itself would not suffice to encode color, because the response at any single wavelength could be the same as that at any other by simply changing the intensity. If there were only one mechanism, all wavelengths would be seen merely as differing in brightness, and, when any two wavelengths were seen as equally bright, they would not be distinguishable. The minimum requirement to discriminate one wavelength from another is two overlapping spectral mechanisms with different shapes or two mechanisms with the same shape but different peaks. The existence of three mechanisms takes color discrimination further than what is possible with two mechanisms. In the context of the Young–Helmholtz theory, dichromatic vision is explained by the loss of one of the three mechanisms found in the normal trichromat.

It is interesting that the principal notion that Young proposed—that information is coded by a comparison of the activity of a few types of broadly tuned mechanisms—appears in other areas of neurobiology and psychophysiology as a unifying construct. Apparently, however, the similarity to Young's idea has gone unnoticed.⁵⁵ In the study of vision, Young figures prominently: there are theories incorporating channels tuned to different spatial frequencies, temporal frequencies, and orientation, as there are three channels for color.

There is strong evidence in support of the trichromatic theory at the receptor level. This evidence comes from studies of photopigments, the neurophysiological responses of individual cones, and from psychophysical studies. Although no one has succeeded in extracting a cone photopigment, the difference spectra for the cones have been obtained *in vivo* via fundal reflectometry and *in vitro* by macro- and microspectrophotometry. Action

spectra from single cones using a suction electrode have also been determined.⁵⁶

With fundal reflectometry, the spectral reflectance of the fundus is measured before and after a bleaching of the retina. The difference between the two fundal reflections is the difference spectrum for the photopigment. The method was applied first to dichromats and then to normal trichromats.^{57–59} Dichromats were used because they lack one of the photopigments, making the method less complicated than with a trichromat. It was found that protanopes have a foveal pigment called *chlorolabe* and that deutanopes have a pigment called *erythrolabe*. Fundal reflectometry failed to provide any evidence for a third photopigment, *cyanolabe*, because of screening by the macular pigment or insufficient numbers of cyanolabe-containing cones.

Macrospectrophotometry is a method for obtaining the difference spectra for photopigments in a small piece of retinal tissue. Although (like fundal reflectometry) this method lacks the resolution necessary to allow for the identification of the receptor type containing the photopigment, it has provided evidence for four photopigments—rhodopsin and presumably three cone photopigments—in the human retina.⁶⁰ The most important finding provided by this method is that the same chromophore—retinal—is present in both rhodopsin and the cone pigments.

With microspectrophotometry, the difference spectrum of a single receptor may be obtained, and results have furnished evidence for three cone photopigments in the human eye.^{61,62} More recently, the photocurrent from single outer segments of monkey cones has been measured, providing evidence for three cone types distinguished on the basis of their action spectra.⁵⁶

Psychophysical methods are also used to study the sensitivity of the fundamental mechanisms. The individual spectral sensitivities of the cones are often concealed in their combined contribution, and so different methods are used to eliminate the contribution of one or more cone types so that the characteristics of the others can be studied. One approach is to study individuals who lack one cone type (i.e., dichromats). Another approach is to select spatial or temporal parameters to which one or more cone types may be insensitive. For example, the S cones feed a mechanism that is relatively insensitive in small fields (small-field tritanopia) and to rapid flicker (rates above 15 Hz). It is also possible to configure a temporal silent substitution for the L and M cones.⁶³ Finally, chromatic adaptation has been used extensively to adapt one or more mechanisms selectively. Vision scientists can combine any of these approaches to gain further advantage when trying to dissect the visual system psychophysically.

Chromatic adaptation has provided considerable leverage for understanding the three component mechanisms that are at the foundation of trichromatic theory.

The most notable approach is the Stiles two-color threshold technique. The usual procedure is to present a small monochromatic test flash of one color (λ) superimposed on a large background of another color (μ). A threshold versus intensity (TVI) curve is determined in experiments in which either the wavelength (μ) of the adapting field or the wavelength (λ) of the test field is changed. Selection of a threshold criterion on the TVI curve allows the spectral sensitivities of several mechanisms, identified by Stiles as π mechanisms, to be determined. The mechanisms are more or less independent of each other at threshold.⁶⁴ There are excellent sources that present the details of Stiles' two-color procedure.^{65,66} Wald⁶⁷ used a limited application of the method by simply determining the increment threshold for different test wavelengths in the presence of chromatic backgrounds of fixed intensity. Wald's approach was applied clinically by Marré and Marré⁶⁸ and Adams.⁶⁹

Opponent-Color Theory

Ewald Hering⁷⁰ proposed an alternate theory of color vision to account for the complementary nature of the four psychological primaries: red, green, yellow, and blue. Yellow is added as a primary because the perception of yellow, like that of the other primaries, is pure and without any hint of another color being present. Hering's theory is fundamentally trichromatic. It includes three mechanisms (red-green, yellow-blue, and white-black), but each responds in an opponent or antagonistic fashion. Hering proposed a response in one direction for the warm colors (red, yellow, and white) and a response in the opposite direction for the cold colors (green, blue, and black).

Hering's opponent-color theory was placed on a strong quantitative basis from results of psychophysical studies conducted by Hurvich and Jameson.⁷¹ The spectral response of paired opponent mechanisms—red-green and yellow-blue—was determined by a hue cancellation technique. In this method, the perception of yellow in a series of wavelengths is canceled by adding a monochromatic blue light so that neither blue nor yellow is seen. The energy of the blue that is required at each wavelength to cancel the hue provides a metric for the response of the yellow half of the yellow-blue pair. Similarly, the perception of blue in a series of wavelengths is canceled by the addition of a given yellow. The response of the red-green opponent pair is determined by using monochromatic red to cancel the sensation of green and monochromatic green to cancel the sensation of red. The results of this approach are shown in Figure 9-18, along with the spectral response of the white-black mechanism; this was determined by measuring spectral increment thresholds, and it is represented by the CIE photopic luminosity curve.

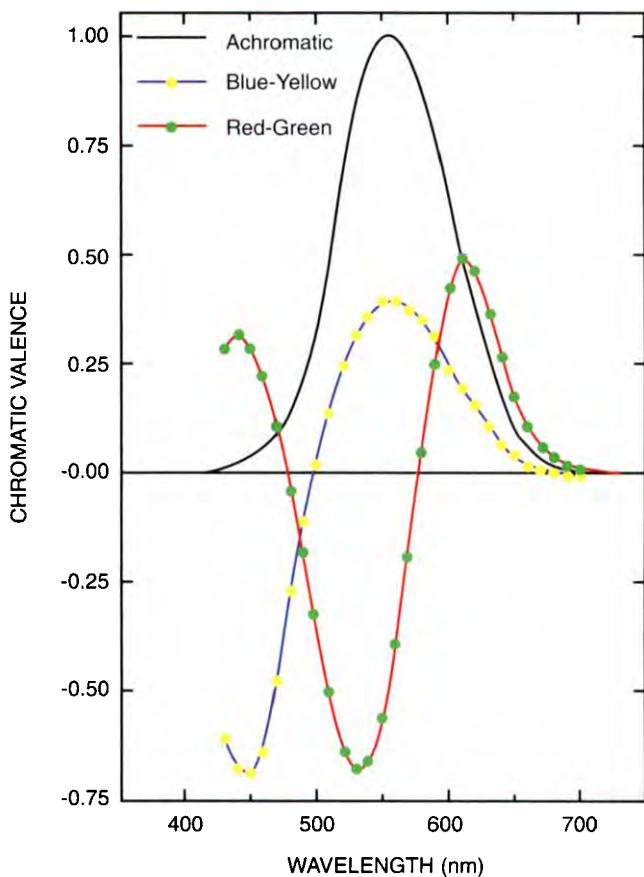


Figure 9-18

The relative responses of the chromatic functions and the achromatic (white) function for the Commission Internationale de l'Eclairage Standard Observer. (Redrawn from Graham CH. 1965. Color: Data and theories. In Graham CH [Ed], Vision and Visual Perception, p 433. New York: Wiley, with permission of John Wiley & Sons, Inc.)

Color vision is currently accounted for by a form of trichromatic-opponent model or theory; this is a combination of theories in the tradition of the Young-Helmholtz trichromatic theory and Hering's opponent-color theory. The combined theory is an appropriate resolution to the Helmholtz-Hering controversy.⁷² Trichromacy has its origin at the receptor level of the retina, and spectral opponency results from the excitatory and inhibitory receptor input to later neural elements. The perception of color results from neural interactions of the different receptor types, and color is encoded at a level beyond the photoreceptors. A single photoreceptor, like any type of photodetector (e.g., the silver halide on a sheet of photographic film or a photodiode), is color blind; the response is related to the number of quanta absorbed, regardless of the wavelength. Single photoreceptors are blind to differences of wavelength.

In most people, foveal color vision is trichromatic. This is because there are three types of cones, each of which is distinguished on the basis of the photopigment found in its outer segment. The three types are often referred to as *red cones*, *green cones*, and *blue cones*. Although the use of color names for cones is convenient, it may lead to confusion, because it implies that the cones themselves are directly responsible for the perception of red, green, and blue. Furthermore, they hardly suffice to describe the color of the photopigment within each cone type or even to define the wavelength of peak absorption for the different cone pigments (i.e., 420, 530, and 560 nm) that a person with normal color vision might label as blue, green, and yellow-green, respectively. To avoid the potential for confusion, the different classes of cones are labeled L, M, and S, which correspond with the peak absorption of the long, middle, and short wavelengths. Figure 9-19 shows a set of representative shapes of the absorbance spectra for the cone pigments plotted on both wavenumber ($1/\lambda_{\text{cm}}$) and wavelength bases. A wavenumber scale is often used to represent the spectra of photopigments. Its advantage over a wavelength scale is that the area under the curves is directly proportional to the total energy absorbed. Note that wavenumber and frequency are directly proportional to each other and that the energy per quantum is directly proportional to frequency ($E = h\nu$, where E is the energy per quantum, h is Planck's constant, and ν is frequency).

Beyond the receptor level, the cone types interact to form both achromatic and chromatic channels. There is evidence that these channels are represented by different classes of cells at the ganglion cell layer of the retina and in the lateral geniculate nucleus of the thalamus.^{73,74} Spectrally nonopponent cells respond with either excitation to all wavelengths or inhibition to all wavelengths. Spectrally opponent cells respond with excitation to certain wavelengths and inhibition to others (Figure 9-20). In addition to numerous neurophysiological studies, psychophysical methods have been used extensively to delineate the characteristics of the chromatic and achromatic pathways. Of particular relevance is the psychophysical approach precipitated by Stiles and Crawford,⁷⁵ developed by Sperling and Harwerth,⁷⁶ King-Smith,⁷⁷ and King-Smith and Carden,⁷⁸ and applied to conditions of disease by Adams.⁶⁹ Sensitivity to flicker measured in the presence of a white background is determined by either the achromatic or the chromatic channel, depending on the flicker rate or the pulse duration of the stimulus. If the test light is flickering at a slow rate (e.g., it is flickering at 1 Hz or it is a long pulse of approximately 500 msec), the spectral sensitivity curve shows three maxima at about 450, 525, and 620 nm (Figure 9-21). With rapid flicker (e.g., 25 Hz or a short pulse of approximately 10 or 20 msec), there is a single maximum at

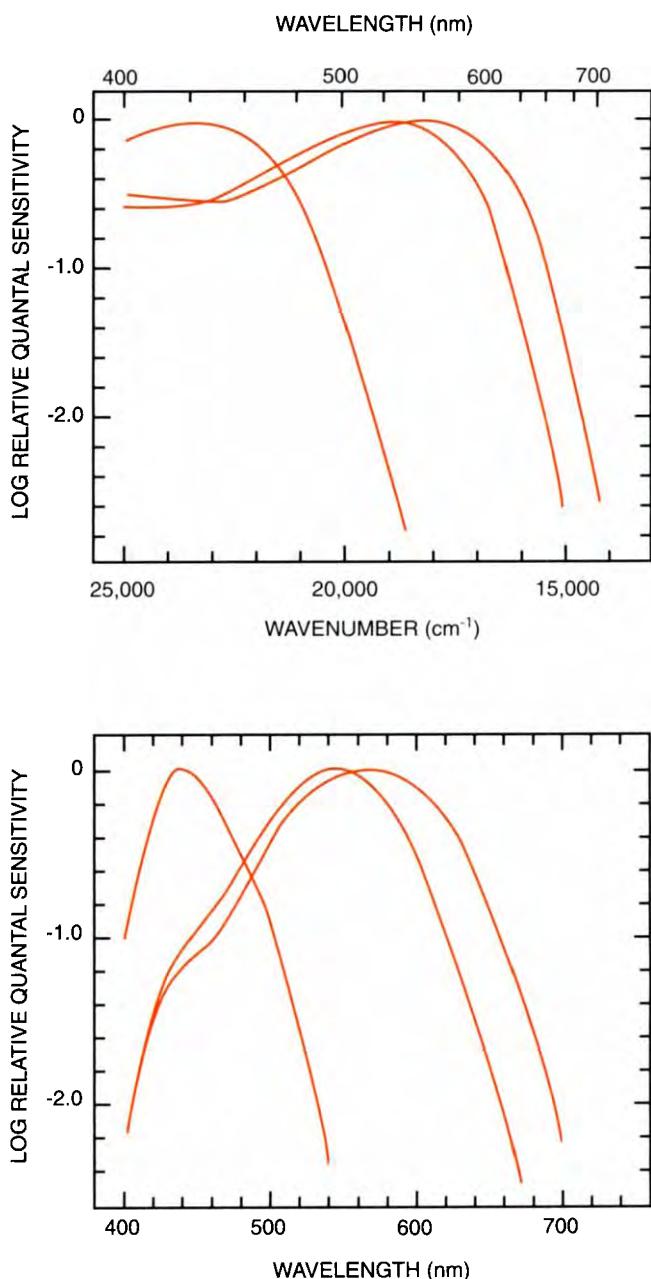
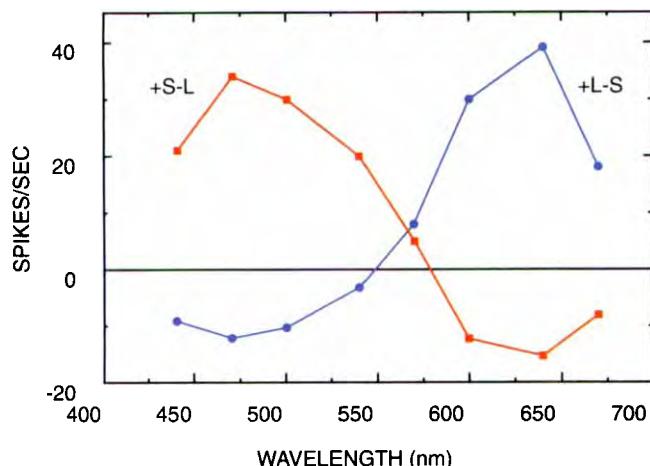
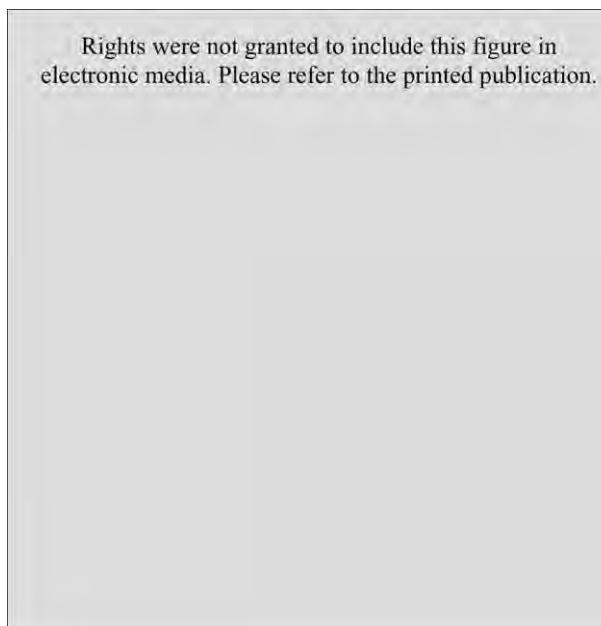


Figure 9-19

Representative spectral sensitivities of the cone visual pigments. The upper panel shows log relative quantal sensitivities at the outer segment of the photoreceptors. The lower panel shows log relative sensitivities in terms of radiant energy at the cornea. All curves are normalized to their own maxima. (Redrawn from Pokorny J, Smith VC, Verriest G. 1979. *Physiological and theoretical bases of normal color vision*. In Pokorny J, Smith VC, Verriest G, Pinckers AJGL [Eds]. *Congenital and Acquired Color Vision Defects*, p 65. New York: Grune & Stratton.)

**Figure 9-20**

The spectral responses (spikes/sec) to an equal-energy spectrum for cells at the level of the lateral geniculate nucleus of the monkey. Shown are the average response of 62 spectrally opponent cells (+L-S) that show excitation to long (L) and inhibition to short (S) wavelengths and the average response of 68 spectrally opponent cells (+S-L) that show excitation to short and inhibition to long wavelengths. (From Pease P. 1975. Spectral Properties of Monkey Lateral Geniculate Cells. Doctoral dissertation, University of California, Berkeley.)

**Figure 9-21**

Spectral sensitivities of the chromatic channel (1 Hz) and the achromatic (or luminosity) channel (25 Hz). The curves are displaced vertically by an arbitrary amount.

555 nm, representing the sensitivity of the achromatic or luminosity channel. The multiple-peaked low-frequency (slow-flicker) data show the sensitivity of the chromatic channel. The long- and middle-wavelength peaks are determined by a subtractive interaction of L and M cones, and the short-wavelength maximum is determined by the S cones.

COLOR PERCEPTION

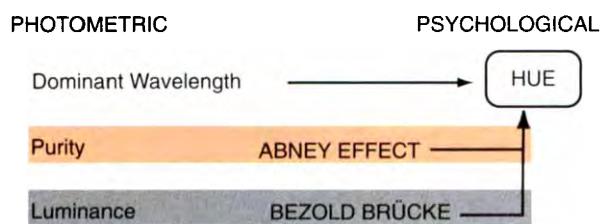
The perception of color depends on a large number of parameters, and only the most fundamental aspects are covered here. The color of an object is influenced not only by the wavelength composition of the object but also by the wavelength composition of adjacent objects or other parts of the visual field and by what was seen before the object was viewed (because of simultaneous and successive color contrast). To study these effects requires examining, at the very least, three photometric quantities: luminance, dominant wavelength, and purity. The psychological counterparts to these photometric dimensions are brightness, hue, and saturation (Figure 9-22).

Understanding the relationship between the photometric and psychological dimensions is central to understanding color perception (Figure 9-23). Color is interesting (or confusing) because the relationship between each of the photometric dimensions and the corresponding psychological dimensions is not one to one. Hue changes not only with changes in dominant wavelength but also with changes in luminance (the

	PHOTOMETRIC	PSYCHOLOGICAL
ACHROMATIC	Luminance	Brightness
CHROMATIC	Dominant Wavelength Purity	Hue Saturation

Figure 9-22

Summary of the relationship between the photometric and the psychological dimensions of color.

**Figure 9-23**

How hue is affected by changes in each photometric dimension of color.

Bezold–Brücke effect) and purity (the Abney effect). Brightness changes not only with changes in luminance, but also with changes in dominant wavelength and purity. Similarly, saturation is also dependent on each of the three photometric quantities.

There are differences in color vision among color-normal individuals. These differences are due to a combination of several factors, including density of the photopigments, waveguide properties of the receptors (Stiles–Crawford effects), effects of rod interaction with cones, screening by the macular pigment, screening by the lens, refractive error, chromatic aberration, and polymorphism of the cone photopigments.

The perception of color is also influenced by both spatial and temporal parameters in a number of ways. A gray disc surrounded by a colored annulus is seen to have the hue that is very nearly the complementary hue of the surrounding annulus; this is called *simultaneous color contrast*. If one were to fixate one color and then look to a uniform white field, the negative afterimage of the colored stimulus would be seen as having a hue that is nearly complementary to the inducing stimulus; this is *successive color contrast*. These contrast effects exaggerate or enhance the color differences that are present in most viewing situations. There is a large literature on these and many other perceptual effects, and one classic is Evans' *An Introduction to Color*.⁴⁷ To begin, it is sufficient to examine fundamental data that show how color discrimination depends on wavelength.

Color Discriminations That Depend on Wavelength

The three psychological dimensions of color—brightness, hue, and saturation—are all wavelength dependent. Discussion of these dimensions illustrates some of the fundamental attributes of perceptual discriminations for both normal trichromats and those with inherited color defects.

Spectral Sensitivity

The brightness of different colors depends on spectral sensitivity. Given an equal energy spectrum, the wavelengths to which individuals are the most sensitive will be judged as being the brightest. Only a narrow portion of the entire electromagnetic spectrum is visible to the human eye, and it is often considered to span just one octave, from 380 nm in the extreme violet to about 760 nm in the extreme red. Wavelengths beyond 760 nm are visible at only extremely high energies, and wavelengths shorter than 380 nm, except for a small window around 320 nm, are absorbed by the crystalline lens and are invisible. Psychophysical experiments are performed to determine the extent of the visible spectrum and to determine how the sensitivity of the eye varies as a function of wavelength; these experiments are

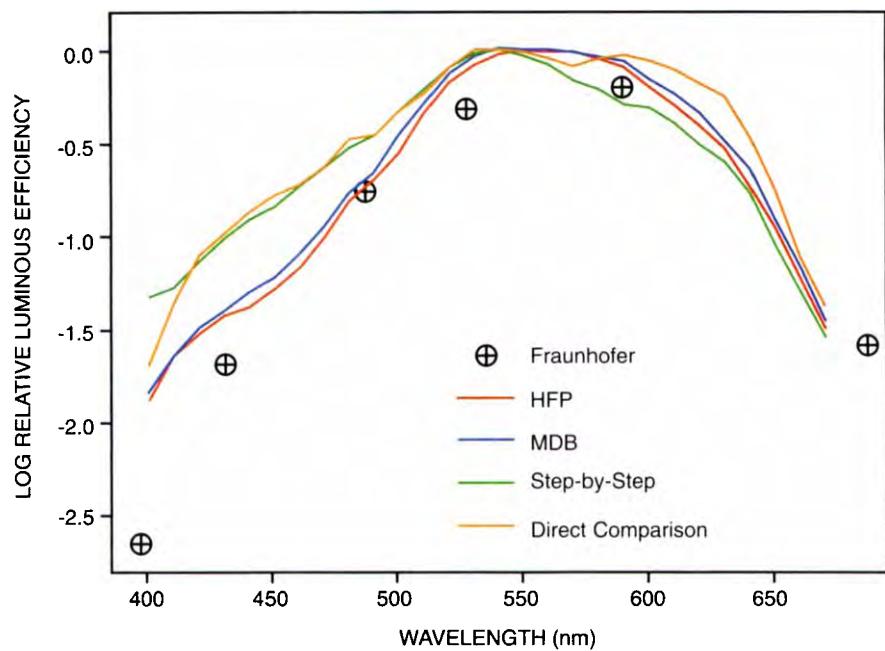
done at either a photopic or scotopic level of adaptation. Scotopic sensitivity is determined by measuring the absolute threshold for the detection of light at different wavelengths. Photopic spectral sensitivity is determined by any of several methods, each providing slightly different results that are largely caused by differences in the threshold criterion.

The methods that are used to measure photopic spectral sensitivity are also used as methods of heterochromatic photometry. Photopic spectral sensitivity can be obtained by matching the brightness of two halves of a bipartite field, with half being a standard of one wavelength and brightness and half being a comparison field. By presenting a number of different wavelengths on the comparison side, one can obtain brightness matches to the standard for the entire visible spectrum; this method is known as the *direct comparison method*. Because color differences are bothersome when one is making heterochromatic brightness matches, other techniques may be used to render the color differences less problematical. These methods include the step-by-step (or cascade) method; heterochromatic flicker photometry; and the minimally distinct border method. Each provides a different result, as is shown in Figure 9-24, along with some early measurements of Fraunhofer.⁷⁹

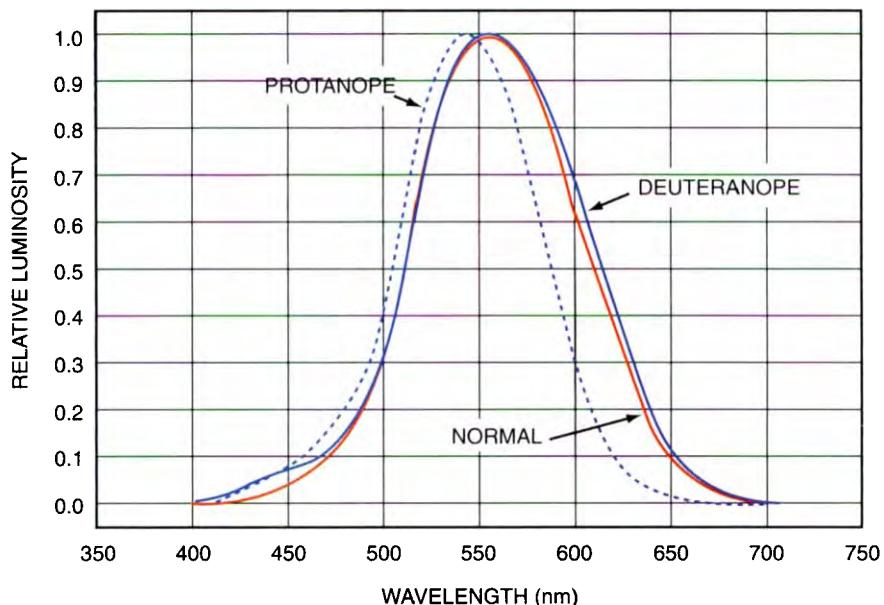
The photopic spectral sensitivity curves for a normal trichromat and the mean curve for six protanopes and six deuteranopes are shown in Figure 9-25. As may be seen, the curve for the protanopes is strikingly different from that for the normal trichromat. Its peak is at 540 nm, and sensitivity in the long wavelengths is reduced. Accordingly, protanopes see the long wavelengths as much darker than normal trichromats see them. This aspect is often described as "shortening of the red end," a phrase that erroneously suggests that protanopes do not see red. In fact they do, but they require a greater intensity than the normal. The spectral sensitivity of the deuteranopes is nearly the same as that of the normal trichromat, with a slight displacement of the peak from 555 nm to 560 nm. The brightness of the spectrum is about the same for deuteranopes and normal trichromats. Because the S cones contribute little to luminosity, the luminosity for a tritanope, who lacks the S cones, is similar to the individual with normal vision.⁸⁰

Hue Discrimination

Trichromats' and dichromats' sensitivities for detecting a change in wavelength are shown in Figure 9-26. When collecting data like these, it is important to equate the luminance (or brightness) and the colorimetric purity of the spectrum so that just one aspect of color vision is measured: in this case, sensitivity to changes in wavelength or perceived hue. However, because saturation depends on wavelength, it is difficult to measure hue discrimination independent of the perceived saturation;

**Figure 9-24**

The relative spectral luminous efficiencies obtained with four different methods of heterochromatic photometry as compared with the early measurements of Fraunhofer. *HFP*, Heterochromatic flicker photometry; *MDB*, minimally distinct border method.

**Figure 9-25**

The mean luminosity curves for an equal-energy spectrum for the protanope, the deutanope, and normal trichromats. (From Wyszecki G, Stiles WS. 1967. Color Science Concepts and Methods: Quantitative Data and Formulas, p 410. New York: Wiley.)

nonetheless, purity can be held constant. For people with normal color vision, sensitivity to hue difference is, for most of the spectrum, small (less than about 2 nm), and so the differences in saturation are negligible. However, this is not the case for color-deficient persons, who sometimes require large differences in wavelength to perceive a change in hue. Protanopes and deutanopes have no hue discrimination for wavelengths longer than about 540 nm, and tritanopes cannot distinguish hues in the region of 450 nm to 475 nm. The regions of the spectrum at which the dichromats have their best sensitivity—the peaks of the curves in Figure 9-26—are at their neutral point. The *neutral point* is the wavelength that dichromats perceive as white, and it is in the vicinity of 500 nm for protanopes and deutanopes and 570 nm for tritanopes.

Saturation Discrimination

The reciprocal of the least detectable change in colorimetric purity is used as an index of the saturation of the spectrum. Colorimetric purity (P_c) is the ratio of the luminance of monochromatic light (L_λ) to the luminance of the mixture of monochromatic and white light ($L_\lambda + L_w$).

Equation 9-13

$$P_c = \frac{L_\lambda}{L_\lambda + L_w}$$

To obtain thresholds for changes in colorimetric purity, one could start with a white stimulus and add monochromatic light until the stimulus is perceptibly differ-

ent from white, or vice versa. The reciprocal of the smallest change in colorimetric purity that can be detected is plotted as a function of wavelength to show sensitivity to differences in purity (Figure 9-27). For normal trichromats, the least saturated part of the visible spectrum is located at about 580 nm, which is in the yellow region of the spectrum. Dichromats are unable to see any differences in purity at their neutral point, and, for all other wavelengths, their sensitivity to purity differences is below that of normal trichromats. Anomalous trichromats' sensitivity to differences in purity (not shown in Figure 9-27) is intermediate between that of the normal trichromat and that of dichromats. These data indicate that individuals with a color deficiency perceive the spectrum as washed out or as less saturated as compared with the perception of normal trichromats.

MacAdam's Ellipses

JNDs in chromaticity (i.e., thresholds for changes in both hue and saturation) may be represented on the 1931 CIE chromaticity diagram. For the normal trichromat, the thresholds have the shape of ellipses, which are referred to as *MacAdam's ellipses* (Figure 9-28). The chromatic discriminations for the protanope, deutanope, and tritanope are shown in Figure 9-29. Instead of ellipses, for each dichromat, there are a number of *confusion lines*, which represent the locus of colors that cannot be distinguished on the basis of their chromaticity. The protanope's confusion line runs from 494 nm—the neutral point—through equal-energy white and toward a point just outside of the “red” corner of the chromaticity diagram. All of the confusion lines

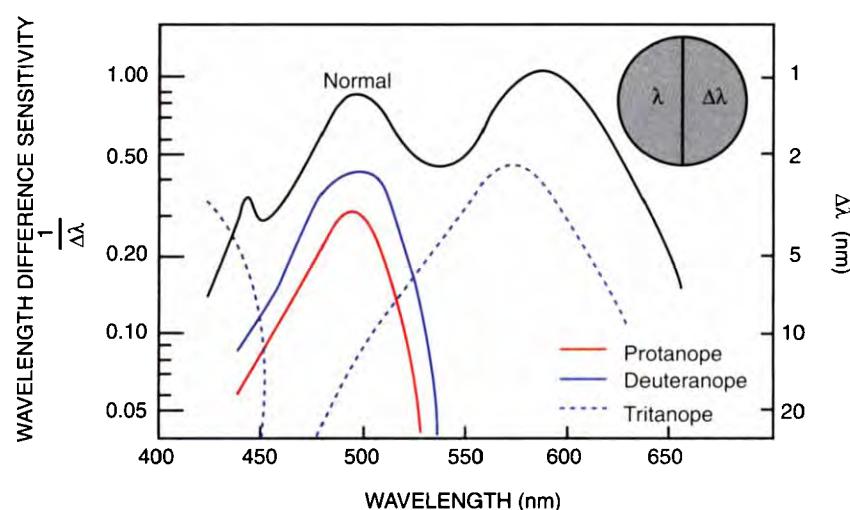
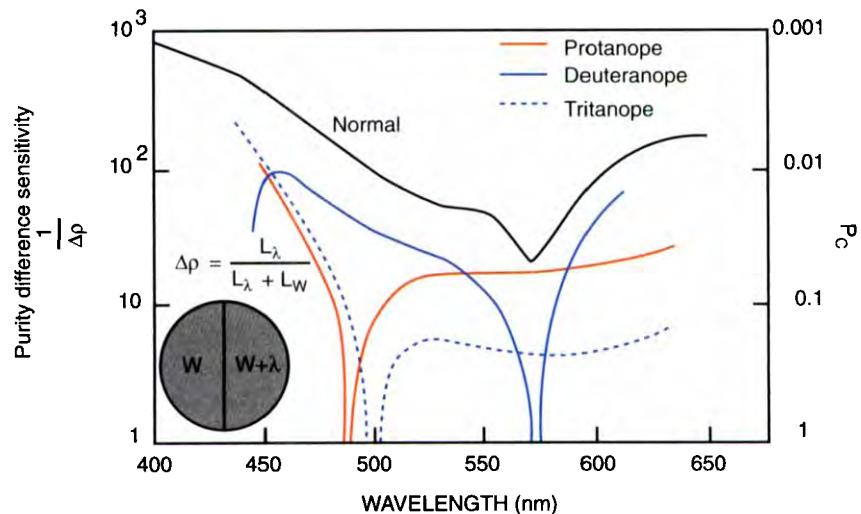
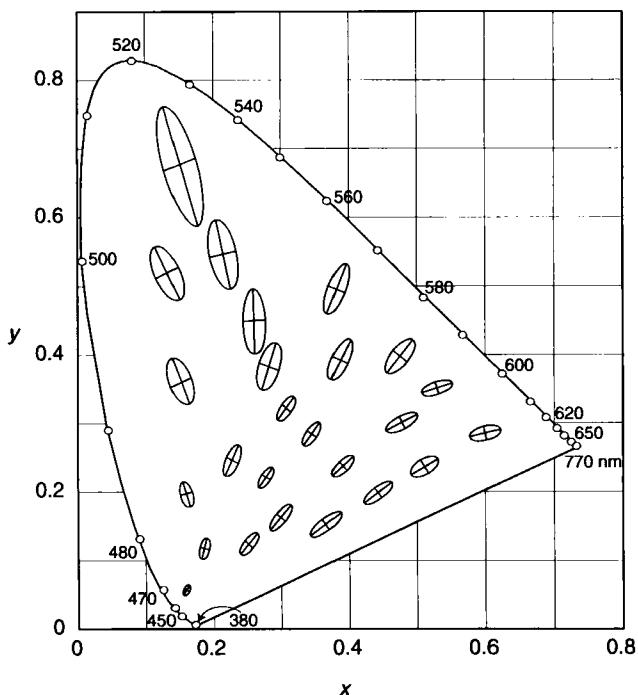


Figure 9-26

Sensitivity to differences in hue for the normal trichromat, protanope, deutanope, and tritanope. Ordinate values on the left are in terms of the reciprocal of the smallest change in wavelength that could be detected, and values on the right are in terms of wavelength. (Redrawn from Padgham CA, Saunders JE. 1975. The Perception of Light and Color, p 159. New York: Academic Press.)

**Figure 9-27**

Sensitivity to differences in purity for the normal trichromat, protanope, deutanope, and tritanope. Ordinate values on the left are in terms of the reciprocal of the smallest change in colorimetric purity that could be detected, and those on the right are in terms of the colorimetric purity. (Redrawn from Padgham CA, Saunders JE. 1975. *The Perception of Light and Color*, p 161. New York: Academic Press.)

**Figure 9-28**

The 1931 Commission Internationale de l'Eclairage chromaticity diagram showing MacAdam's ellipses, enlarged 10 times. (Redrawn from Wyszecki G, Stiles WS. 1967. *Color Science Concepts and Methods: Quantitative Data and Formulas*, p 521. New York: Wiley. © 1967, with permission of John Wiley & Sons, Inc.)

converge to a point—the *copunctal point*—as would be expected if an individual lacked one of the three fundamental color mechanisms.⁸¹ The straight line for the long-wavelength end of the spectrum (>540 nm) is also a confusion line common to protanopes and deutanopes. The confusion lines for the tritanope cross the CIE diagram with a very different slope than the slopes of the lines for the protanope and deutanope. Although there is some variability in the location of the copunctal point for each dichromat, knowing the approximate positions of these points and where different colors are located on the CIE diagram is helpful for visualizing and describing which colors will be confused by a person with a color defect.

TYPES OF COLOR VISION TESTS

There is a large inventory of color vision tests. Most of the tests that are available fit into one of the following four categories, and most practitioners use tests from the first two categories.

1. *Pseudoisochromatic (PIC) plate tests* are the most commonly used tests, and they are easily and rapidly administered. Most are designed to screen for the presence of red-green inherited color vision defects.
2. *Arrangement tests* are easily administered and useful for both inherited and acquired color defects. The results permit diagnosis of the type of defect, and they may be analyzed quantitatively for assessment of severity.

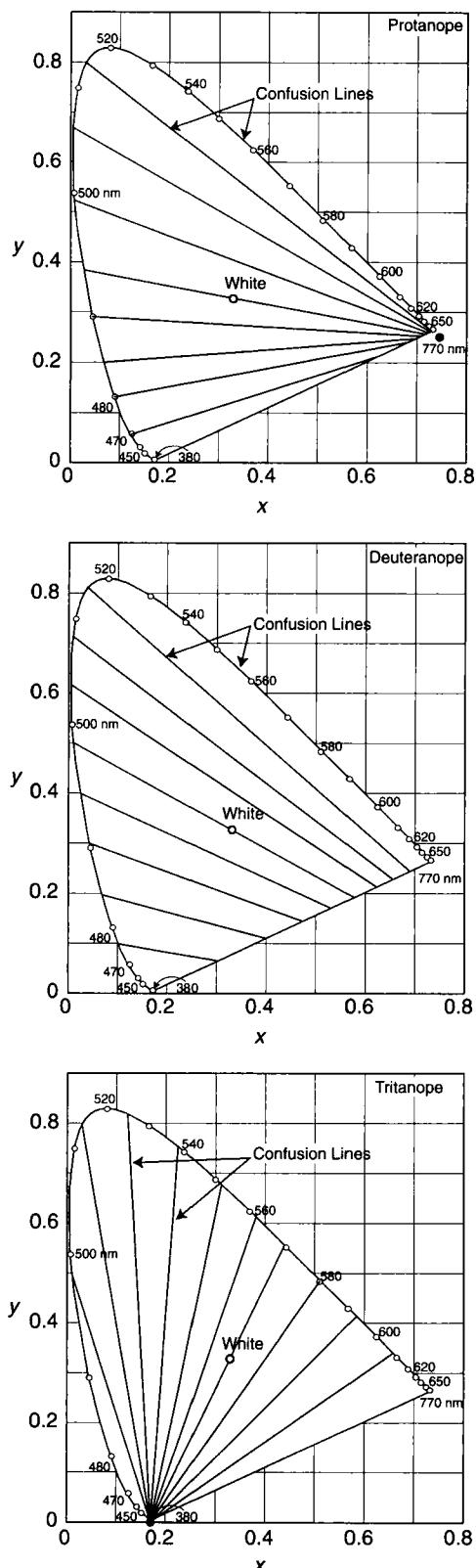


Figure 9-29

The 1931 Commission Internationale de l'Eclairage chromaticity diagram and the location of the confusion lines for a protanope, a deutanope, and a tritanope. All of the confusion loci converge to the copunctal point (•).

3. *Anomaloscopes* are generally accepted as the most accurate for diagnosis, but, unlike most other tests, they require a fair amount of skill on the part of the examiner.

4. *Occupational tests* are generally the same as those used clinically (PIC and arrangement tests), but there are also special tests designed for particular vocational requirements.

Assessment of Validity

Validity and reliability data for many color vision tests were published by the National Research Council (NRC) in 1981 in its monograph *Procedures for Testing Color Vision*.⁸² A test's validity is usually established by comparing the results from it with the results from a standard test. The Nagel anomaloscope is considered the standard for the red-green color defects.

The findings from an experimental study⁸³ illustrate how the validity of a test can be established. A new test, the Pease–Allen Color Test (PACT), and the Nagel anomaloscope were administered to a group of 233 adults. The number of individuals who passed or failed each test is indicated by the number in each cell of the 2×2 matrix shown in Figure 9-30. True-negative responses represent those who passed each test. They are considered negative responses, because the test is designed to identify a color defect. If the test is passed, the result is negative for the purposes of the test; posi-

		PACT		SPECIFICITY FOR NORMALITY $\frac{TN}{TN+FP} = 100\%$
		PASS	FAIL	
NAGEL (STD)	PASS (COLOR NORMAL)	210 TRUE NEGATIVE (TN)	0 FALSE POSITIVE (FP)	SENSITIVITY TO COLOR DEFECT $\frac{TP}{TP+FN} = 87.0\%$
	FAIL (COLOR DEFECT)	3 FALSE NEGATIVE (FN)	20 TRUE POSITIVE (TP)	
		VALIDITY FOR COLOR NORMAL	VALIDITY FOR COLOR DEFECT	
		$\frac{TN}{TN+FN} = 98.6\%$	$\frac{TP}{TP+FP} = 100\%$	

Figure 9-30

Contingency table for comparison of the Pease–Allen Color Test (PACT) results with responses obtained with the Nagel anomaloscope. The Nagel was used as the standard (STD) test for classifying a subject as having normal (pass) or defective (fail) color vision. (From Pease P, Allen J. 1988. A new test for screening color vision: Concurrent validity and utility. Am J Optom Physiol Opt 65:734.)

tive responses occur when the test is failed. The top row of the matrix shows the responses of subjects with normal color vision to each test, and the bottom row shows the responses of subjects with defective color vision. The validity of the PACT for identifying persons with normal color vision was 98.6%, meaning that some color-deficient persons (3, in this example) passed the test or gave false-negative responses. The validity of the test for identifying those with a color defect was 100%; that is, there were no false-positive results in this example. The sensitivity of the test for identifying individuals with a color defect was 87%, which means that, of the 23 subjects with color defects, 3 passed the test. The results of the two tests may be statistically compared by calculating the coefficient of agreement (\hat{K}). The coefficient of agreement can range from -1 to $+1$; the closer to $+1$ it is, the stronger the indication that the results of a test are in agreement with a standard test. The equation for calculating the coefficient of agreement, which was erroneously presented in the NRC monograph without the brackets, is as follows:

Equation 9-14

$$\hat{K} = \frac{(a+d) - \left[\frac{(a+c)(a+b)}{N} + \frac{(b+d)(c+d)}{N} \right]}{N - \left[\frac{(a+c)(a+b)}{N} + \frac{(b+d)(c+d)}{N} \right]}$$

where a represents the number of true negatives; b represents the number of false positives; c represents the number of false negatives; d represents the number of true positives; and N represents the total number of observers. The coefficient of agreement for the PACT is 0.92.

Pseudoisochromatic Plate Tests

PIC tests are designed on the basis of the color confusions made by persons with color defects. In essence, a figure or symbol in one color is placed on a background of another color so that the figure and background are isochromatic for the color-defective person. PIC tests are used primarily as screening tests to identify those with an inherited color defect, although some of the tests permit a diagnosis of type and severity. Because the inventory of PIC tests is extensive, only the more widely used tests are covered here.

PIC plates have been designed in basically four different ways⁸⁴⁻⁸⁶:

1. *Transformation plates*, in which the person with a color defect reads one figure and the person with normal color vision another;
2. *Vanishing plates*, in which the person with a color defect cannot read a figure that is easily read by the person with normal color vision;

3. *Hidden-digit plates*, in which persons with normal color vision fail to read a figure that persons with a color defect are able to read; and
4. *Diagnostic plates*, in which a figure is isochromatic for one type of defect but not another.

Depending on the purpose of a test (i.e., whether screening for red-green defects or diagnosing the type of defect), different colors are selected. For example, the Ishihara is designed as a screening test, and it incorporates colors from the red and green regions of color space. Because the confusion lines for protanopes and deutanopes have nearly the same slope for reds and greens, these colors are useful for identifying persons with a red-green defect, but they are not well suited for differential diagnosis of the type of red-green defect. The colors used in the AO-HRR test, on the other hand, are selected from a region on either side of white, where the slopes of the confusion lines for the protanopes and deutanopes are more divergent. Consequently, the results allow a diagnosis of the type of color defect. The success of any PIC test depends on a number of parameters of test construction, including careful selection and reproduction of the colors, the color contrast of figure and background, and careful attention to luminance contrast. In several of the tests, dots of different lightness are used to bracket the usual variation in brightness perception so that the figure is discriminated from the background on the basis of color contrast and not brightness contrast. If color contrast is too high, many color-deficient persons will be able to discriminate the figure; if it is too low, many with normal color vision may have difficulty. Variation of the degree of color contrast can be used to assess severity, as is done with the AO-HRR diagnostic plates.

Ishihara

The Ishihara is perhaps the most popular PIC test, and it comes in three different forms: 16 plates, 24 plates, and 38 plates. It is a screening test for protan and deutan defects, and it is unique in that it includes all four PIC plate design patterns. The symbols are Arabic numerals or wandering trails. Patients who are not able to read a numeral are asked to trace along the length of the wandering trail. Note, however, that a patient could just as easily trace a numeral, and tracing a numeral gives the examiner more useful visual feedback than tracing a wandering path. The first plate in each of the different editions is a demonstration plate that can be read correctly regardless of the status of color vision, including rod monochromacy, in which case the number is recognized because of a difference in lightness. The information provided in the manual for the test indicates that rod monochromats will miss all of the remaining test plates, but this may occur with some dichromats as well.

The pass-fail criteria for inherited color vision defects are anything but clear. For example, in the 24-plate

edition, which is probably the most frequently used of the three editions, there are 15 plates with numerals: the demonstration plate (1), six transformation plates (2–7), six vanishing plates (8–13), and two hidden-digit plates (14 and 15). According to the NRC, “The demonstration plate is included in the score” and “two errors or fewer is normal; six errors or more is deficient.”⁸² Nothing is said about three, four, or five errors. Recent data show that persons with a protan or deutan color defect will make five or more errors on the first 13 plates, excluding the demonstration plate, and three or fewer errors may occur by chance.⁸⁷ Hence, five or more errors on the first 13 (or 15) plates is a useful criterion for failure when it is necessary to report the results of the Ishihara for job requirements.

Plates 16 and 17 in the 24-plate edition are not scored for deciding upon a pass or fail of the entire test, but they are useful for making a differential diagnosis. Strong protans and deutans report only one digit on each of these plates, whereas individuals with mild color defects see both digits on each plate. The relative visibility of each digit is assessed by asking the patient which numeral is brighter or easier to see. The remaining seven plates are wandering trails with the same design features as the other plates, which use numerals.

Variations in color from one printing of the Ishihara to another are apparent, but they do not seem large enough to materially affect the validity of the test. These variations in printing quality are particularly noticeable on the two hidden-digit plates (14 and 15), which makes it possible for persons with normal color vision to easily read a figure when it should not be legible. If these plates are not scored, the consequence of this variation is removed.

AO-HRR

The AO-HRR Pseudoisochromatic Plates⁸⁴ were published initially by the American Optical Company in 1955 and 1957, but they have not been available since around 1970. Richmond Products produced a third edition in 1991 and a fourth in 2002. The third edition is out of print, and it is not recommended for clinical use. The fourth edition has been favorably evaluated both in terms of a colorimetric comparison to the original⁸⁸ and for its clinical utility for screening and diagnosis.^{89,90} There are two sections to the test: one for screening and the other for the diagnosis of type and the grading of severity. The test plates are preceded by four demonstration plates, one of which has no symbols. A plate with no symbols is a good design feature, because it can be used to demonstrate to a color-deficient patient how plates will appear when there is no symbol to be seen; it also characterizes the response that a patient gives when a symbol cannot be seen. In this test, the background color of every plate is a neutral gray printed with dots of different lightness. There are

one or two geometric symbols on each plate, and these may each be a triangle, a cross, or a circle. The screening section consists of six vanishing plates: two plates for tritan defects and four plates for protan and deutan defects. The usual criterion for failing the screening test is when any symbol (or its location) on a plate is missed or named incorrectly. The screening plates are followed by 10 plates for the diagnosis and grading of severity (mild, medium, or strong) of protan and deutan defects. As this series of plates progresses, there is an increase in the purity of the symbols. These plates are followed by four plates for the diagnosis of tritan and the hypothetical tetartan defects. People with a congenital or acquired tritan defect may be able to read both symbols on each of these four plates, but they may find the tritan symbol less bright or less visible than the tetartan symbol. According to Smith and colleagues,⁹¹ the AO-HRR plates are useful for congenital tritan defects “only if the precaution is taken of asking the subject about the relative brightness of the symbols.”

American Optical Company Plates

The American Optical Company (AOC) Plates, which is a screening test for protan and deutan defects, appears to be a composite of other tests. In addition to a demonstration plate, there are 14 test plates that include 6 transformation and 8 vanishing plates. The figures are single- and double-digit Arabic numerals. There are at least two different fonts used on different plates. The use of different fonts is not a good design, and it may account for why there are significantly more errors on this test than on the Ishihara when the viewing duration for each plate is short.⁹² Five or more errors on the 14 test plates constitute failure of the test. Plates with double-digit numbers are failed if the response to either digit is incorrect.

Dvorine

The Dvorine Test is another widely used screening test for protan and deutan defects. The test booklet contains both PIC plates and a Nomenclature Test, which is a unique and valuable feature of this test. The PIC plates are presented in two sections: 15 plates with Arabic numerals and 8 plates with wandering trails, with 1 demonstration plate in each section. Any symbol missed is an error. Three or more errors in the first section constitute a failure. Severity (mild, moderate, or severe) can be graded by the number of errors made. Dvorine published a personal account of the development of this test.⁹³

The Nomenclature portion of the Dvorine Test is used to assess color-naming ability (Figure 9-31). There are eight discs (2.54 cm in diameter) of saturated color and eight discs of unsaturated or pastel colors, which include red, brown, orange, yellow, green, blue, purple, and gray. A rotatable wheel allows the presentation of



Figure 9-31

The Dvorine Nomenclature Test. The disc is rotated to reveal each of eight saturated colors, including gray, and then the page is turned over for the presentation of eight pastel shades.

one disc at a time. Color-naming aptitude adds another dimension to a color vision assessment, and the results are appreciated by patients and employers curious to know the impact of a color defect on the ability to name colors.

The Tritan Plate (F-2)

The Tritan plate, or F-2, is a single plate that Farnsworth designed to screen for tritan color defects. It performs well in this regard,⁹¹ and it can also be used as a screening test for red-green (protan-deutan) defects. The test is a vanishing plate that consists of outlines of two interlocking squares with different chromaticities on a purple background. One square is purple-blue and vanishes for patients with red-green defects; the other square is green-yellow, and it vanishes—or is seen less distinctly as compared with the purple-blue square—for the tritan. Patients with normal color vision see both squares, but the green-yellow one is more distinct. This plate has never been commercially available, but it was supplied gratis by the Submarine Base in New London for a number of years until the supply was exhausted. It was reproduced as the frontispiece in Kalmus' book,²⁶ and instructions for constructing it have been published.⁹⁴ In its original form, the F-2 is more complex than necessary. If the interlocking squares are separated or one square is placed on a single plate, the task and the instructions to the patient become simpler, and hence results are more rapidly obtained. An appendix to this chapter includes instructions for making a modified version of the F-2. Haegerstrom-Portnoy⁹⁵ described a similar modification, and Pease and Allen⁸³ used the colors of the F-2 for the PACT (see later).

Standard Pseudoisochromatic Plates Part 1 and Part 2

Standard Pseudoisochromatic Plates Part 1⁹⁶ is a screening test for red-green defects. It has one or two digits per plate, comes with a score sheet, and has reference plates in shades of gray that can be used to familiarize a patient with the task of reading the digits. The digits are in block form, similar to those seen on handheld calculators. There are 4 demonstration plates, 10 screening plates for red-green defects, and 5 plates for the differentiation of protans and deutans. The test is reasonably effective for screening for red-green color defects, but it is less sensitive than the Ishihara.⁹⁷

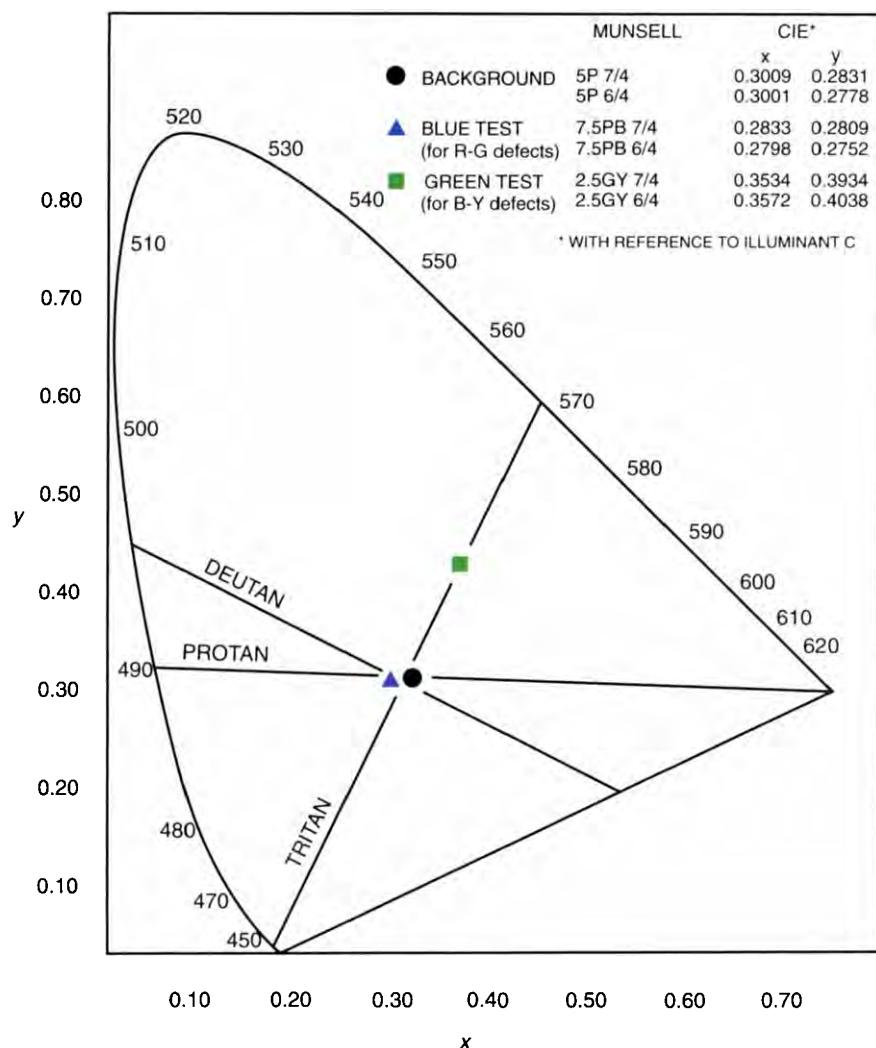
Standard Pseudoisochromatic Plates Part 2 is a screening test for acquired color defects.⁹⁸ It includes 2 demonstration plates and 10 test plates for blue-yellow defects, red-green defects, and rod monochromacy. Age-related norms for this test have been published.⁹⁹

Pease–Allen Color Test

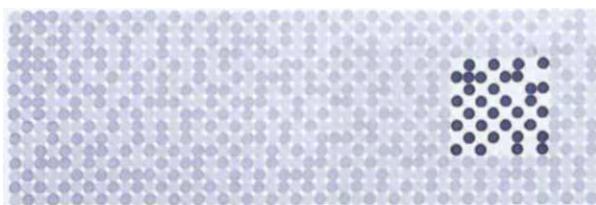
The PACT is a screening test for both red-green and blue-yellow color defects. Although it is not commercially available, the details of its construction have been published.⁸³ The test consists of four plates that can be administered to both verbal and nonverbal observers (including infants) using pointing or preferential looking. Figure 9-32 shows the chromaticity coordinates of the colors, which are essentially the same as those on the F-2. The four plates include a demonstration plate, two test plates, and a plate with no figure. Each plate (Figure 9-33) is a relatively large rectangle (10 cm × 30 cm) prepared with small discs of Munsell papers. On the demonstration plate and on each of the test plates, a square test figure is located to one side of the background. Depending on the orientation of the plate, the patient is asked to identify on which side the test figure is located. The PACT can be administered rapidly, and the task is often self-evident: young children (3 to 5 years old) will frequently point to the test figure on the demonstration plate before the instructions are given. A finger puppet is an effective pointer for young children, and it also protects the plates from fingerprints. The PACT is especially useful for toddlers, who fail many other tests, probably because the task demands are inappropriate for their age. Using a preferential-looking paradigm, the PACT can be used with infants, mentally retarded or nonverbal adults, and adults with disabilities.⁸³

Arrangement Tests

There are several different arrangement tests that require the observer to place colored samples in sequential order on the basis of hue, saturation, or lightness or to sort samples on the basis of similarity. One of the earliest tests of this nature that is still available but is rarely

**Figure 9-32**

The 1931 Commission Internationale de l'Eclairage (CIE) chromaticity diagram showing the coordinates for the color of the Pease-Allen Color Test and representative confusion loci for protans, deutans, and tritans. R-G, Red-green; B-Y, blue-yellow. (From Pease P, Allen J. 1988. A new test for screening color vision: Concurrent validity and utility. Am J Optom Physiol Opt 65:731.)

**Figure 9-33**

The Pease-Allen Color Test demonstration plate made of 6.4-mm ($\frac{1}{4}$ -inch) discs of neutral Munsell papers of two values: N6 and N7. The square test figure on the right side of the background was made of neutral Munsell papers: N3.5 and N9.5. (From Pease P, Allen J. 1988. A new test for screening color vision: Concurrent validity and utility. Am J Optom Physiol Opt 65:731.)

used today is the Holmgren Wool Test. In this matching test, 46 numerically coded comparison skeins of yarn are selected to match three test colors: yellow-green, pink, and dark red. The comparison skeins differ from the test skeins by being lighter or darker. The test is not accurate for screening or classification, and it is not recommended for clinical use.¹⁰⁰ It is of historical significance as an early occupational test.

The clinical arrangement tests that are in use today all use colored papers mounted in black plastic caps. The caps are placed in order according to specific instructions, and the order is recorded as the sequence of numbers printed on the underside of the caps. Results are plotted on score forms for analysis and interpretation, and quantitative scores are computed. The tests are standardized for CIE standard illuminant C.

Farnsworth–Munsell 100-Hue Test

The Farnsworth–Munsell 100-Hue Test (FM 100-Hue) is a sensitive test for assessing color discrimination, and it has been used extensively. Results are especially useful for monitoring changes in the status of color vision and for assessing differences between the two eyes. The colors are Munsell papers of different hues but of about the same Munsell value and chroma that are placed in black plastic caps that subtend about 1.5 degrees at the usual test distance (i.e., eye to desktop). The test includes 85 different caps that are numbered on the underside and divided into four trays (Figure 9-34). Lakowski¹⁰¹ has published the CIE coordinates for every fifth cap in the test. Adjacent caps are separated by about the same number of JNDs in chromaticity for persons with normal color vision under standard illuminant C. Originally, there were 100 different hues in the test, but 15 were removed by Farnsworth¹⁰² to make the series more uniform. Even so, Lakowski¹⁰³ believed the color difference in trays 1 and 4 is smaller than those in trays 2 and 3.

Each tray contains two caps at either end that are fixed in position as reference points; these are the same as the last and first moveable caps in the immediately preceding and succeeding trays. The trays are hinged so

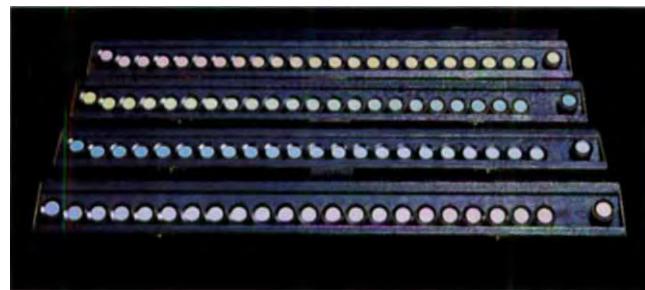


Figure 9-34

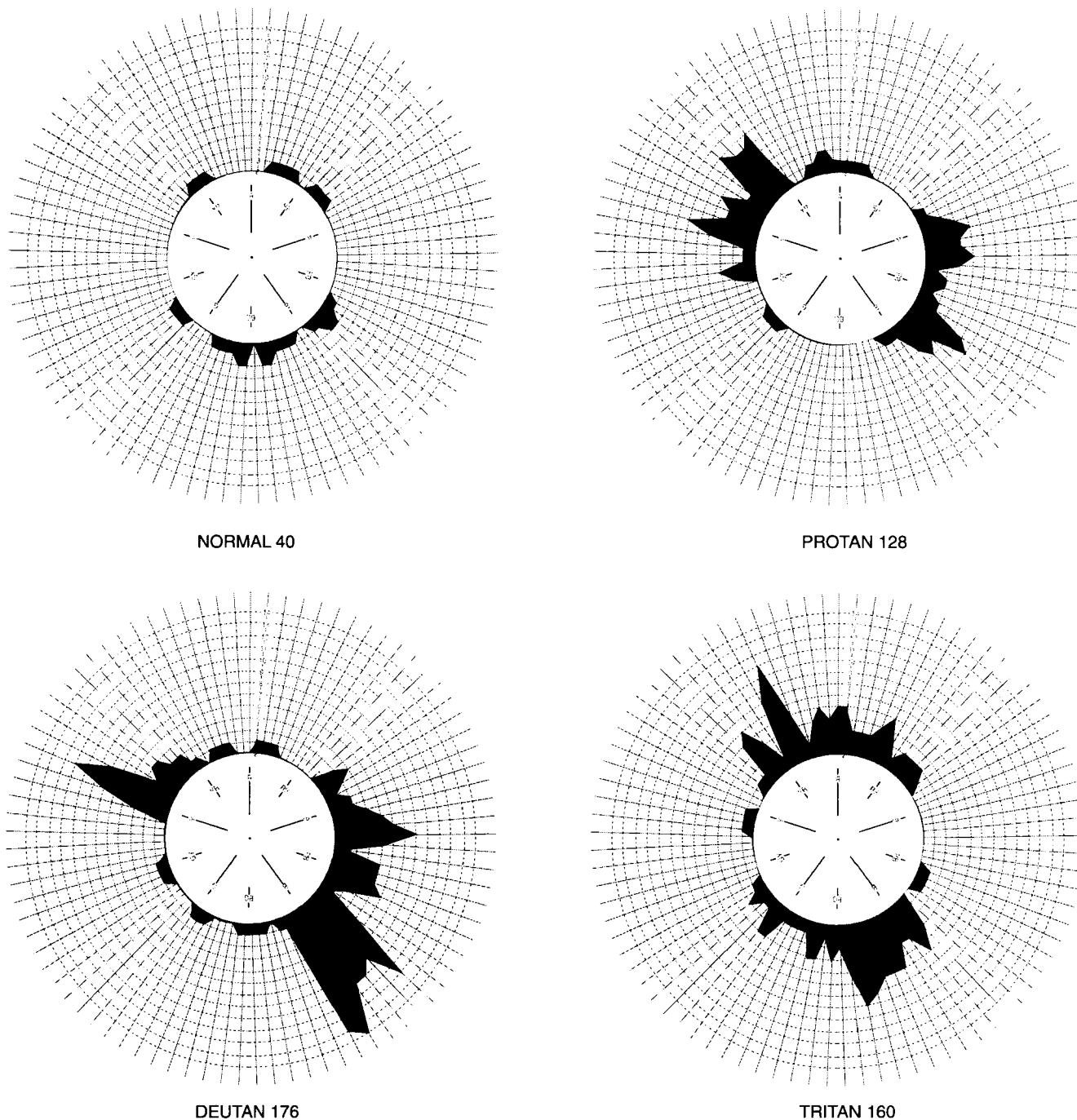
The four trays of the Farnsworth–Munsell 100-Hue Test.

that, when they are opened, the surface is normal to the line of sight and 45 degrees to the direction of illumination. To administer the test, the clinician removes the moveable caps from one tray at a time and arranges them in random sequence before the opened tray. The patient is instructed to “[s]elect a cap that is most like the reference cap, place it next to it, and then select another cap most like the one just selected, and so on.” It is better not to use the word *color* in the instructions, because it may be confusing to some patients. Each tray can be completed in about 2 minutes. Repeat testing is important, because improvements occur with learning.^{104–106}

The sequence of numbers for each tray is recorded on a score form that includes a polar coordinate graph for plotting the error score for each cap. The error score for a cap is equal to the sum of the absolute differences between the number of the cap and those adjacent to it, and it is an indication of the magnitude of perceived color difference between adjacent caps. Results are interpreted by looking at the plotted pattern of error scores. Graphs are plotted in one of two ways: either the Farnsworth method, in which the error score is plotted on a radial line for each cap, or the Kinnear method, in which error scores are plotted in sequential order. If one is plotting by hand, the Kinnear method is by far the easier of the two. Regardless of the method, the diagnosis remains the same.¹⁰⁷ The polar coordinate graph of error scores for color-deficient patients has a distinctive bipolar pattern or butterfly appearance, as shown in Figure 9-35. Patients with normal color vision make errors, but they are more or less random. Test performance can be rated by computing a total error score, which is obtained by subtracting 2 from each cap error score and summing the remainders or by summing all of the cap error scores and subtracting 170 (i.e., 2×85) from this total.

Several sets of normative data and data on the effects of age have been provided,^{38,86,102,108–110} and the significance of a difference in test results between the two eyes has been assessed.¹¹¹ Normograms have been published that allow the clinician to determine whether a patient's score is within normal limits for age and interocular difference.¹¹² Random cap arrangements result in an error score of 984; scores greater than this are probably not dependent on useful vision.^{113,114}

Scoring by hand is tedious and subject to simple arithmetic errors. Automated recording and scoring devices are available,¹¹⁵ and bar code readers have been developed.^{110,116} In addition, there are several different approaches for computer analysis and scoring once the patient's results have been manually entered.^{117–125} Provided the numbers are entered correctly, computer scoring is faster and more accurate than hand scoring. Furthermore, computer scoring can facilitate the diagnosis.

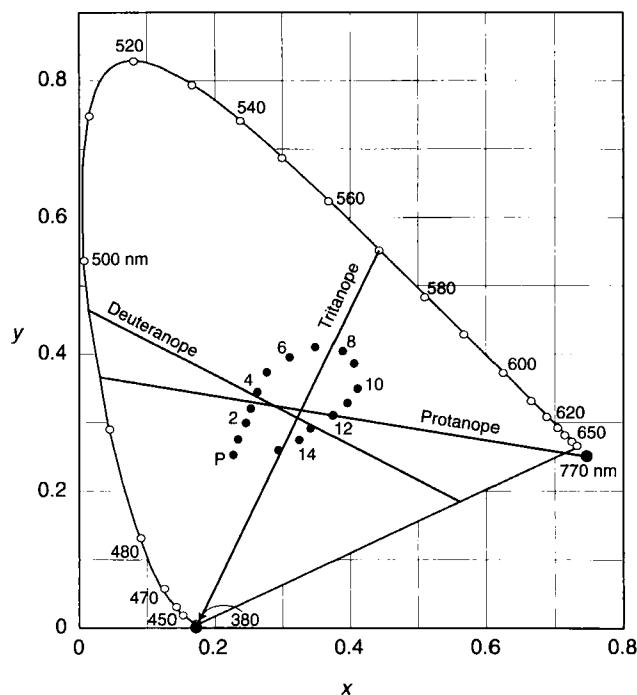
**Figure 9-35**

Results of the Farnsworth–Munsell 100-Hue Test from a person with normal color vision and representative examples for a protan, a deutan, and a tritan. Numbers are total error scores.

Two tests derived from the FM 100-Hue are the Ohta 40-Hue Test, which uses about every other cap from the FM 100-Hue, and the Roth 28-Hue Test, which uses every third cap.^{126,127} Pincker's version of the Panel D-15 test (see below) uses caps from the FM 100-Hue,^{128,129} and there are also caps in the 100-Hue that are similar to box 4 of the New Color Test.¹²⁷

Farnsworth Dichotomous Test for Color Blindness (Panel D-15 and Large Panel D-15)

The Farnsworth Dichotomous Test for Color Blindness (Panel D-15) is designed to "distinguish the functionally color blind from the moderately color defective and the normal."²² The results are dichotomous: pass or fail. Those who fail are likely to experience problems with

**Figure 9-36**

Commission Internationale de l'Eclairage chromaticity coordinates of the colors of Panel D-15 and representative confusion lines for a protanope, a deutanope, and a tritanope. P, Pilot or reference cap.

color in some everyday situations and to not be able to meet certain occupational requirements. The test is designed on the basis of the characteristic color confusions shown in Figure 9-36. The Panel D-15 includes 1 fixed or reference color and 15 different colored papers (Table 9-4) placed in moveable caps within a tray. The instructions for administering the test are as described for the FM 100-Hue.

Test results are analyzed with a score form that is supplied with the test. The form has a color circle indexed with the slopes of confusion lines for protan, deutan, and tritan color defects. Missing from the score form is a scotopic axis (Figure 9-37), which is found with typical achromats (rod monochromats). Lines are drawn by connecting numbers on the circle according to the sequential order of the caps as arranged by the patient; the test is failed when one or more of these lines cross the circle. The slope of the lines is used to diagnose the type of defect. Representative examples are shown in Figure 9-37.

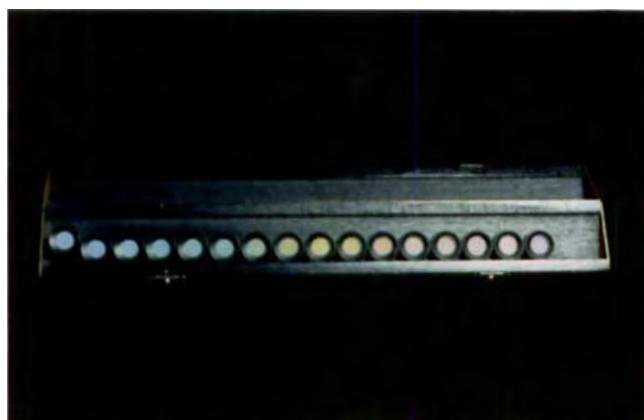
The clinical utility of the Panel D-15 can be increased with a more stringent pass/fail criterion that specifies that a failure occurs when there is more than one single-place error or when there is any error greater than a single-place error.¹³⁰ Some examples illustrate this criterion. The sequence 1, 2, 4, 3, 5, 6 through 15 includes a single-place error, with only caps 3 and 4 out of proper

TABLE 9-4 Colorimetric Specifications for the Panel D-15

Cap no.	CIE CHROMATICITY COORDINATES		MUNSELL NOTATION	
	x	y	Hue	Value/ Chroma
Pilot	.228	.254	10.0 B	5/6
1	.235	.277	5.0 B	5/4
2	.247	.301	10.0 BG	5/4
3	.254	.322	5.0 BG	5/4
4	.264	.346	10.0 G	5/4
5	.278	.375	5.0 G	5/4
6	.312	.397	10.0 GY	5/4
7	.350	.412	5.0 GY	5/4
8	.390	.406	5.0 Y	5/4
9	.407	.388	10.0 YR	5/4
10	.412	.351	2.5 YR	5/4
11	.397	.330	7.5 R	5/4
12	.376	.312	2.5 R	5/4
13	.343	.293	5.0 RP	5/4
14	.326	.276	10.0 P	5/4
15	.295	.261	5.0 P	5/4

CIE, Commission Internationale de l'Eclairage.

From Paulson HM. 1973. Comparison of color vision tests used by the armed forces. In *Color Vision*, p 62. Washington: National Academy of Sciences.



sequence. Persons with normal color vision occasionally make one single-place error, but they do not make more than one. The occurrence of two single-place errors, as in the sequence 1, 2, 4, 3, 5, 6, 8, 7, 9, 10 through 15, may not suffice to diagnose the type of color defect, but it is certainly not normal. The sequence 1, 2, 5, 3, 4, 6, 7 through 15 contains a two-place error, which indicates a tritan defect. Patients with normal color vision do not make two-place errors or errors greater than a two-place error.

A quantitative index similar to the total error score for the FM 100-Hue can be used to describe perform-

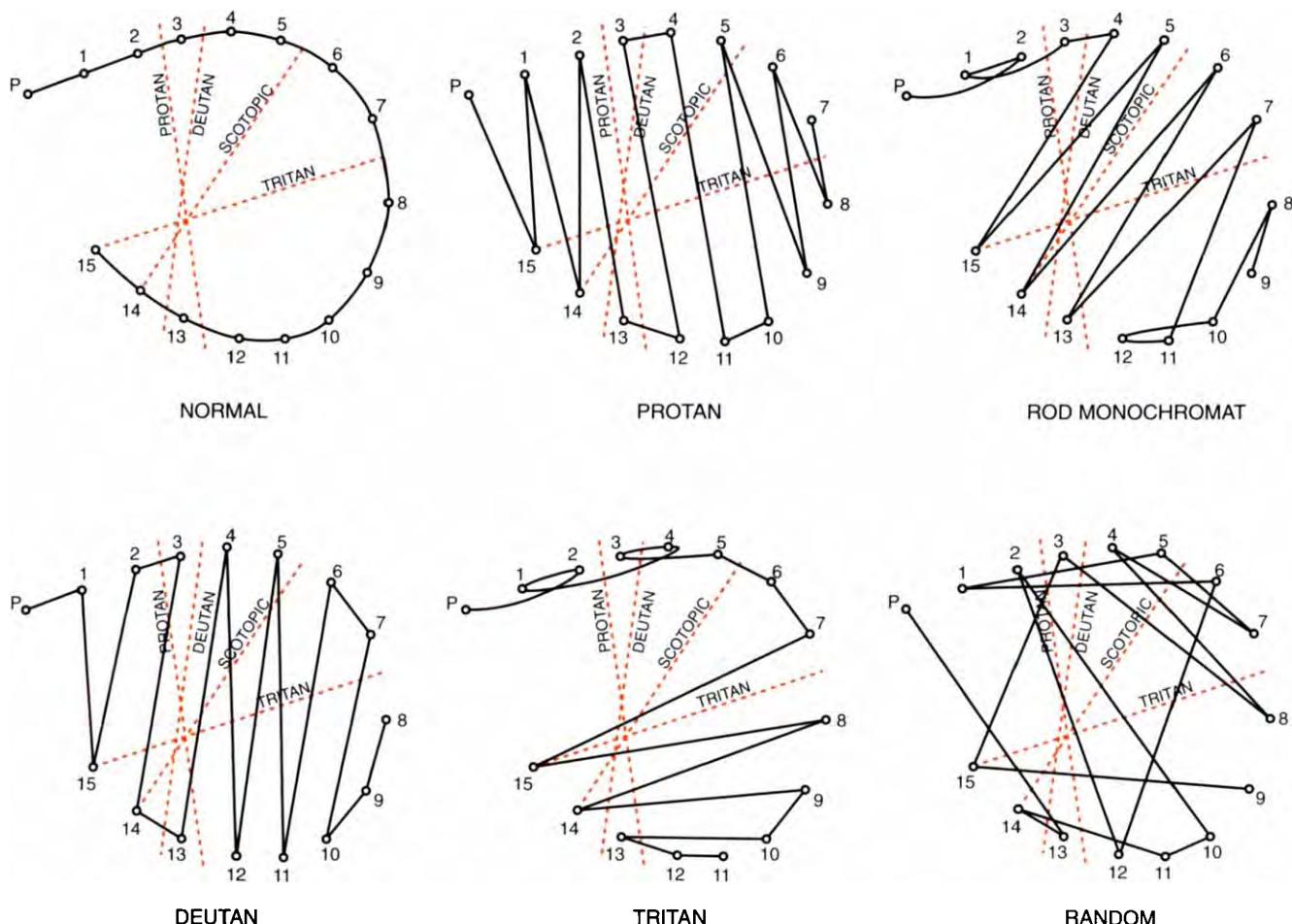


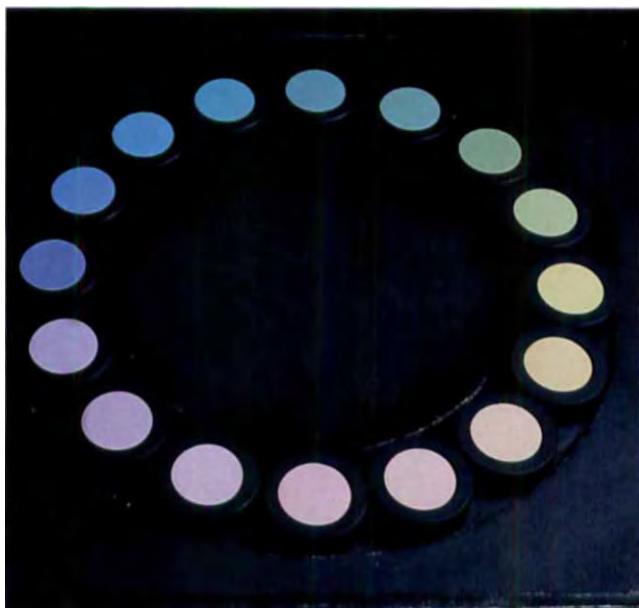
Figure 9-37

Results of Panel D-15 for a subject with normal color vision (no errors), and examples from a protan, a deutan, a tritan, and a typical rod monochromat. A random arrangement is also shown. (From Dain SJ. 1993. Characteristics of random arrangements of the Farnsworth Panel D-15 test. In Drum B [Ed], Colour Vision Deficiencies XI, p 322. Dordrecht, The Netherlands: Kluwer.)

ance on the Panel D-15. The total error score on the FM 100-Hue is straightforward to calculate using the numbers (1 through 85) printed on the inside of the caps. The total error score for the FM 100-Hue reflects the sum of the color differences between adjacent caps, because, when the caps are in proper sequence, each is separated by approximately the same number of JNDs. The caps in the Panel D-15 are not separated by an equal number of JNDs, and, hence, the color difference between any two adjacent caps needs to be computed. Bowman¹³¹ made these calculations on the basis of the CIELAB system, and, accordingly, a score for the test can be computed by looking up the color difference between any two adjacent caps and then summing the differences for all of the caps in the test. Vingrys and King-Smith¹³² devised another method, based on the CIELUV system. Quantitative scoring of the test results

is particularly useful for monitoring change in test performance for those suspected of having an acquired color defect, and it can be used to grade the severity of the defect.^{133,134} Color differences based on CIELAB for both the Panel D-15 and the Lanthony Desaturated Panel-15 Test are provided in Appendix 9-3.

A Large Panel D-15, in which the cap subtends about 4 degrees (or more) instead of 1.5 degrees as is usual, has been constructed (Figure 9-38). The Large Panel D-15 is useful with patients who have low vision (see Chapter 36) and also for the assessment of the degree of functional impairment. It is known that some individuals who are dichromatic with small fields (<2 degrees) may be trichromatic with larger fields.^{135,136} Results of both sizes of the D-15s may be useful with persons with acquired color defects, who may show errors on the small D-15 that are apparent only on the

**Figure 9-38**

A large version of the Panel D-15 test. Each cap in this homemade test has a diameter of 5 cm (2 inches) and a 6.4-mm (1/4-inch) annular border to the colored paper. The pilot cap is shown at the 9:00 position. All of the caps are moveable, and they are arranged in a circular channel or indentation.

large test at a subsequent testing session as a consequence of the progression of the condition. Results on both the regular and Large Panel D-15s from a typical rod monochromat and a patient with inherited deutanomaly are shown in Figure 9-39. Notice that, for the rod monochromat, the diagnosis of the type of color defect is more certain with the large test than with the regular-sized test, on which the results appear to be random. For the patient with a deutanomaly, the color defect is apparent on the regular-sized test but not on the large version.

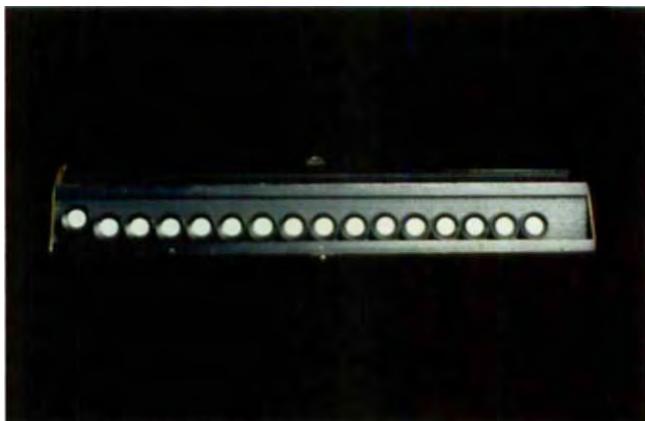
Lanthony Desaturated Panel-15 Test

The Lanthony Desaturated Panel-15 Test is similar to the Panel D-15, except the colors are three units higher in Munsell value and two units lower in Munsell chroma.¹³⁷ The colorimetric specifications are given in Table 9-5. A.J. Adams designed another desaturated D-15 panel that is like the Lanthony test, but the Munsell value of the colors is the same as the standard Panel D-15.¹³⁸ These tests are administered and scored in the same fashion as the Panel D-15. Patients with normal color vision may make minor errors, such as two single-place errors or a two-place error, which constitutes a failure on the Panel D-15 but not the Lanthony test. A failure occurs when the scored results show one or more lines across the circle. This test is more sensitive to mild

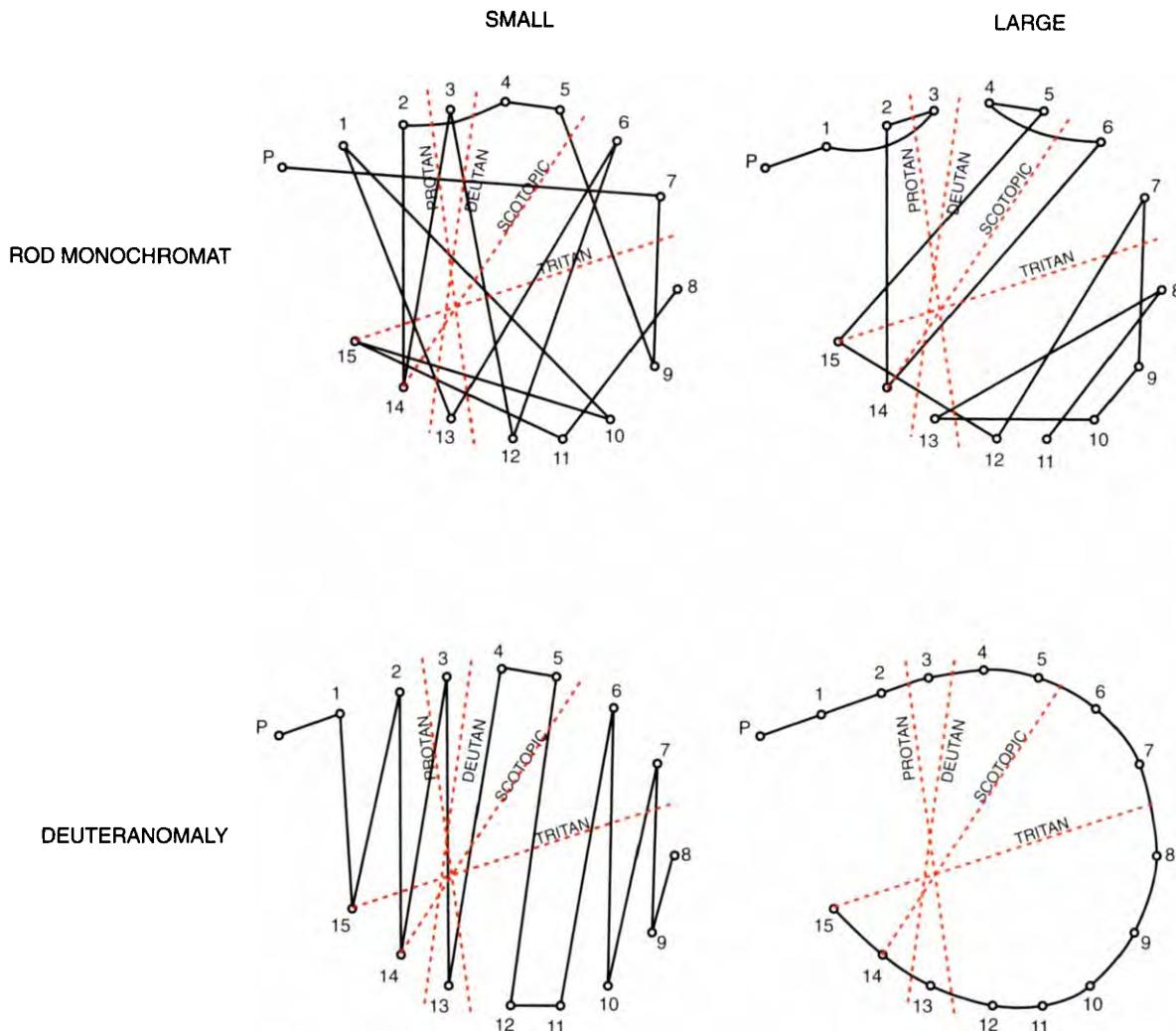
TABLE 9-5 Colorimetric Specifications for the Desaturated Panel D-15

Cap no.	CIE CHROMATICITY COORDINATES		MUNSELL NOTATION	
	x	y	Hue	Value/ Chroma
Pilot	.292	.306	10.0 B	8/2
1	.291	.309	5.0 B	8/2
2	.290	.316	10.0 BG	8/2
3	.291	.323	5.0 BG	8/2
4	.295	.330	10.0 G	8/2
5	.301	.337	5.0 G	8/2
6	.313	.348	10.0 GY	8/2
7	.329	.355	5.0 GY	8/2
8	.340	.352	5.0 Y	8/2
9	.340	.343	10.0 YR	8/2
10	.334	.329	2.5 YR	8/2
11	.328	.322	7.5 R	8/2
12	.324	.317	2.5 R	8/2
13	.319	.312	5.0 RP	8/2
14	.316	.309	2.5 RP	8/2
15	.305	.302	5.0 P	8/2

CIE, Commission Internationale de l'Eclairage.



defects than is the Panel D-15, and it is of particular value with acquired color defects when used in conjunction with the Panel D-15 for monitoring change in the status of color vision. An individual may show errors on the Lanthony test that appear only at a later time, as the condition progresses, on the Panel D-15. For mild protans and deutans, the diagnosis of type of defect may be incorrect, because the slopes of the confusion lines for protans and deutans are nearly the same over the extent of colors used in this test. The Adams Desaturated D-15¹³⁰ is intermediate between the Lanthony and the Farnsworth Panel D-15; the Adams test has the same Munsell value as the Farnsworth D-15, but the Munsell chroma is 2 rather than 4. The Adams test is not

**Figure 9-39**

Comparison of the results of small and large Panel D-15 tests for a rod monochromat and a case of deuteranomaly. P, Pilot or reference cap.

commercially available, but it can be constructed from Munsell papers.

New Color Test

The New Color Test was designed specifically for acquired color defects, and it has become known as the Lanthony New Color Test.¹³⁹ The test uses the same format as the Panel D-15, but there are some important differences. For instance, there are four trays of 15 different caps each. The colors in each of the different trays have the same sequences of Munsell hue and value, but they differ in Munsell chroma (8, 6, 4, and 2). In addition, there are 10 gray or neutral caps that have Munsell values in the range of N4 to N8 in 0.5 steps. To administer the test, the clinician mixes together the caps from the high-chroma tray and the 10 gray caps, and he or she asks the patient to separate the caps that appear

colored from those that appear gray; this is referred to as the *separation phase*. The patient then arranges the gray caps from dark to light and the colored caps in order of color: this is the *classification phase*. Unlike the Panel D-15, there is no pilot or reference cap to identify the beginning of the hue circle. The procedure of separating and arranging according to lightness and color is repeated for each of the other trays. A score form permits an estimate of the size of the neutral zone, which is determined by the range of hues confused with gray. The clinician shows the order of the caps at each chroma level by recording the cap sequence in a manner similar to that for the Panel D-15.

H-16

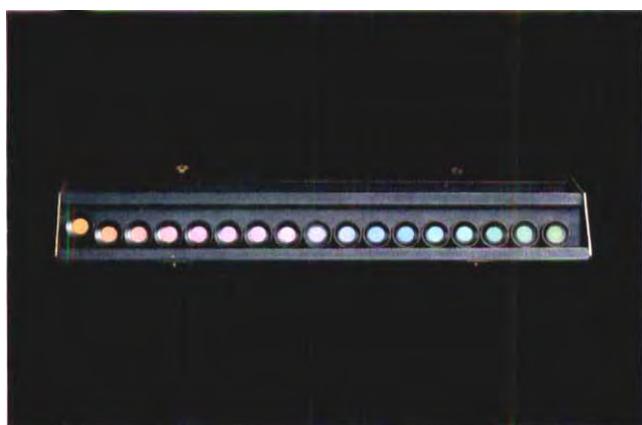
The H-16 was designed by Farnsworth and consists of 17 colored samples (Table 9-6). It is similar to the Panel

TABLE 9-6 Colorimetric Specifications for the H-16

Cap no.	CIE CHROMATICITY COORDINATES		MUNSELL NOTATION	
	x	y	Hue	Value/ Chroma
Pilot	.504	.383	2.5 YR	5/10
1	.496	.341	7.5 R	5/10
2	.469	.318	5.0 R	5/8
3	.431	.304	2.5 R	5/8
4	.398	.288	7.5 RP	~5/8
5	.365	.268	5.0 RP	~5/8
6	.334	.246	10.0 P	5/8
7	.313	.225	7.5 P	5/8
8	.275	.228	2.5 P	5/6
9	.230	.236	5.0 PB	5/8
10	.211	.263	5.0 B	5/6
11	.217	.289	10.0 BG	5/6
12	.230	.327	5.0 BG	5/6
13	.238	.364	10.0 G	5/6
14	.261	.391	5.0 G	5/6
15	.289	.421	2.5 G	5/6
16	.316	.454	10.0 GY	5/6

CIE, Commission Internationale de l'Eclairage.

From Paulson HM. 1973. Comparison of color vision tests used by the armed forces. In Color Vision, p 62. Washington: National Academy of Sciences.



D-15, but it uses colors that are different, most notably because of their higher Munsell chroma. The cap sequence for this test is recorded on the score form shown in Figure 9-40, and lines are drawn to indicate the numerical sequence of the cap arrangement. The direction of the lines is used to diagnose the type of defect, either protan or deutan. The test is useful for identifying dichromats, who invariably make three or more crossings, excluding the sequence for caps 7, 8, 9,

and 10. This test is not commercially available, but it can be easily constructed. The specifications for the colors are given in Table 9-6.

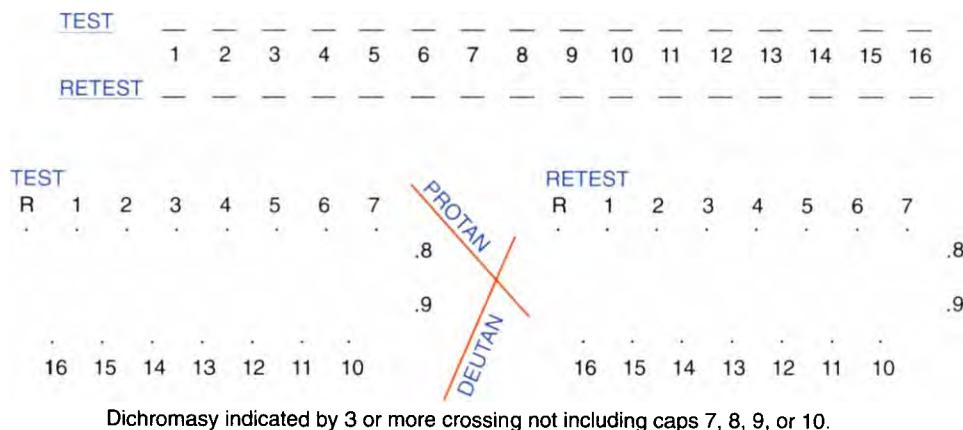
Anomaloscopes

Anomaloscopes are instruments that assess the ability of an individual to make metameristic matches. The results are used for the definitive diagnoses and quantitative assessment of color vision status. The first anomaloscope was designed by Nagel, and it is based on the color match known as the *Rayleigh equation*: $R + G \equiv Y$. Anomaloscopes are much more difficult to administer than the PIC tests and the arrangement tests, and, because of their relatively high price, they are rarely used in private practice.

Nagel (Model I) Anomaloscope

The Nagel anomaloscope (Figure 9-41) remains the standard instrument for the classification of each of the four types of red-green defects: protanomaly, deutanomaly, protanopia, and deutanopia. There are small differences in the wavelengths for the stimuli from one instrument to the next. On the instrument in our laboratory, monochromatic yellow (589 nm) light is presented in half of a 2-degree circular field, and a mixture of monochromatic (664 nm) red and green (549 nm) light is presented in the other half. Two knobs, which are indexed with arbitrary numerical scales, are used to obtain a match: one knob changes the brightness of the yellow, and the other changes the mixture of red and green. Pure red is indicated by a scale reading of 73, and pure green is shown by a scale reading of zero. Because luminance of the mixture field remains fairly constant as the mixture is changed, the brightness is nearly constant for patients with normal color vision and for deutans but not for protans. The observer views the matching field monocularly through an adjustable-focus, telescopic eyepiece that, through its focusing range, changes the size of the field to a small extent. Beneath the eyepiece, there is a large (9 cm in diameter) white adapting field—the Trendelenburg screen—for preadaptation to a neutral field. This is an important step before making the color match, because matching points may become unstable with the continuous viewing of the colors.

Useful guidelines for administration and interpretation of the results have been published.^{1,140} The midpoint and the matching range are determined, and the size of the matching range and the position of the midpoint determine the diagnosis. For people with normal color vision, the matching range is usually very small (typically not more than 3 or 4 Nagel scale units). As compared with the observer with normal color vision, the protanomalous observer requires more red, and the deutanomalous observer requires more green. Simple

**Figure 9-40**

Score form for the H-16 test. R, Reference cap.

**Figure 9-41**

The Nagel anomaloscope.

protanomalous and deuteranomalous individuals have small matching ranges, and their means are considerably higher or lower than the normal. Individuals with large matching ranges, in which the midpoint may be near that for persons with normal color vision, are categorized as extreme protanomalous and extreme deuteranomalous.²¹ Because dichromats have no hue discrimination for wavelengths longer than about 540 nm, they accept any red and green mixture to match yellow; they are differentiated on the basis of a brightness match of the yellow to pure red and pure green. This is a relatively simple task for the dichromat, who is unable to discriminate any difference in hue for green, yellow, and red. The protanope can be distinguished from the deutanope by the low luminance of the yellow to match pure red and the comparatively high luminance of the yellow when matching pure green; this occurs because of the "shortening of the red" that characterizes protanopes. Deutanopes perceive the spectrum as having about the same brightness that persons

with normal color vision perceive and, accordingly, their brightness match of pure red and pure green is comparable to that made by persons with normal color vision.

An *anomaly quotient* is used for quantitative comparisons. The anomaly quotient is the number obtained by dividing the R/G ratio for any individual by the R/G ratio for persons with normal color vision,⁵⁰ as shown below.

Equation 9-15

$$(R/G)_x / (R/G)_N \quad \text{or} \quad (R/G)_x \times (G/R)_N$$

where X is the value for a given individual and N is the normal value. The anomaly quotient for persons with normal color vision will then have a value of 1. The usual interpretation is that an anomaly quotient greater than about 1.33 indicates protanomaly and a quotient less than 1/1.33 indicates deuteranomaly. The following example illustrates calculation of the anomaly quotient. The value of the normal R + G setting varies from one instrument to the next; it is 43 on the instrument that our laboratory uses. Let 43 represent the amount of red; the amount of green is then 30 ($73 - 43 = 30$). If an observer obtained the same values as those for persons with normal color vision, the anomaly quotient would be 1, as follows:

Equation 9-16

$$(R/G)_x / (R/G)_N \quad \text{or} \quad 43/30 \times 30/43 = 1$$

If an individual had a match point at 50, the anomaly quotient would have a value of 1.52, as is the case with protanomaly:

Equation 9-17

$$(R/G)_x / (R/G)_N \quad \text{or} \quad 50/23 \times 30/43 = 1.52$$

Pickford–Nicolson Anomaloscope

The Pickford–Nicolson anomaloscope can be used for three different matches or colorimetric equations:

1. the Rayleigh equation [$R + G \equiv Y$],
2. the Engelking equation [$B + G \equiv CY$], and
3. the Pickford–Lakowski equation [$B + Y \equiv W$].

The matching field is presented on a screen for free viewing at a variety of distances, and there are no intervening optics between the patient and the matching field.¹⁴¹ The size of the field is changed by selecting different apertures: the largest is 2.54 cm (1 inch) in diameter, and the smallest is 0.48 cm ($\frac{1}{16}$ inch). Different colors are obtained by inserting broadband filters. The Pickford–Lakowski equation is used to assess the consequence of senescent changes in the spectral transmission of the ocular media (yellowing of the lens), but it also has value for the examination of acquired color defects. The Engelking equation is used for the diagnosis of the blue-yellow, or tritan, color defects. Individual variability in the density of the macular pigment and lens pigmentation affects both the Engelking and the Pickford–Lakowski equations and, accordingly, it confounds the interpretation of an individual result. Norms for a relatively small group of patients for each of the three equations used on the Pickford–Nicolson anomaloscope have been published by Lakowski.¹⁴²

Other Tests

City University Colour Vision Test

The City University Colour Vision Test is a matching test that comes in a book that consists of 10 test plates and 1 demonstration plate.¹⁴³ Each plate consists of a color disc surrounded by four discs located in each of the four cardinal directions of the compass. The observer's task is to select the one of the four colored discs that is most like the center. For each plate, there is a normal response and a response for each of the major color defects: protan, deutan, and tritan. The colors are similar to those on the Panel D-15.^{144,145} Whereas the results of the test usually permit a correct classification of dichromats, anomalous trichromats are not always correctly classified, with many who fail the Panel D-15 making no errors on the City test. Nonetheless, the test is useful for those who lack the manual dexterity to perform the Panel D-15. The difference between the results of the City and the Panel D-15 is likely due to the different tasks: forced-choice matching is used with the City, whereas serial ordering of colors is used on the Panel D-15.

The Sloan Achromatopsia Test

The Sloan Achromatopsia Test (Figure 9-42) is a matching test designed for rod monochromats.¹⁴⁶ The test consists of seven plates, each with a different color: gray, red, yellow-red, yellow, green, purple-blue, and

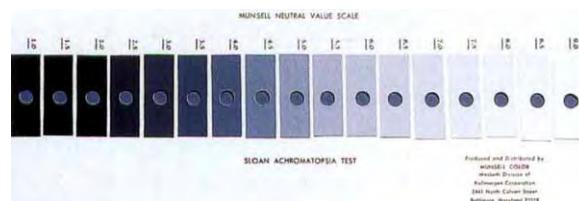


Figure 9-42

The Sloan Achromatopsia Test.

red-purple. Each plate includes 17 rectangular strips forming a gray scale from dark to light in 0.5 steps of Munsell value. In the center of each rectangle is a colored disc that has the same Munsell value from one end of the gray scale to the other. The patient's task is to identify the rectangle that matches the lightness of the colored disc. This is a difficult task for persons with normal color vision because of the color difference, but it is readily and precisely accomplished by complete achromats, who see the colors as grays of different lightness. There are normative data for both persons with normal color vision and achromats.

Titmus Color Perception Test

The Titmus Color Perception Test is included as one slide in the Titmus Vision Screener. The slide is a photographic reproduction of six plates from the Ishihara. The Titmus Vision Screener is a table-mounted stereoscopic instrument with a light source that transilluminates the slide and provides the patient with either a monocular or binocular view of the test slides. One error on any of the six plates on the color slide constitutes a failure. This test is not a good screening test because it has a fairly high false-positive rate and, as a result of exposure from the lamp in the instrument, the colors on the plate are not stable over time. The test is, however, frequently used in occupational settings.

Occupational Tests

Color vision requirements exist for many occupations, and there are some excellent resources about the topic.^{147,148} Color vision requirements may affect employment in a variety of ways: they may exclude people with any type of color vision defect; they may allow for the hiring of individuals who have a color defect, provided that they can adequately perform certain tasks or pass certain tests; or they may require normal color vision with a very high ability to make fine color discrimination (Box 9-3).

The color vision tests that are used for occupational testing are often the same as those commonly used in the clinical setting, such as the PIC tests, most often the Ishihara, and arrangement tests such as the Panel D-15 and the FM 100-Hue. There are, however, occupational

Box 9-3 Occupational Aspects of Color Vision
Occupations that Require Normal Color Vision

Color matcher in textile, garment, paint, and other industries requiring exact color matching
Auto body painter (spraying and retouching)
Restorer of paintings and works of art

Occupations that Have a Color Standard But May Admit Those with Mild Color Vision Defects

Armed forces
Aviation (pilot and air traffic controller)
Electrical and telecommunications trades
Maritime
Commercial driving (truck, taxi, and bus)
Railroad

Occupations in Which Normal Color Vision Is Desirable But the More Severe Forms of Color Vision Defect May Be a Limitation

Architect
Biologist
Botanist
Butcher
Farmer
Florist
Forester
Furrier
Gardener
Geologist
Gemologist
Graphic artist
Interior designer
Jeweler
Meat inspector
Medical sciences and health care (anatomist, anesthetist, dentist, physician, bacteriologist, microbiologist, nurse, nutritionist, optometrist, ophthalmologist, pathologist, pharmacist, physiotherapist, prosthodontist, surgeon, veterinarian, zoologist)
Metallurgist
Paint industry
Photographer
Tailor
Theater, film, and television

color vision tests that are not ordinarily used in a clinical setting, such as the Farnsworth Lantern Test (FALANT), the ISCC Color-Matching Aptitude Test, and the Davidson and Hemmendinger (D & H) color rule. There are also field tests, in which the task may be identical or nearly identical to that which the individual would have to perform on the job. Sometimes a test is administered in such a way to approximate the actual

viewing situation. For example, pilots may be required to name the color of a colored-light gun signal from the tower while standing on the ground instead of being seated in the cockpit of an airplane and flying by the tower.

When a color vision test is required for a particular occupation, there may be precise guidelines that specify the particular color vision test that must be administered, that exclude certain tests, and that specify the conditions for administering the test. Sometimes only the general form of a test is specified, such as the requirement that a PIC test be administered. Some requirements are precise, but no standardized test is readily available. For example, to obtain an interstate truck driver's license in the United States, a person must be able to identify the traffic signals red, amber, and green, but there is no standard test for assessing this ability. Although the Dvorine Nomenclature Test does not use traffic signal colors, results from this test can be used to document that an individual can correctly name colors.

The Davidson and Hemmendinger Color Rule

The D & H color rule was designed for use in industrial colorimetric applications to evaluate the effects of different light sources on color matching and to determine individual differences in color matching. It is a good device for demonstrating the effects of different light sources on metamerism. The rule is like a mathematical slide rule with two movable slides, each painted with hues of nearly constant lightness. One slide is a gradient from green through gray to purple, and the other is a gradient from blue through gray to orange. A portion of each slide is visible through a rectangular aperture (3.2 cm × 3.5 cm). The slides are adjusted until a color match is achieved, and the results are recorded by noting the positions of the two slides, which are indexed on the reverse side. Small changes in color temperature cause changes in the match point, and it is in this way that illuminants are evaluated. The rule can also be used to examine individual differences in color matching. Results from color-deficient subjects show that all of the types of red-green inherited color defects can be differentiated, although the testing routine is fairly long.¹⁴⁹ Tritan defects associated with ocular hypertension and glaucoma affect the matching range on the D & H rule, although, again, testing is lengthy.¹⁵⁰

Lantern Tests

A few different types of lantern tests are in use today. The FALANT is used in the United States by maritime and aviation authorities; the Holmes Wright Type A is used in the United Kingdom by aviation authorities; and the Holmes Wright Type B is used in Australia, the United Kingdom, and other Commonwealth countries by maritime authorities. The Edridge-Green Lantern is included in United States Coast Guard requirements,

but it is surpassed by the FALANT. Because there is no standard procedure for administering the Edridge-Green Lantern, it is not recommended.¹⁵¹ The Williams Lantern test is no longer available; although it is still listed in Coast Guard requirements, it is also surpassed by the FALANT.

The FALANT is required for occupations in the aviation, military, and maritime fields. Instructions for administering the test are printed on a panel attached to the lantern (Figure 9-43). It is administered in a normally lighted room with the examinee positioned at 8



Figure 9-43

The Farnsworth Lantern Test.

feet from the lantern. The instructions are as follows: "The lights you will see in this lantern are either red, green or white. They look like signal lights at a distance. Two lights are presented at a time in any combination. Call out the colors as soon as you see them, naming the color at the top and then the color at the bottom. Remember, only three colors—red, green and white—and top first." Nine pairs of lights are presented, with each pair exposed for 2 seconds. The test is passed if there are no errors during the first administration. If any errors occur during the first administration, two more complete administrations are given. The test is failed if the average of the errors on the last two administrations is greater than 1. An error consists of misnaming one or both of the lights; the score form allows for a record of the responses to all 9 pairs (Figure 9-44).

Arrangement Tests

The arrangement tests that are used in occupational settings are the FM 100-Hue and the ISCC Color-Matching Aptitude Test. Both are used for quantitatively grading color discrimination. The Color-Matching Aptitude Test requires a much finer level of color discrimination than the FM 100-Hue. The Color-Matching Aptitude Test was developed during the early 1940s.^{152,153} Although it is now out of stock, a modification is under development. This matching test consists of 48 chips of four colors (red, yellow, green, and blue) that vary in saturation. The chips are made of acrylic plastic, with fairly permanent pigments. Twelve chips of each of the four colors are displayed in different rows on an easel. The examinee selects one chip at a time from a box that contains 48 loose chips; he or she matches the chip to one of the 48 chips on the easel and then identifies its position by writing the number of the chip on the score form that is located on the easel. The loose chip is returned to the box before the next chip is selected. Instructions for scoring and interpreting scores are provided. The results are quantified into one of five categories of color-

	1 GR	2 WG	3 GW	4 GG	5 RG	6 WR	7 WW	8 RW	9 RR	# of errors per run
1st run										
2nd run										
3rd run										

Figure 9-44

Score form for the Farnsworth Lantern Test. G, Green; R, red; W, white.

matching ability: poor, fair, average, good, or excellent. The test is not timed, although time can be used as a factor for assessing ability. The test ordinarily takes 40 to 60 minutes to complete. Patience when completing the test is essential. The color differences are very subtle so that, according to the instructions, the person with "average" color discrimination is "expected to choose one of several colors as a match for a given chip."

TEST ADMINISTRATION

The administration of color vision tests is fairly straightforward. The most frequently used plate and arrangement tests have requirements that pertain to lighting, viewing time, and test distance. Tests should be administered monocularly unless there is an occupational requirement for binocular testing or the test is part of a screening, in which case binocular testing might be used in the interest of saving time. Patients should wear their refractive correction, and there should be no tint in the glasses or contact lenses. Testing should be performed before the use of diagnostic drugs and not immediately after the use of instruments having bright light sources (e.g., an ophthalmoscope, a slit lamp). Although it is customary to adhere to the conditions specified for each test,

there are instances in which a departure from standard procedure may reveal a color defect that would otherwise go undetected. The test results of those who have an acquired color defect are often more dramatically affected by a change in testing conditions than the results of those who have an inherited defect. This difference in test performance can be used to facilitate the diagnosis of an acquired color defect, as the results in Figure 9-45 illustrate. This patient had chorioretinal degeneration and made only one single-place error on the Panel D-15 under standard conditions; however, when the light level was reduced, there were many errors that clearly indicated a tritan defect. The same reduction in light level had no effect on the performance of a patient with normal color vision. For another case, increasing the light level for the Lanthony test with a 1000-lux halogen lamp revealed early glaucomatous changes in color vision.¹⁵⁴ Changes in test performance will not necessarily occur with all tests, because some—notably the Ishihara—are rather impervious to alterations in test conditions.¹⁵⁵

Lighting

Most of the PIC tests and the arrangement tests were standardized for illumination with a source having the characteristics of CIE standard illuminant C or natural

TEST	1	2	3	4	5	6	7	8	9	10	12	11	13	14	15
1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	
RETEST	4	3	5	2	1	6	7	15	8	14	13	12	10	11	9

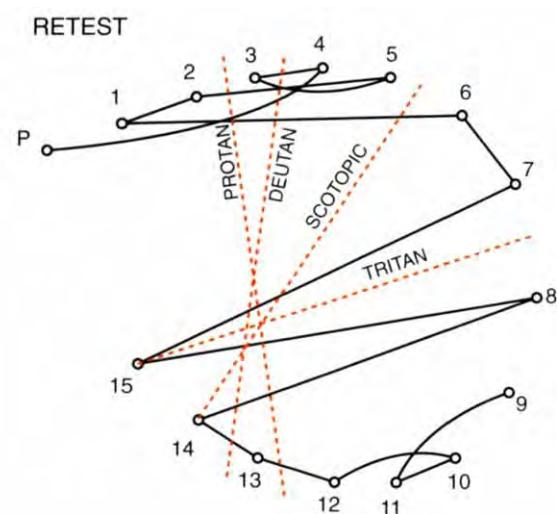
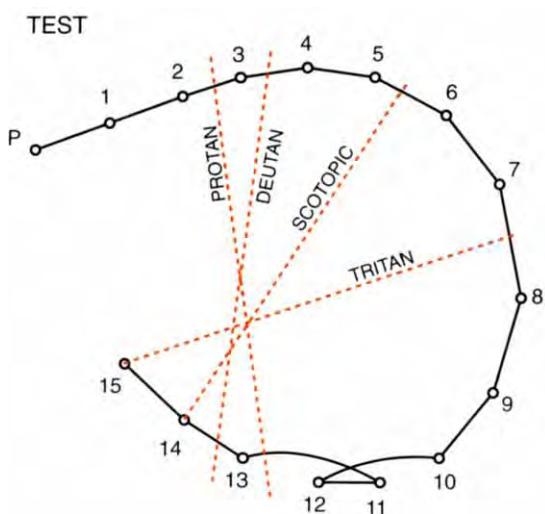


Figure 9-45

Results of the Panel D-15 test for a person with choroideremia. The result on the left was obtained under standard conditions of illumination; on the right, the patient was wearing glasses that had a luminous transmittance of 7%. Results from a person with normal color vision are the same under both conditions of viewing. P, Pilot or reference cap. (From Verriest G. Further studies on acquired deficiency of color discrimination. J Opt Soc Am 53:195.)

daylight. Lighting with natural daylight is not recommended, because both the amount and spectral quality are too variable. Although the illuminance for these tests is not always specified, it should be in the range of 100 to 650 lux. For many years, the Macbeth easel lamp was the lamp of choice, but it is no longer commercially available. The Macbeth uses a 100-watt clear, incandescent bulb that is covered with a blue glass filter to achieve the right color temperature. Except as noted below, most fluorescent lamps are not good replacements for the Macbeth. Although some fluorescent lamps may have the right color temperature, most do not have the correct *color rendering index* (CRI). The CRI is a number (from 0 to 100) that expresses the effect of a light source on the color appearance of objects as compared with the standard, which, for color vision tests, is standard illuminant C. The CRI for any lamp used for color testing should be greater than 90. Certain Kodak Wratten gelatin filters meet this requirement when used in conjunction with incandescent sources: a single Kodak Wratten #78AA¹⁵⁶ and a sandwich of two filters, #78B and #80B.¹⁵⁷ The filters are placed before the patient's eye, because the gelatin would melt if placed close to the lamp. Unfortunately, Kodak has discontinued some of the Wratten color filters, although equivalent glass filters are available (Fish-Schurman, New Rochelle, NY). Unfiltered incandescent lamps are not

recommended, because they frequently allow persons with mild color defect to pass PIC tests.

An alternative to the Macbeth easel for clinical testing is the True Daylight Illuminator (Richmond Products, Boca Raton, FL), which has been evaluated with the Ishihara test.¹⁵⁸ The True Daylight Illuminator (Figure 9-46) consists of a platform for resting plate tests and two VERILUX Full Spectrum lamps (Model F15T8/VLX). These lamps could also be placed in a commonly available fluorescent desk lamp with a white reflector. Other alternatives are the Philips TL40W/55,¹⁵⁹ and, from a recent evaluation of lamps for the Panel D-15, the GE Chroma 75 and two lamps from Duro-Test, the Color Classer 75 and the 34 watt Vita-Lite Plus.¹⁶⁰ It is good practice to specify the lighting conditions in any report that includes the results of color testing.

The test, the lamp, and the patient should be arranged appropriately to avoid glare and visible surface reflections. Interestingly, some rod monochromats have mistakenly thought that they could see color because they were able to read the numerals on the Ishihara by holding the plates at the proper angle to detect a difference in the specular reflections of the inks.¹⁶¹ Plate tests are usually administered at about 0.75 m and arrangement tests at 0.50 m. The patient is ordinarily seated at a table, which can be covered with gray cardboard to avoid high lightness contrast between the test

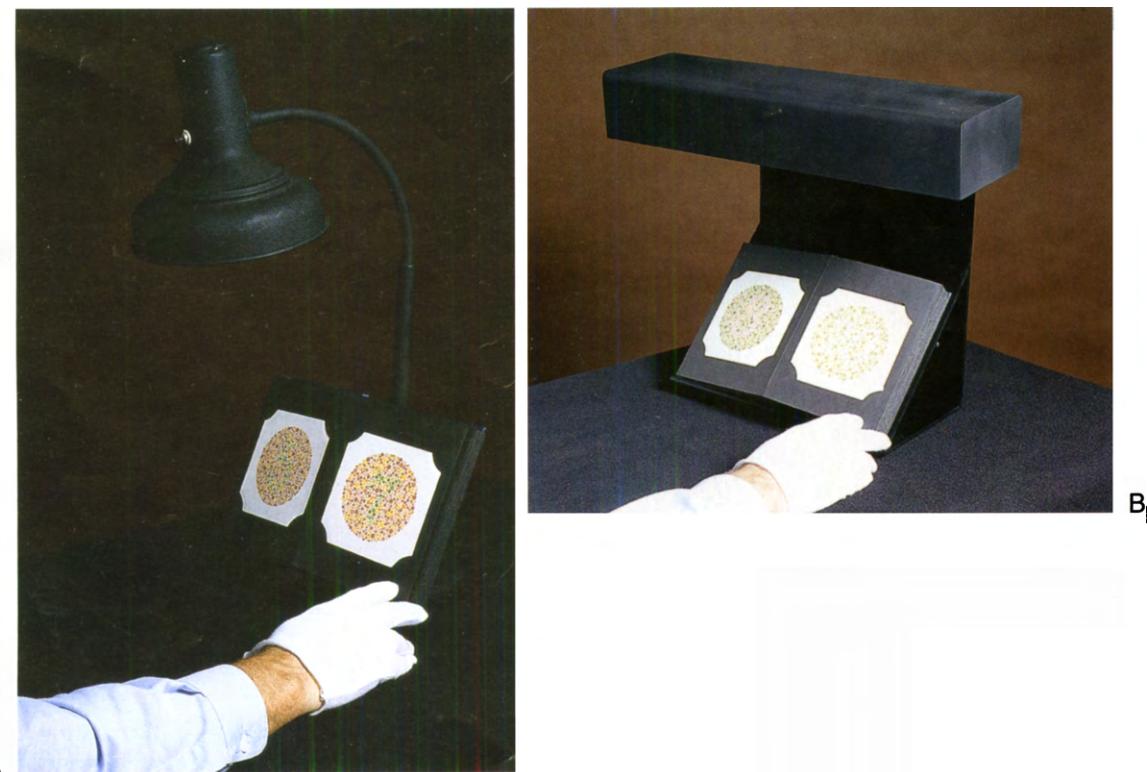


Figure 9-46

A, The Macbeth easel lamp. B, The True Daylight Illuminator.

and the table. All of the tests should be handled carefully to avoid soiling with fingerprints, and they should be kept in the dark when not in use to avoid fading. When a patient traces a figure on a plate, it is best done with a white cotton glove, a soft stylus (e.g., a camel hair brush), or, for kids, a soft finger puppet.

Acuity

Poor visual acuity and blur may affect test results. Frequently, there are concomitant changes in acuity with acquired color defects, and these may be reflected in the results of color tests. The effect of blur on performance on PIC tests has been studied with persons with normal color vision. The results show that up to 4.00 D blur induced with spherical plus lenses (corresponding with a visual acuity of about 20/300 [6/90]) has virtually no effect on performance on the Ishihara, but it has a significant effect on performance on the AOC plates and a somewhat smaller effect on performance on the Dvorine test.⁹² Although the effect of blur on color-deficient persons' performance on PIC tests has not been thoroughly studied, results with color-deficient persons may be different from those of persons with normal color vision.¹⁶² There is a significant effect on the results of the FM 100-Hue when near acuity is J20 or 20/300 (6/90) or worse, a finding that was again obtained with persons with normal color vision who were blurred with plus lenses.¹⁶³ Lanthony¹³⁹ reported that the New Color Test can be used even with acuity at 20/400 (6/120). A conservative conclusion is that test results can certainly be interpreted to be free of the effects of poor acuity or blur if acuity is 20/100 (6/30) or better and that they are probably unaffected if acuity is better than 20/200 (6/60).

Tints

Spectacle and contact lens tints can distort test results and cause a patient with normal color vision to fail a test or a person with a color defect to pass a test (see Chapter 25). Color tests should be administered without a tint, and the record should include information about whether or not a tint was worn. Some of the light contact lens tints ("visitints") have no effect on the Panel D-15 and FM 100-Hue,¹⁶⁴ but they may affect the results of certain plate tests. Light tints and some nearly neutral spectacle tints can distort results from color tests having narrow-band spectral sources, such as the Nagel anomaloscope.

Recommended Test Battery for Color Testing

It is wise to have more than one test available. How many tests should be administered depends on whether the intent is screening or the diagnosis of a specific type

of color defect. For a routine eye examination, a minimum of three tests is recommended for achievement of a definitive diagnosis:

1. The Ishihara or other PIC test,
2. The Panel D-15, and
3. The Lanthony.

The Ishihara is included because it permits a distinction between normal and defective color vision, and it is a sensitive test for identifying mild red-green color defects. However, the Ishihara does not include plates for tritan color defects. The Panel D-15 is included because the results permit a diagnosis of the type of defect: protan, deutan, tritan, or rod monochromacy. Administration of the Panel D-15 to persons known to have a color defect allows for an assessment of severity, because those with a mild defect will pass and those with a moderate to strong defect will fail. The Lanthony test results are of particular importance when a tritan color defect is not severe enough to manifest itself on the Panel D-15. The Lanthony and the Panel D-15 are good companions for monitoring the change in color vision that often occurs with acquired color defects.

Assessment of the severity of a color defect is important for patient management, because this information allows the clinician to counsel those with an inherited color defect more effectively and to make judgments about the likelihood of a patient's passing an occupational test. Acquired color defects may either progress or regress, and this change will only be manifested with test results that are sensitive to different levels of severity. Making a qualitative diagnosis of the type of color defect—protan, deutan, or tritan—is also important for patient management.

A set of simple guidelines for the administration of the recommended battery follows. Start with the Ishihara, and have the patient occlude one eye with a paddle. Give the following instructions: "Read the numbers on the card as I turn the pages. Some cards don't have a number, so if you don't see a number, just tell me quickly, 'no number.' Do not touch the plates, and tell the patient not to touch them. Turn the pages so that each is presented no longer than about 3 to 4 seconds, and write down the response the patient gives to each plate on a score form. The Ishihara does not come with a score form, but one could easily be constructed (see Appendix 9-2). Recording the patient's response to each plate is important, because there may be a change in color vision from one testing session to another, with the possible result that, although the same number of errors are made, the errors are made on different plates. This change in performance would go undetected if one were to simply record the number of plates misread or the number of plates read correctly, as some practitioners do, writing, for example, 4/13 to indicate 4 correct out of 13 plates.

Administer the two arrangement tests in sequence, performing repeat testing as needed. Begin with the Panel D-15, and occlude one of the patient's eyes with an eye patch. Both the physician and the patient should wear a white cotton glove (available from art supply stores, camera shops, or photo catalogs). Spill the 15 moveable caps onto the table top (covered with gray cardboard), and mix them into a random assortment in front of the open tray (Figure 9-47). There is no need to follow the directions in the instruction booklet to place the moveable caps in the upper part of the opened tray; this takes more time and also makes it more difficult for the patient to grasp a cap, particularly if the patient is wearing a glove. The surface of the tray in the Panel D-15 is maintained at an angle that is about normal to the line of sight when the tray is placed correctly on the table. Give the patient the following instructions: "Find a cap from here [indicate the random array of the 15 moveable caps] that looks most

like this cap [indicate the reference or pilot cap], and place it next to the fixed cap in the tray. Then find the cap that looks most like the one you already selected, and repeat this until you have placed all of the caps in the tray." Administration of the Panel D-15 to the right eye can be followed by the administration of the Lanthony to the right eye. Retest if there are any caps out of order. Switch the eye patch, and begin testing of the other eye.

Other tests may be performed in addition to this minimum test battery to enhance the scope of a color vision assessment. Plate tests for tritan color defects, such as the Standard Pseudoisochromatic Plates Part 2, the AO-HRR, the Richmond HRR (Fourth Edition) and the F-2 are useful, although arrangement tests are generally considered superior for acquired blue-yellow color defects. The Dvorine Nomenclature Test permits the assessment of color-naming ability, and results can be easily interpreted when included in written reports that describe the consequence of having a color defect. The FM 100-Hue would provide a large increment in diagnostic ability. It is particularly effective with persons with acquired color defects, because the results are both quantitative and qualitative, and it is also useful for grading normal color vision for certain occupational applications. The H-16 can be used to establish a fairly certain differential diagnosis between anomalous trichromacy and dichromacy, but only for those individuals who have a protan or deutan color defect. The H-16 is inexpensive to construct as compared with the cost of an anomaloscope, which, of course, is of value for making definitive diagnoses.

Patient Selection

The following is a set of guidelines for selecting patients for color testing. This list is similar to that suggested by Adams and Haegerstrom-Portnoy.²

1. Test all children at an early age, preferably before they enter first grade. This is important, because color is used as an aid to learning.
2. Test all patients during their first office visit. This provides a baseline against which any future changes of an acquired nature can be compared. Patients with inherited defects are not immune to acquired color defects, and, hence, baseline data are essential.
3. Test all patients who have an unexplained reduction in visual acuity or low visual acuity (e.g., 20/25 [6/7.5] or worse).
4. Test any patient who reports a recent color disturbance or any difference in color vision between the eyes.
5. Test any patient who exhibits a sign of abnormality in the fundus or who gives you reason to suspect

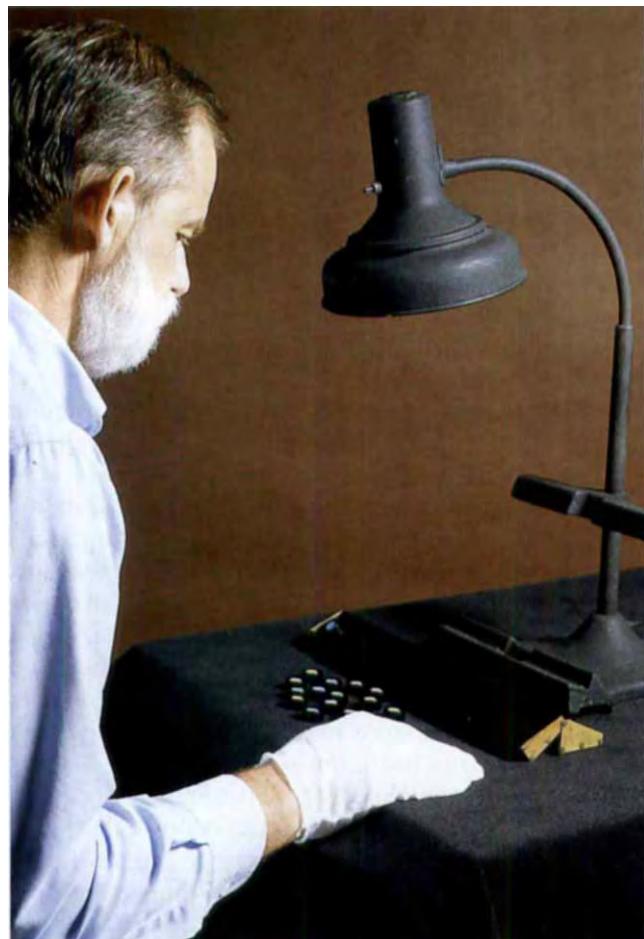


Figure 9-47

Administration of the Panel D-15 test under the Macbeth easel lamp.

an abnormality in the fundus, and also test any patient for whom another examination procedure suggests disease or who has a symptom that lacks an explanation.

Screening for Color Defects

Rapid screening for inherited red-green color defects can be accomplished with one of the plate tests. The most effective are the Ishihara, the AO-HRR screening plates, and the Dvorine. Screening for tritan defects requires the AO-HRR screening series (it is advisable to add the last four plates of the diagnostic series), the F-2 or a variant of it, or the PACT. Screening is often done binocularly; however, if time permits, monocular testing is preferable. Arrangement tests are usually too time-consuming to be included in a screening. As is the case for any screening regimen, keep in mind that some persons with a color defect will pass the screening (false negatives) and that some with normal color vision will fail it (false positive).

Occupational Testing

Occupational requirements often include one of the plate tests and, if that test is failed, a specialized test such as the FALANT. For some occupations, it is important to grade color discrimination; the two tests that are useful in this regard are the FM 100-Hue and the ISCC Color-Matching Aptitude Test.

Because the FALANT is rarely found in private practice, the results of clinical color vision tests can be used to predict performance on the FALANT. The findings of Cole and Vingrys¹⁶⁵ indicate that people with mild color defect were likely to pass the FALANT. One third of people with a color defect who failed a PIC test (Ishihara, AO-HRR, or Dvorine) passed the FALANT. The Panel D-15 was a good predictor of FALANT performance, more so for persons with color defects who failed the Panel D-15 than for those who passed. About 67% of persons with color defects who showed no errors on the Panel D-15 passed the FALANT, whereas 90% of those who failed the Panel D-15 failed the FALANT as well. All of the people with color defects who failed the H-16 (three or more diametrical crossings, excluding crossings for caps 7, 8, 9, and 10) failed the FALANT. About 50% of those with color defects who passed the H-16 failed the FALANT.

In the United States, the Federal Aviation Authority waives the color vision standard for those who fail the FALANT but who pass a supplementary field test. This test requires the recognition of the colors (red, green, and white) of the control tower signal gun. About 30% of those who fail the FALANT are likely to pass the signal gun test.

PATIENT MANAGEMENT

The quality of management of patients who have a color defect depends on whether the practitioner has the ability to communicate effectively with the patient, which requires careful listening to what the patient has to say. Listening well gives the practitioner who is a relative novice in the area of color testing the chance to understand what it is like to have a color defect. A careful listener has a much easier time when it is necessary to convey advice that the patient will understand. Some practitioners are under the false impression that there is nothing that can be done for the color-deficient person and so there is no reason to test. It is true that there is no cure for an inherited color defect, but there is a lot that can be done for those who have these defects, be they inherited or acquired.

Management of Patients with Inherited Red-Green Color Defects

Patients with inherited red-green color defects are managed with appropriate counseling. Some will request advice or assistance with an aid (i.e., a filter to improve color discrimination). For the most effective management, a differential diagnosis for protanomaly, deuteranomaly, protanopia, or deutanopia is the starting point for explaining to the patient the severity of the defect and the impact of the deficiency on color tasks. Because many people are not aware of their color deficiency, they are quite surprised when they fail a color vision test. Others may be vaguely aware that they have trouble with color, and some know that they must have a color defect but dispute that they are "color blind." Persons with defective color vision as well as those with normal color vision are naturally curious to know how their vision compares with that of others. They appreciate receiving a complete diagnosis and, when applicable, being informed of the likelihood of passing or failing occupational tests. Patients like to know how they inherited their defect, and sharing Figure 9-1 with patients is helpful during such a discussion.

The type of counseling provided depends on the needs of the patient and the type of color defect. Counseling relating to school, driving, occupational requirements, and the use of color filters is discussed in the following sections. In general, the young child and his or her parents should be advised, in most situations, to notify the teacher that the child has a color defect. Sometimes it is the child's teacher who first suspects a color defect. Parents typically welcome information about how color defects are inherited and are frequently surprised to learn their son inherited his defect from his mother. Advice about potential career limitations, driving, and recognition of color traffic signals is appropriate for adolescents

and adults. Some people with a color defect are interested in the use of color filters or the X-Chrom contact lens as an aid or "cure" for their deficiency.

Schooling

Because of the role color coding plays in instructional materials used during the early school years, the color-deficient student may find some tasks difficult and, as a consequence, may develop a dislike for school and learning.¹⁶⁶ Although it has been a rare occurrence, some children have been needlessly held back in school because of an undiagnosed color deficiency that kept them from making satisfactory progress. The color-deficient student may experience difficulty with some tasks in geography, chemistry, and biology. After the defect is diagnosed, the solution to most of the problems at school is to inform the parents and teachers that the child has a color defect and to identify the colors that are likely to be difficult for the child to distinguish. In some situations, it is inappropriate to expect the color-deficient child to succeed or to have it as easy as others. An example is a lesson in which students are required to identify countries on a map by coloring each with a different colored pencil (e.g., "Color Switzerland green, Belgium brown . . ."); for this task, the color-defective student might do well with crayons because they are labeled with their colors, whereas colored pencils often are not labeled. Teachers should be apprised of the potential color confusions and should consider alternative solutions. For example, the color-defective student could be asked to write the name of the country in the appropriate place on the map. Students will learn to cope with the defect and, with experience, learn when it is appropriate to rely less on color and more on other clues. These young children need to be assured that they need not be ashamed of any color mistakes about which other children might enjoy teasing them. Young peers are often harsh.

Driving

It is important for color-defective persons to know of their color deficiency and the limitations it may place on their driving ability. Some will take more time than

persons with normal color vision to respond to traffic signals and, to a certain extent, must rely on other clues, such as position.^{167,168} Protanopes and deutanopes will likely see a distant green traffic signal as white; should they confuse the green light with a street light or a storefront light, they may not be aware that they are approaching an intersection. A single flashing traffic signal—yellow or red—will pose a problem, because the usual position clue is missing; these individuals may then rely on what other drivers are doing, look for a stop sign, or slow down to allow for more time when making a decision. Ideally, they will not mistake a flashing yellow for a flashing red and inappropriately stop. Protans should be cautioned that their perception of the brightness of red lights may pose a limitation in driving. Cole and Brown¹⁶⁷ have shown that the intensity of the red signal should be increased about 4 times to make it as visible to the protan as the usual intensity is to others. A protan may follow cars closer than do people with normal color vision because of the reduced brightness of red lights; this problem may manifest itself in adverse driving conditions, such as driving in fog or at night on a country road behind a farm tractor, when clues about the presence of a vehicle other than the red taillights are absent or reduced. Practitioners should be aware that tinted contact lenses can significantly increase the reaction time for the detection of red signal lights among protanomals, protanopes, and deutanopes, which may increase their risk for an accident.¹⁶⁹

Occupational Aspects

The practitioner is required to process forms or write letters of evaluation for patients who must take a color vision test as part of an occupational requirement. Most agencies that have a color vision requirement supply a form that stipulates the test or tests to be administered and that provides the physician with space to record or comment on the results. In the absence of a form, a written narrative report may be submitted. Two sample reports follow. Each includes the information that is usually required: a description of the tests and test conditions, the results, and an interpretation of the results.

Sample 1

Color Vision Assessment: John Pease, age 22

On August 4, 2006, I administered a battery of color vision tests to John Pease, who has a career plan in law enforcement. The tests included the Ishihara (24-plate edition) and the AO-HRR pseudoisochromatic plates and the Farnsworth Dichotomous Test (Panel D-15), all of which were administered monocularly to each eye under standard conditions of illumination with the Macbeth easel lamp. In addition, I administered the

Dvorine Nomenclature Test and the Nagel anomaloscope.

The results of the Ishihara and AO-HRR plate tests indicate a red-green color deficiency that, on the basis of each of these tests, is a protan (red) deficiency of mild severity. There were no errors on the Panel D-15, which is designed to pass those with a mild color defect. Mr. Pease used incorrect color names for two of the eight saturated colors and two of the eight unsaturated (pastel) colors on

the Dvorine Nomenclature Test. The results obtained with the Nagel anomaloscope indicate that Mr. Pease has a protanomalous color deficiency, which is the less-severe form of the protan color deficiencies.

All of the test results indicate that Mr. Pease has a protan or red color deficiency of the same type and severity in each eye. This color defect is likely to be inherited and as such will not change with age. In my opinion, individuals like Mr. Pease who have a mild

color deficiency and pass the Panel D-15 should be able to adequately perform the color tasks required in law enforcement.

For your information, I have attached copies of the score forms for the Ishihara, the AO-HRR, the Panel D-15, and the Dvorine Nomenclature Test. Do not hesitate to contact me if you have any questions.

Sincerely,

Sample 2

Color Vision Assessment: Robert Babbit, age 47

On April 25, 2006, I administered a battery of color vision tests to Robert Babbit, who is in the process of applying for membership in the Seafarers International Union and obtaining a Coast Guard approval. The tests included the Ishihara (24-plate edition) and the AO-HRR pseudoisochromatic plates, the Farnsworth Dichotomous Test (Panel D-15), and the H-16, all of which were administered monocularly to each eye under standard conditions of illumination with the Macbeth easel lamp. In addition, I administered the Dvorine Nomenclature Test and the Farnsworth Lantern Test (FALANT).

The results of the Ishihara and AO-HRR plate tests indicate a red-green color deficiency that, on the basis of each of these tests, is a deutan (green) deficiency of mild severity. The Panel D-15 was passed, an expected result for those with a mild color deficiency. There were no errors on the H-16, which also indicates a mild color deficiency. Mr. Babbit used correct color names for all

eight saturated and all eight unsaturated (pastel) colors on the Dvorine Nomenclature Test. With the FALANT, there was one error on the first run, no errors on the second, and one error on the third. An additional run of the FALANT also showed one error.

All of the test results indicate that Mr. Babbit has a deutan or green color deficiency of the same type and severity in each eye. This color defect is likely to be inherited and as such will not change with age. Despite his color defect, Mr. Babbit used correct color names for all of the colors on the Dvorine Nomenclature Test, and he passed the FALANT and the Panel D-15. In my opinion, Mr. Babbit meets the requirements for an able-bodied seaman and, therefore, should also meet the requirements of the Seafarers International Union.

For your information, I have attached copies of the score forms for the Ishihara, the AO-HRR, the Panel D-15, the FALANT, and the Dvorine Nomenclature Test. Do not hesitate to contact me if you have any questions.

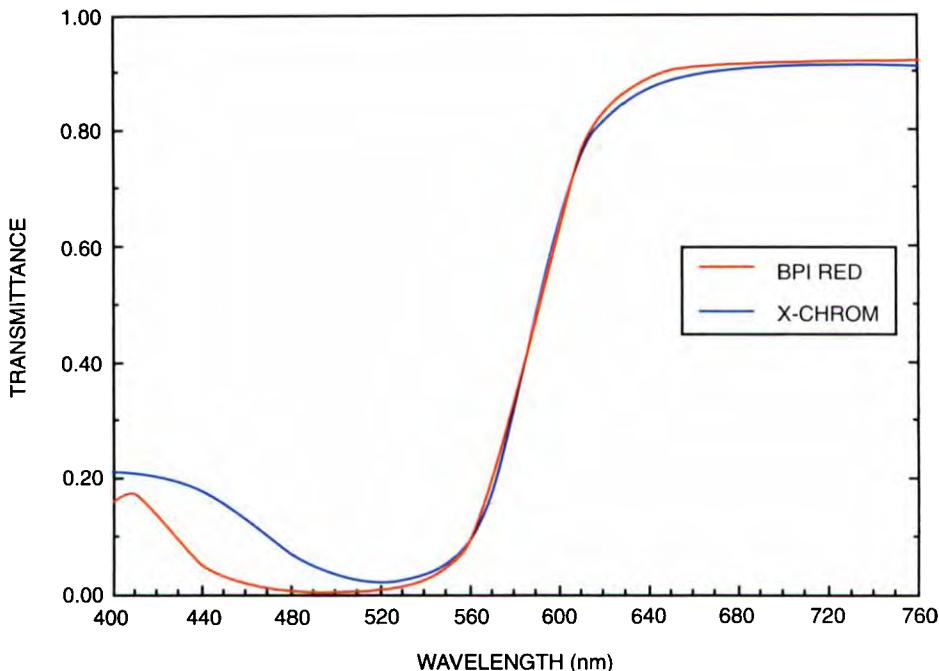
Sincerely,

The Use of Color Filters

Color filters can be used in a variety of ways to help people with a color defect recognize colors. Color filters work by changing the luminosity (lightness) or chromaticity of colors. The best-known filter is a red-tinted polymethylmethacrylate contact lens known as the X-Chrom lens.¹⁷⁰ Soft lenses are also available.^{171,172} Alternatively, a color filter can be handheld, or a dyed spectacle lens can be used. The contact lens is a better solution cosmetically than a tinted spectacle lens, but there are benefits to a spectacle tint when cosmesis is not important. The use of colored filters to help color-defective persons has been reviewed by Schmidt¹⁷³ and Fletcher and Voke.¹⁷⁴

Zeltzer¹⁷⁵ described the procedures for fitting the X-Chrom lens, and a manual of these procedures and case reports is available from X-Chrom Corporation

(Ipswich, MA). The X-Chrom is worn on one eye, usually the nonsighting eye. The lens has a high transmittance in the long wavelengths, absorbs the middle wavelengths around the neutral point (ca. 500 nm) of the red-green dichromats, and transmits some of the short wavelengths. The spectral transmission curve is shown in Figure 9-48, along with BPI Red (BPI North America, Miami, FL), which can be used for tinting spectacle lenses. The depth of the tint is decided empirically: it should be dense enough to allow a benefit but not dense enough to cause suppression. The luminous transmittance of the X-Chrom depends on the thickness of the lens and is usually in the range of 15% to 50%. The X-Chrom lens is effective for some—but not all—persons with red-green color defects. Some individuals do not benefit, because they suppress the eye with the tint. A trial with a handheld filter before one eye is the

**Figure 9-48**

Comparison of the spectral transmittances of the BPI Red and the X-Chrom contact lenses.

first step in determining whether there will be any benefit with the X-Chrom or any other filter. Most color-defective individuals who are unable to identify the numerals on the Ishihara plates will succeed when viewing them through a red filter. This is to be expected, because the Ishihara plates are composed with red and green colors. The numerals on the plates can be seen through the red filter because of a new color contrast of the figure and background; the red filter will appreciably darken the green but not the red. If the plates can be read monocularly through the filter but not binocularly when the filter is before one eye, the patient is suppressing.

A word of caution to the practitioner who wishes to use the Ishihara plates to show how a filter works: because there is often a dramatic improvement in performance on the Ishihara, patients naturally expect the same improvement in other tasks, and this may not always occur. Performance on the AO-HRR is only marginally changed with the X-Chrom. The Panel D-15 will generally be performed with fewer errors, but there will be little if any change in performance on the FM 100-Hue.¹⁷⁶ An explanation about why the filter works so well with the Ishihara but not with other tasks may fall on deaf ears if the patient previously could not see any number on the plates but reads all of them correctly with the red filter. In short, be careful not to create undue expectations about the magnitude of the potential benefit. Of course, the X-Chrom lens or any other tinted lens should not be used when administering

color vision tests as part of an occupational requirement. Use of the X-Chrom lens is prohibited by the Federal Aviation Administration for pilots and by the Coast Guard for those holding a Coast Guard License or Merchant Marine certification.

Filters that aid color discrimination do not restore normal color perception. The color-defective person will be able to correctly name some—but not all—colors with the filter. For other colors, correct naming may come with experience. Binocular luster (seeing one color through another) occurs when a person attempts to fuse dissimilar colors, and it is experienced by some individuals when a color filter is placed before one eye. This effect may provide a new clue to help people with color defects expand their world of color recognition.

Because the X-Chrom lens decreases the amount of light to one eye, there is the potential for a visual distortion of the perceived distance and velocity of moving objects (the Pulfrich effect) or the perceived distance of stationary objects when the observer is moving (e.g., when driving or flying). These alterations in depth and movement perception could be hazardous, although there is the possibility that one may adapt to the distortions.¹⁷⁷ It is good practice to insist that patients not use the X-Chrom while driving or in any situation in which they or others may be at risk for serious injury (see also Chapter 25).

If cosmetic appearance is not a concern, an alternative to the X-Chrom is to tint a spectacle lens or to use a handheld filter.¹⁷⁴ A handheld filter can be assembled

with theater gels (Roscolux filters), Kodak Wratten filters placed in 2×2 slide mounts, or other clear material to protect the filter from wrinkling or fingerprints. A light magenta filter (e.g., Kodak Wratten #30) has been used as an aid for identifying histological stains when clues such as size, shape, and luminosity cannot be used reliably.¹⁷⁸ For use with a microscope, the filter can be placed in front of the eye or, better yet, over the light source, thus making it convenient to alternate between the filtered and the unfiltered conditions. Successive viewing with and without the filter enhances the perceived color differences. A heat filter is recommended when gelatin or acetate filters are used close to a hot light source. Electricians can use the Kodak Wratten #30, mounted in a slide mount and handheld, to avoid joining wires of different colors that should remain separate. Plastic spectacle lenses can be tinted with an appropriate dye, such as BPI Red. One approach is to tint the lower half of both lenses, which provide the benefit of the tint by simply changing the direction of gaze. The advantage of the spectacle lens or a handheld filter over the X-Chrom is that these options permit a rapid successive comparison of how things look with and without the filter. The disadvantages of a red filter are that green objects on a dark background and red objects on a white background disappear—or, rather, their apparent contrast is reduced—when viewed through the filter. It is wise to demonstrate these effects to any patient contemplating an X-Chrom or other filter as an aid. Fletcher and Voke¹⁷⁴ described a procedure for selecting a filter, and they presented a few case reports.

Management of Patients with Rod Monochromacy

Patients who, on the basis of color vision test results, are diagnosed as rod monochromats (typical achromats with reduced visual acuity) should be referred for electrodiagnostic testing to obtain an electroretinogram (ERG) to confirm the diagnosis. Rod monochromats have a characteristic pattern on the Panel D-15, they attain certain values with the Nagel anomaloscope, and they fail to read any of the plates on the Ishihara. With the Sloan Achromatopsia Test, not only do rod monochromats produce a distinctive result, but the ease with which they accomplish the task is a telltale sign of the condition. The ERG shows no evidence of photopic function, although this is also true of the incomplete achromat (blue-monocone monochromat). Differentiation between complete and incomplete achromacy is accomplished with tests of spectral sensitivity. The distinction is often difficult to achieve, and it may require referral to a clinic that is capable of this type of measurement. A simpler solution is to use a plate test devised by Berson and colleagues.¹⁷⁹ The test consists of six color plates (two demonstration and four test plates), and,

when it is used in conjunction with the results of measurements of visual acuity and rod and cone ERGs, it may facilitate the differentiation of the blue-cone from the rod monochromat. Results of the Panel D-15 can also be used to identify the rod monochromat. The rod monochromat's poor visual acuity and aversion to bright lights (photophobia) can be alleviated by an ophthalmic tint of appropriate density. Some patients may benefit from low-vision aids such as handheld magnifiers for near viewing and telescopes for distance viewing. A refractive correction is in order, but, even after correction, acuity will remain poor (typically around 20/150 to 20/200). Uncorrected rod monochromats improve their vision and relieve their photophobia by blinking and squinting.

When selecting a tint for the rod monochromat, it is important to recognize that the usual transmittance of sunglasses is often too high to relieve the photophobia that he or she will experience outdoors. Recall that the spectral sensitivity of the rod monochromat follows the scotopic curve and, therefore, sensitivity to the long wavelengths is greatly reduced (shortening of the red). For this reason, red-tinted lenses, which have a low scotopic luminance transmittance, are often preferred by the rod monochromat.¹⁸⁰ Red and amber tints are a convenient way to achieve a low scotopic luminous transmittance, but they have the disadvantage of reducing the visibility of objects reflecting in the short wavelengths. Lenses to consider to meet the needs of a rod monochromat are the NoIR 107 (dark amber), the NoIR 108 (dark gray-green), and the U-70 or other red lenses. The NoIR series of glasses wrap around or have side shields that are of benefit to most monochromats. For a better cosmetic appearance, contact lenses can be used.¹⁸¹ The advantage of a contact lens is that it allows a tint to be worn constantly indoors; the patient can use a spectacle tint over the contact lens when going out into bright daylight.

The goal is to find a tint that provides the best acuity for the individual, and this may differ from one person to the next by as much as 2 log units.^{182,183} Visual acuity for the rod monochromat improves and then decreases with increasing luminance. Because the change in visual acuity with luminance is quite variable from one observer to the next, there are no set guidelines for choosing the best density. Trial and error with different tints may lead to an acceptable solution. Often a rod monochromat ends up left to his or her own devices, sometimes wearing two pairs of sunglasses at the same time, or else he or she may get no relief and succeed only by squinting. Changes in acuity with light level can, of course, be measured at 4 or 5 light levels to find the light level of maximum acuity. However, because acuity is measured several times, memorization of the chart is likely to occur. A good solution is to use a projected S chart¹⁸⁴ and to vary the light level of the pro-

jector with neutral-density filters or an iris diaphragm over the projector lens. The patient must be told not to squint, because squinting will negate the effect of changing the light level. There is a great deal of satisfaction to be had in helping the rod monochromat who, unfortunately, is all too often dismissed with a diagnosis and the statement that nothing can be done. Nordby's account¹⁶¹ of his own condition is worth reading for insight into patient management. Also of interest is Oliver Sacks' book, *The Island of the Color-blind*.¹⁸⁵ The Achromatopsia Network is another useful resource (<http://www.achromat.org>).

Management of Patients with Acquired Color Defects

General Management

Given that acquired color defects are the result of an anomaly or lesion that affects some part of the visual pathway (including the ocular media), the management of acquired color defects is directed to the treatment of the primary cause. Acquired color defects may affect each eye differently; this is an important characteristic to educate patients about, and it is also the reason that each eye is tested separately. Most patients are not aware that the eyes may be affected differently, and they are unlikely to think of alternately occluding the eyes to make the comparison. If an acquired color defect is suspected and not confirmed, sensitizing a patient to be alert for differences in color vision between the two eyes may bring an earlier diagnosis.

Determining the etiology of an acquired color defect obviously involves other tests and procedures (e.g., acuity, visual fields, tonometry) and referral to or consultation with other health care providers. Repeat color testing is frequently used to monitor the success of treatment. Acquired color defects may, of course, be iatrogenic because of the side effects of drugs or medications. These changes in vision, which may precede other systemic effects, can be anticipated from a drug inventory when the history is taken. For example, color vision testing is important for patients taking digoxin, for which the prevalence of toxicity has been reported to be as high as 20%.¹⁸⁶ With digoxin, there may be a red-green or a blue-yellow defect. Color defects associated with other commonly used drugs are listed in Box 9-2. Lyle^{41,42} provided an extensive inventory of drugs and their effects on vision; other sources include the Physician's Desk Reference and Fraunfelder's book.⁴³

The toxic effect of a drug or another chemical may also produce *chromatopsia*, an abnormal condition in which objects are seen in a particular color or are tinged with that color. The various forms of chromatopsia, summarized in Table 9-7, are classified either according to the color that white is perceived as having or according to the predominant color that is seen. The most

TABLE 9-7 Classification of the Chromatopsias and Their Associated Color Perceptions

Classification	Perception	Cause
Erythropsia	Red	Snow blindness, atropine
Xanthopsia	Yellow	Digitalis, fluorescein
Chloropsia	Green	Epinephrine, lead
Cyanopsia	Blue	Viagra, cataract surgery
Ianthinopsia	Violet	Cannabis

common form appears to be xanthopsia. There does not appear to be any documentation about how common chromatopsia is or how frequently it occurs as a side effect of particular drugs. Aphakes and, to some extent, pseudophakes are likely to have cyanopsia for a short period of time after removal of the crystalline lens. It has been suggested that the increased blue in Monet's paintings during the last 3 years of his life was a result of his modified color perception after cataract surgery^{187,188}; however, it is clear that what an artist chooses to do may not follow a simple interpretation. Some may paint with more blue before surgery to compensate for the short-wavelength absorption by the lens; others may choose to avoid using blue. A patient of mine who once experienced erythropsia as a result of snow blindness subsequently chose to paint with a red wash because of that visual experience.

Chromatopsia may or may not be associated with an acquired color defect. It is often the result of the effects of chemicals or medications and is temporary, even if the cause is not eliminated, because color perception adapts to the new situation. A large variety of drugs and chemicals can produce chromatopsia.^{41,42} A case report of chloropsia resulting from the prolonged viewing of red numbers on the digital readout of a scale used to weigh chemicals is an interesting account of what is sometimes necessary to reach a differential diagnosis. In this case, chloropsia was the complementary (or negative) afterimage of the red light on the visual display of the scale. Other candidates, such as disease, chemicals, and psychological problems (hysteria), were considered but excluded as the cause.¹⁸⁹ Prolonged complementary afterimages have been reported in users of monochrome video display terminals.¹⁹⁰ These may be orientation-contingent after effects, which are known to have a much longer time course than ordinary afterimages.¹⁹¹

Management of Older Patients

As a normal consequence of aging, there are progressive changes in color vision that manifest as a tritan defect.

Age-related changes in performance have been documented by Verriest and colleagues¹⁰⁸ on the FM 100-Hue and by Bowman and colleagues¹⁹² on the Panel D-15 and Lanthony Desaturated Panel-15. These changes are primarily due to yellowing of the crystalline lens,¹⁹³ although there is also evidence for a change in the S cones or the S-cone pathway.¹⁹⁴⁻¹⁹⁶ A younger person can readily experience the effect by looking through a yellow filter, which attenuates the short wavelengths and shifts blues to green and white to yellow. For the older person, increased scattered light and a reduction in light level caused by cataract formation also bring about poorer color discrimination. The effects of yellowing of the lens are accentuated by senile miosis, because the small pupil restricts light to the central and thickest part of the lens.¹⁹⁷

The older person may not recognize the changes in his or her color vision, but they may become apparent when family members notice that the person is misnaming the colors of familiar objects; for example, the blue suitcase is now being called green. The older patient may have difficulty with discriminating some pills on the basis of color¹⁹⁸; this is of significance to the doctor who prescribes drugs to older patients. Because of age-related changes in color vision, it may be difficult to differentiate between green and blue tablets, green and yellow tablets, and yellow and white tablets and between tablets that are different shades of white, yellow, green, or blue. It is wise to conduct a trial with the patient to see that he or she correctly identifies the tablets by their color; the results of this trial may reveal a color defect that may have otherwise gone unnoticed. Not all testing is necessarily done with commercially available color tests.

The ultimate solution to the brunescent, cataractous lens is its removal. After monocular lens extraction, patients should be prepared for a difference in the appearance of colors between their normal eye and their pseudophakic or aphakic eye. Not only will the world look brighter and objects clearer with a monocular implant, but there will be striking differences between the eyes in the perception of colors. Patients need to be educated in this regard, because not all understand the changes in color vision that accompany a lens implant.¹⁹⁹ The situation is different for the aphake who, without an ultraviolet absorber incorporated into a spectacle prescription, is able to see the ultraviolet spectrum that the individual with normal vision cannot; this is because the crystalline lens almost completely absorbs wavelengths between 300 nm and 380 nm. Interestingly, colors seen by the aphake in the short visible wavelengths and in the ultraviolet spectrum do not, as one might expect, become increasingly more violet as wavelengths get shorter. Beginning at 420 nm, there is some loss of saturation, and colors shift a little in the red direction; at 400 nm, there is a shift toward

green; and at 350 nm, there is a blue sensation nearly equivalent to that of 445 nm.²⁰⁰ These changes for the aphake are likely caused by the ultraviolet absorption bands of the cone photopigments, which are ordinarily shielded from the ultraviolet spectrum by the lens.

THE HEALTH PROFESSIONAL WITH DEFECTIVE COLOR VISION

Two color-deficient physicians—Currier²⁰¹ and Spalding²⁰²—have described some of the problems they experienced because of their perception of color or their poor color discrimination. To my knowledge, there has been only one written report from a color-defective optometrist, Cockburn.²⁰³ Currier is a neurologist who has a deutan color deficiency (perhaps he is a deuteranope), and he states that he has had no serious problems with practicing his specialty. He gives an interesting account of his color-related problems encountered during daily activities and professional situations, including difficulty with distinguishing veins and arteries, venous and arterial blood hemorrhages in the fundus, colors on microscope slides, and some colors used in lectures. Spalding is a deutanope who did not appreciate the extent of his problem until he retired from practicing general medicine. He admits to a number of problems, such as "missing the pallor of severe anaemia," having "difficulties in detecting traces of blood," and not recognizing the cyanosis associated with severe illness. Both of these physicians conclude that color vision defects may impair clinical skill in some situations and that prevacational testing is important. Cockburn has extreme deuteranomaly and an "obsession with sailing." He wrote more about difficulties with sailing, dealing with navigational aids, and his strategies for compensating for his color defect with regard to his hobbies than he did about his profession. The most severe difficulty he had as an optometrist was to differentiate between retinal hemorrhage and choroidal pigment, which was made more apparent with the successive use of colored filters in the ophthalmoscope, switching from orange to red free. He also admitted to problems with detecting inflammation of the skin and erythematous changes in the ocular adnexa. In addition, he avoided assisting with frame selection. Health care providers should know if they have a color defect, and they should be aware of the potential limitations it may impose. Although normal color perception may not be essential for the practice of medicine or optometry, color does play an important role in the diagnosis of many conditions. Color-defective practitioners should know their limitations and when to rely more on other procedures to arrive at a diagnosis.

Voke¹⁴⁸ reviewed some of the problems experienced by color-defective persons in the health professions, where there are many activities for which a color defect is a handicap (see Box 9-3). A deutanopic nurse reported having difficulty reading color-coded charts, differentiating yellow pus and blood, seeing blood in vomit, and detecting jaundice. A physician admitted to having similar problems and placing more reliance on procedures other than color differentiation. Similarly, a protanomalous anesthesiologist compensated by relying more on instrument assessment than on skin coloration. A protanopic ophthalmologist admitted to problems with fundus evaluation, as did a protanopic optometrist, who also mentioned problems with handling tinted lenses and administering color vision tests.

My experience with color-defective optometry students clearly indicates some potential limitations. Educational materials, including color illustrations in books and color slides, may not be fully appreciated by the color-defective student. A protanope described having difficulty seeing a choroidal melanoma and a choroidal nevus illustrated in a color atlas of retinal disease and finding black-and-white illustrations that accompany some color illustrations to be very helpful. Of course, difficulty with detecting abnormalities in color illustrations or photographs does not indicate that abnormalities would go undetected in a patient, or vice versa. A protanopic optometrist offered that red-free ophthalmoscopy was not as big a benefit to him as he believed it to be to those with normal color vision. This makes sense given that protans perceive reds as fairly dark. It is wise for the color-defective student not to conceal his or her difficulty but rather to seek advice and learn how to compensate properly. Spalding²⁰² contended that doctors who have a defect may not be willing to acknowledge their color deficiency, because it may indicate a lack of competence. It is better to admit any limitation and learn as much about it and how to compensate for it than it is to commit an error. All persons contemplating a career in the health professions should be tested for color vision. Those with a color defect should not be excluded, but rather they should be counseled and made aware of the deficiency early in their training so that they can maximize their learning. Vision tests for those contemplating a career in the health professions should be as routine as admission tests of academic aptitude; they should not be given to exclude color defectives but rather guarantee that appropriate counseling and advice are provided.

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9-1

Making Your Own Color Test

These instructions are for making a set of pseudoisochromatic plates that can be used for screening for red-green and blue-yellow color defects. The test is a modified version of the Farnsworth tritan plate, or F-2, and it consists of three plates; a black-and-white demonstration plate can also be added.

Plate 1: A blue test figure on a purple background

Plate 2: A green test figure on a purple background

Plate 3: A plate with just the purple background

The advantage of this modification of the F-2 over the original is that the task is more readily apparent to the patient, thus making the instructions easier to give and the results easier to interpret.

The following materials are needed to construct the test:

- Eight 3 × 5-inch matte Munsell papers having the following specifications (available from Munsell Color, Baltimore, MD):
 - 7.5 PB 7/4, 7.5 PB 6/4 (One each; these are for the blue test figure.)
 - 2.5 GY 7/4, 2.5 GY 6/4 (One each; these are for the green test figure.)
 - 5P 7/4, 5P 6/4 (Two each; these are for the background color on each plate. Although only one each of these papers is needed, two are suggested in case you waste some of the paper when making the plates.)
- 3M #568 Positionable Mounting Adhesive (available at art supply stores)
- Paper punch that produces $\frac{1}{4}$ -inch discs (a commonly available punch, but it must be sharp—buy a new one for this purpose)
- Three or four 5 × 5-inch pieces of white cardboard (poster board) with a matte finish
- Teflon tweezers
- Cotton gloves

Each of the plates is made from $\frac{1}{4}$ -inch discs of Munsell paper that you will mount on the white cardboard. Each plate has 121 discs: a square with 11 discs on each side. The position of the light and dark shades—Munsell values of 7 and 6, respectively—is shown in Figure 9-49, A. An outline shows the location of the discs for the blue and green test figures.

Apply the mounting adhesive to the back side of each of the Munsell papers following the product's instructions. Avoid getting your fingerprints on the Munsell papers; it helps to wear white cotton gloves. After applying the mounting adhesive, punch the Munsell paper, and save the $\frac{1}{4}$ -inch discs that the punches create. Make your punches close together to avoid wasting paper, and keep the different shades in separate containers. Each disc can then be picked up with Teflon tweezers (the kind used for contact lenses are fine); the protective backing of the mounting adhesive can be flipped off by lightly striking the edge of the disc. Now, lay down the light and dark shades of discs for the purple background that will make the top row on the card. Place the discs so that the edges touch and form a straight row that is parallel to the top of the card. Create the first column of discs. Use care when setting up the first row and column, as they set the stage for where all of the other discs are eventually placed. It is important to get the alignment of the first row and column correct.

Remember that you are using a positionable mounting adhesive, which means that the discs of paper can be moved around on the white cardboard stock if you have not pressed them too firmly in place. Firm pressure can be applied after the entire plate has been assembled.

A black-and-white demonstration plate can be made by photocopying Figure 9-49, B, and mounting it to a 5 × 5-inch white card. The test is designed for illumination with a Macbeth easel lamp or a suitable substitute, and it should be viewed from a distance of 50 to 75 cm. When administering each of the color plates, ask the patient if a square is visible. Administer each plate, and, if both squares are seen, show both plates with the square test figure and ask the patient which square is easier to see. Persons with normal vision will see both the blue and green squares, and, when asked to compare the relative visibility of the two, they will specify the green square. Persons with red-green defects will see only the green square. Those with blue-yellow defects may see just the blue square, or they may see both squares, with the blue one being more prominent. The results of the test can be recorded in the following manner:

- 2/Gr: Two squares were seen, with the green one more prominent (normal color vision).
2/Bl: Two squares were seen, with the blue one more prominent (a possible blue-yellow defect).
1/Gr: One square was seen, and it was green (red-green defect).
1/Bl: One square was seen, and it was blue (blue-yellow/tritan defect).

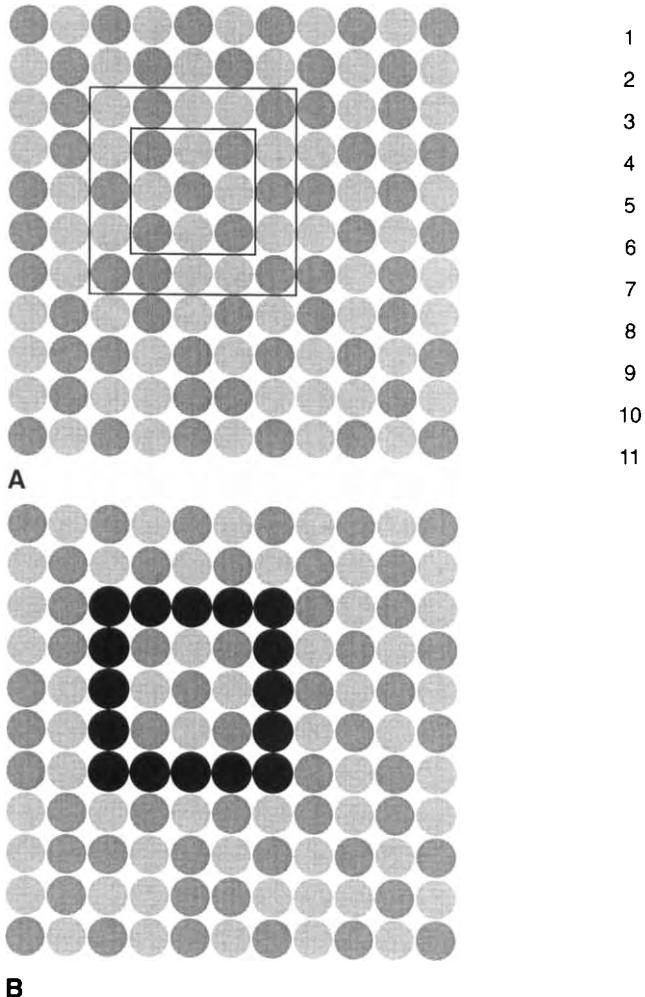


Figure 9-49

A blueprint (A) and demonstration plate (B) for the modified F-2 test. The dark and light gray discs identify the positions of the Munsell papers having a value of 6 and 7, respectively.

9-2

Sample Score Forms

The following score forms for the Ishihara (Figure 9-50), Panel D-15 (Figure 9-51), Lanthony Desaturated Panel-15 (Figure 9-52), and H-16 (Figure 9-53)

tests are made available for the practitioner to copy. The heading of each could be appropriately modified for use in any practice. Copy, cut, and paste as you wish.

ID No. _____

ISHIHARA PSEUDOISOCHROMATIC PLATES
24 PLATE EDITION

COLOR VISION SERVICE

University of Houston College of Optometry
Houston, TX 77204-6052
(713) 743-1961

PATIENT: _____ DATE: _____

EXAMINER: *Paul Pease, O.D., Ph.D.*

ISHIHARA			Response		
PLATE	No. on Plate	R-G Deficiency Response	RE	LE	
1	12	12			
2	8	3			
3	29	70			
4	5	2			
5	3	5			
6	15	17			
7	74	21			
8	6	X			
9	45	X			
10	5	X			
11	7	X			
12	16	X			
13	73	X			
14	X	5			
15	X	45			
		PROTAN	DEUTAN		
		Strong	Mild	Strong	Mild
16	26	6	(2) 6	2	2 (6)
17	42	2	(4) 2	4	4 (2)
<input type="checkbox"/> PASS <input type="checkbox"/> FAIL <input type="checkbox"/> PROTAN <input type="checkbox"/> DEUTAN					
A failure occurs with 5 or more errors out of the first 13					

The mark X shows that the plate cannot be read. The numerals in parentheses show that the numerals can be read but they are comparatively unclear.

Figure 9-50

Ishihara Pseudoisochromatic Plates, 24 Plate Edition.

ID No. _____

FARNSWORTH DICHOTOMOUS TEST FOR COLOR BLINDNESS: PANEL D-15**COLOR VISION SERVICE**

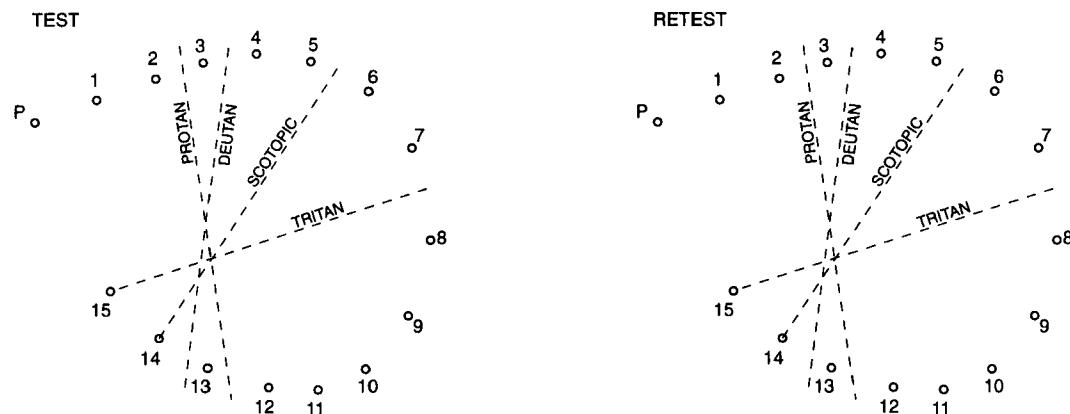
University of Houston College of Optometry Houston, TX 77204-6052 (713) 743-1961

DATE: _____

PATIENT: _____ EXAMINER: *Paul Pease, O.D., Ph.D.*

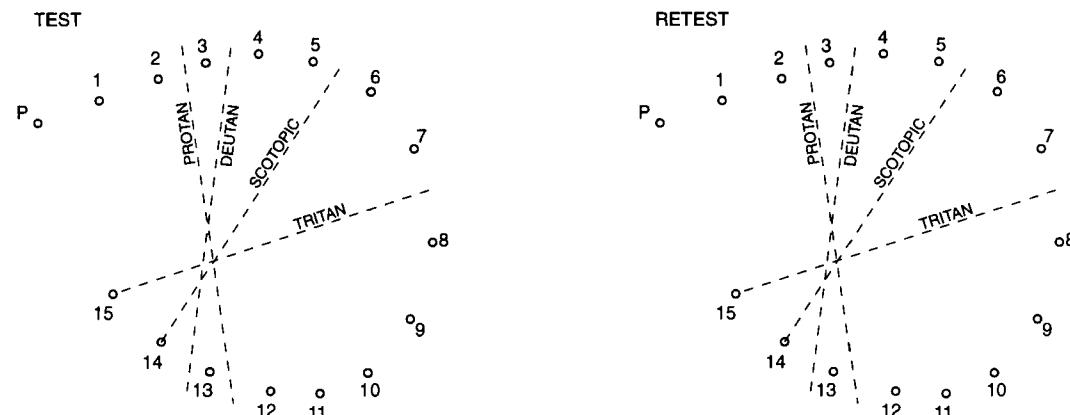
RIGHT EYE	<input type="checkbox"/> PASS	<input type="checkbox"/> FAIL	<input type="checkbox"/> PROTAN	<input type="checkbox"/> DEUTAN	<input type="checkbox"/> TRITAN	<input type="checkbox"/> SCOTOPIC
------------------	-------------------------------	-------------------------------	---------------------------------	---------------------------------	---------------------------------	-----------------------------------

TEST	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
RETEST	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-



LEFT EYE	<input type="checkbox"/> PASS	<input type="checkbox"/> FAIL	<input type="checkbox"/> PROTAN	<input type="checkbox"/> DEUTAN	<input type="checkbox"/> TRITAN	<input type="checkbox"/> SCOTOPIC
-----------------	-------------------------------	-------------------------------	---------------------------------	---------------------------------	---------------------------------	-----------------------------------

TEST	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
RETEST	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-

**Figure 9-51**

Farnsworth Dichotomous Test for Color Blindness: Panel D-15.

ID No. _____

DESATURATED PANEL 15 TEST FOR COLOR BLINDNESS**COLOR VISION SERVICE**

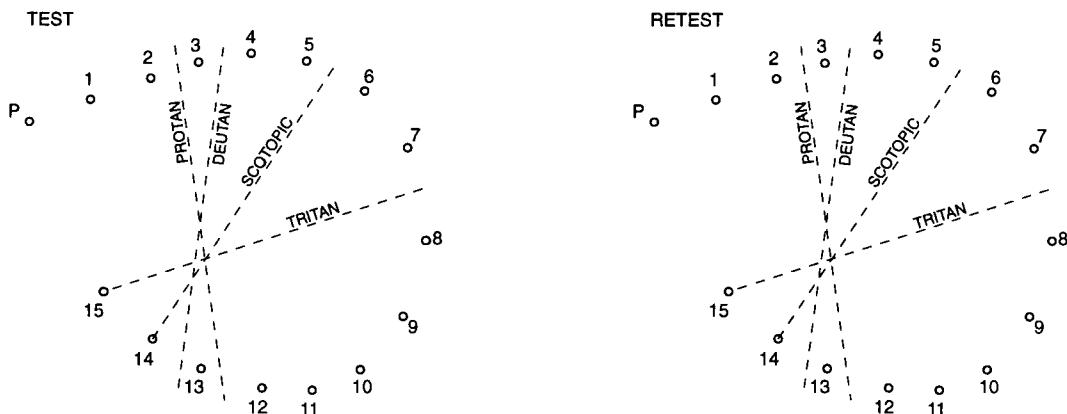
University of Houston College of Optometry Houston, TX 77204-6052 (713) 743-1961

DATE: _____

PATIENT: _____ EXAMINER: *Paul Pease, O.D., Ph.D.*

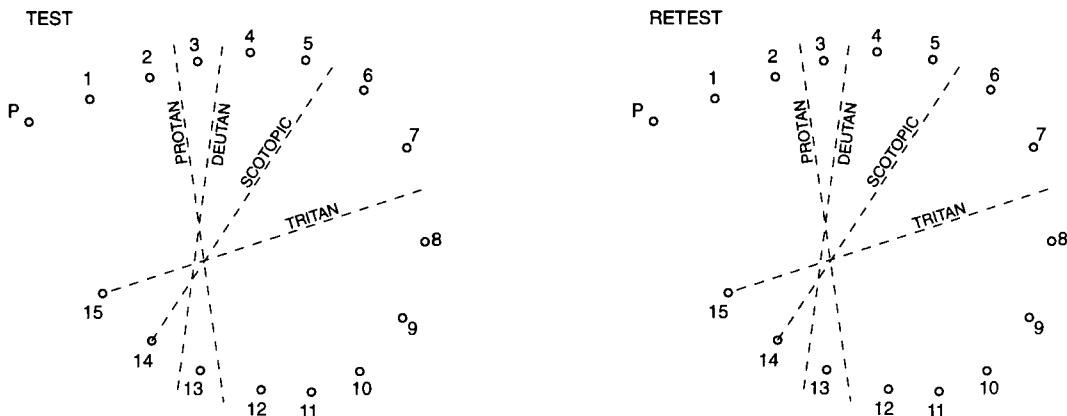
RIGHT EYE PASS FAIL PROTAN DEUTAN TRITAN SCOTOPIC

TEST	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
RETEST	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-



LEFT EYE PASS FAIL PROTAN DEUTAN TRITAN SCOTOPIC

TEST	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
RETEST	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-

**Figure 9-52**

Desaturated Panel 15 Test for Color Blindness.

ID No. _____

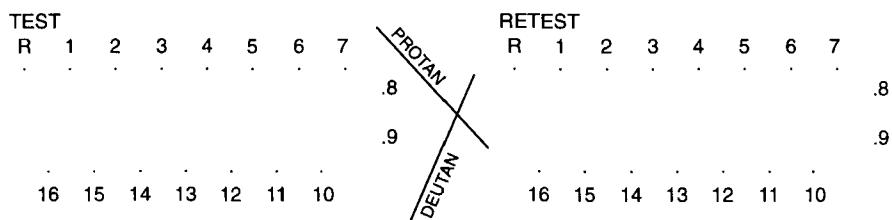
FARNSWORTH H-16 TEST**COLOR VISION SERVICE**

University of Houston College of Optometry
 Houston, TX 77204-6052
 (713) 743-1961

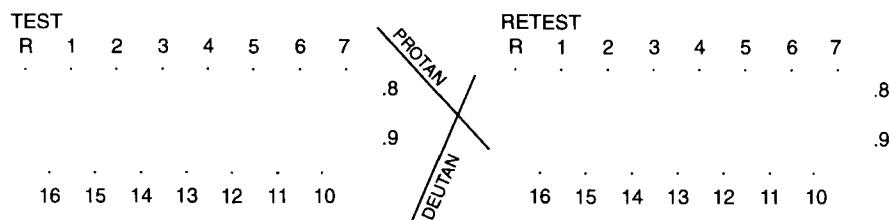
PATIENT: _____ DATE: _____

EXAMINER: *Paul Pease, O.D., Ph.D.***RIGHT EYE** PROTAN DEUTAN

<u>TEST</u>	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
<u>RETEST</u>	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—

**LEFT EYE** PROTAN DEUTAN

<u>TEST</u>	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
<u>RETEST</u>	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—



Dichromasy indicated by 3 or more crossing not including caps 7, 8, 9 or 10.

Figure 9-53

Farnsworth H-16 Test.

9-3

Color Difference Tables

To calculate the color difference score for the Panel D-15 with a cap sequence without error (P, 1, 2 through 15), the color differences in the table are summed along the diagonal ($9.4 + 6.7 + 5.9 + 5.9 + 4.5$, and so on). The total score is 117. For the cap sequence

for the Deutan in Figure 9-37, also shown in the column heads of Tables 9-8 and 9-9 with the corresponding color difference scores, the sum resulted in a total color difference score of 362.2 (Tables 9-8 and 9-9).

TABLE 9-8 Color Differences Between Each Cap of the Panel D-15 and All Other Caps

Cap no.	P	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
P	—	9.4	15.9	21.7	27.5	31.9	39.1	46.4	51.1	49.4	44.3	40.4	37.2	33.3	30.3	23
1	—	—	6.7	12.5	18.4	22.8	30.5	38.7	44.9	44.4	41.5	38.8	36.7	34.3	33	26.5
2	—	—	5.9	11.7	16.1	24	32.6	39.7	40	38.8	37	35.8	34.6	34.6	28.9	
3	—	—	—	5.9	10.4	18.8	28.1	36.4	37.7	38.3	37.5	37	36.9	37.8	32.8	
4	—	—	—	4.5	13.3	23.2	32.6	35	37.4	37.6	38	38.8	40.6	40.6	36.4	
5	—	—	—	9.4	19.7	29.9	33.2	37	38	38.9	38.9	40.5	42.9	42.9	39.3	
6	—	—	—	10.5	21.7	26.3	32.7	35.1	32.7	35.1	37.3	40.3	43.9	43.9	41.9	
7	—	—	—	—	12.1	18.4	27.8	31.8	31.8	35.3	39.8	44.6	44.6	44.6	44.3	
8	—	—	—	—	7.9	19.7	25.2	29.8	25.2	29.8	35.6	41.4	43.1	43.1	43.1	
9	—	—	—	—	—	12.2	18.1	23	18.1	23	29.3	35.4	38.1	38.1	38.1	
10	—	—	—	—	—	—	—	6.3	—	11.5	18.2	24.5	24.5	24.5	28.6	
11	—	—	—	—	—	—	—	—	—	5.2	11.9	18.2	22.8	22.8	22.8	
12	—	—	—	—	—	—	—	—	—	—	7	13	18	18	18	
13	—	—	—	—	—	—	—	—	—	—	—	—	6.5	12.2	12.2	
14	—	—	—	—	—	—	—	—	—	—	—	—	—	7.5	—	
15	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	

P, Pilot cap.
From Bowman KJ. 1982. A method for quantitative scoring of the Farnsworth Panel D-15. Acta Ophthalmol 60:907-916.

TABLE 9-9 Calculated Color Differences Between Each Cap of the Lanthony Desaturated Panel-15 and All Other 15 Caps

Cap no.	P	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
P	—	3.3	6.7	9.9	11.9	14.4	17.7	21	21.6	19.7	15.2	13.2	12	10	8.6	6.7
1	—	—	3.4	6.8	9	11.7	15.6	19.6	21	19.9	16.4	15	14	12.4	11.4	9.8
2	—	—	3.6	6.1	9	13.4	18.2	20.5	20.2	17.8	16.9	16.2	15	14.3	12.9	—
3	—	—	2.6	5.6	10.2	15.5	18.5	19.1	17.9	17.7	17.3	16.7	16.3	15.3	—	—
4	—	—	3	7.7	13.3	16.7	17.9	17.6	17.9	17.8	17.5	17.5	17.5	16.8	—	—
5	—	—	4.8	10.8	14.8	16.7	17.5	18.3	18.5	18.6	18.9	18.6	18.5	18.5	—	—
6	—	—	—	6.2	10.9	14	16.6	18.2	18.9	19.7	20.5	20.6	20.6	20.6	—	—
7	—	—	—	5.5	9.9	14.7	17.1	18.3	19.9	21.3	22.1	—	—	—	—	—
8	—	—	—	—	5.1	11.3	14.2	15.7	17.9	19.6	20.9	—	—	—	—	—
9	—	—	—	—	—	6.7	9.9	11.6	14	15.9	17.6	—	—	—	—	—
10	—	—	—	—	—	—	3.2	4.9	7.6	9.6	11.6	—	—	—	—	—
11	—	—	—	—	—	—	—	1.8	4.5	6.6	8.7	—	—	—	—	—
12	—	—	—	—	—	—	—	—	2.7	4.8	7	—	—	—	—	—
13	—	—	—	—	—	—	—	—	—	2.1	4.3	—	—	—	—	—
14	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
15	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—

P, Pilot cap.

Courtesy of Dr. Kenneth J. Bouman.

10

Ocular Motility

C. Denise Pensyl, William J. Benjamin

Clinicians are faced with the challenge of differentiating the etiologies of asthenopia, blur, diplopia, and headaches associated with the use of the eyes by their patients. Oculomotor deficiencies can be one of several possible causes of such symptoms and are the result of defects in the central nervous system, afferent or efferent nerve pathways, or local conditions of a nature so as to impede appropriate oculomotor function. Oculomotor function will be extensively analyzed during phorometry (see Chapter 21) in terms of binocularly and muscle balance after the subjective refraction has been completed. This chapter focuses on clinical procedures that are typically used to analyze oculomotor function before the subjective refraction is performed, though in some cases the practitioner may decide to use a few of these tests after the refraction is determined.

By using primarily handheld devices or instruments, the normality or abnormality of the functions of the ocular muscles are estimated: the iris sphincter and dilator muscles, the eyelid muscles involved in maintenance of the palpebral aperture, the extraocular muscles, and the ciliary muscles. Hence, ocular motility is assessed by observation of (1) the pupils and pupillary reflexes, (2) the palpebral apertures and eyelid movements, (3) monocular and binocular eye movements, (4) monocular and binocular eye alignment, and (5) accommodative amplitudes and facility.

THE PUPILS AND PUPILLARY REFLEXES

The pupil controls retinal illumination and determines retinal image quality. The entrance pupil of the eye is formed by refraction of the real pupil by the cornea and is just over 3 mm behind the anterior corneal surface. Retinal illumination is proportional to the square of the pupillary diameter. Depths of field and focus for clear vision are inversely proportional to pupil diameter. The lower limit of pupil size for optimal visual acuity is

approximately 2 mm, below which the effects of reduced retinal illuminance and diffraction outweigh the beneficial aspects of an increase in depth of field and reduction of ocular spherical aberration. The entrance pupil also controls blur circle size at the retina for object rays not originating from the far point plane of the eye.

The entrance pupil averages 3.5 mm in diameter in adults under normal illumination but can range from 1.3 mm to 10 mm. It is usually centered on the optic axis of the eye but is displaced temporally away from the visual axis or line of sight an average of 5 degrees. The entrance pupil is decentered approximately 0.15 mm nasally and 0.1 mm inferior to the geometric center of the cornea.¹ This amount of decentration is not distinguished in casual observation or by the clinician's normal examination of the pupils. In general, the diameter of the pupil gradually becomes smaller after about the age of 12 to 18 years. This appears to be a linear relationship in which pupil size for light-adapted and dark-adapted eyes at age 20 (means nearly 5 mm and 8 mm, respectively) both diminish to about 2 mm and 2.5 mm, respectively, at age 80^{2,3} (see Figure 25-4). The progressive change in size is known as senile miosis and is shown in Table 10-1. It is not totally explained by increased iris rigidity or loss of muscle fibers in the iris, but apparently also includes a progressive delayed latency of response time, indicating some neurological involvement.² Aging is presumed to cause a reduction in sympathetic tone.⁴ Because it is only the entrance pupil that can be examined noninvasively by the practitioner, in clinical parlance the word "entrance" is dropped from the term and the single word "pupil" is meant to apply to that which is observed, which is the entrance pupil.

Pupil size is always changing in the normal eye because of convergence/accommodation (near or triad response), pupillary responses to light and small, regular oscillations from fluctuations in the sympathetic and parasympathetic nervous systems. Pupil size can be influenced by drugs and medications (see Chapter 12) and is slightly larger in persons with light irises

TABLE 10-1 Approximate Pupil Diameters According to Age

Age (years)	Photopic Diameter (mm)	Scotopic Diameter (mm)
20	5.0	8.0
40	4.0	6.0
50	3.5	5.5
60	3+	4.25
70	2.5	3.0
80	2+	2.5

compared with dark irises. Pupils become mydriatic in response to large sensory, emotional, or psychological stimuli. Hyperthyroidism and ingestion of lead can produce mydriasis. Various medications, including antihistamines, over-the-counter decongestants, anticholinergics, phenothiazines, amphetamines, cocaine, and antianxiety agents, may also enlarge the pupils.^{5,6} The pupils become miotic in response to pain or irritation within the globe as a result of the oculopupillary reflex during keratitis, iritis, or trauma.⁷ However, pain elsewhere in the body tends to cause pupil dilation. Long-standing diabetes, sleep, and intraocular inflammation produce miosis. The pupils of infants and the elderly are small.⁸ Medications, including cholinergic agents used to treat glaucoma, chlorpromazine, cholinesterase inhibitors (found in insecticides and toxic nerve gases), and morphine derivatives, constrict the pupil.^{5,6} Under ordinary circumstances, the pupils of females may be larger than those of males, and the pupils of myopes larger than those of hyperopes. However, the pupil sizes of females and males are no different, and of myopes and hyperopes no different, when specialized conditions ensure that accommodative differences have been accounted for.⁹

The pupils should appear round, roughly centered within the iris, and of equal size in the normal patient. The irises should be of the same coloration. The clinician should be aware of deviations from this norm, which could appear in the form of a unilateral or bilateral irregular pupil (a nonround pupil), ectopic pupil (significantly decentered pupil), polycoria (more than one pupil in an eye), anisocoria (pupils of different sizes in the two eyes), or heterochromia (irises of different color or lightness/darkness in the two eyes). In ocular albinism and oculocutaneous albinism, the irises of the two eyes appear to contain little or no color and scattered light enters the eye through the iris, such that the pupil is not allowed to perform its optical functions. In aniridia, the iris is absent or only partially present; therefore, the pupil does not exist or is considered to be expanded to the ciliary body.

Pupil size is controlled by smooth muscles innervated by the autonomic nervous system. An effective competition between the radial dilator muscle, which is sympathetically innervated and acts to dilate the pupil, and the annular sphincter muscle, which is parasympathetically innervated and acts to constrict the pupil, determines the pupil size. The parasympathetic innervation has more control over the pupil size, because the sphincter muscle is the stronger of the two. The dilator muscle acts in opposition such that mydriasis occurs when the sphincter tone is released. As a result, constriction of the pupil to light (miosis) is slightly faster than dilation (mydriasis) when the light is extinguished. The unstable equilibrium reached between the sphincter and dilator muscles creates small continuous variations in the pupil size, which are normal and called *pupillary unrest* or *hippus*.¹⁰ The normal function of the pupil and its innervation are extensively covered in Chapter 4, and pharmacological manipulation of the pupil is covered in Chapter 12. The reflexes controlling pupil size are important in the diagnosis of neuro-ophthalmic conditions.

The principal sensor for the pupillary light response is the photopic system (the cones); therefore, illumination of the fovea is the primary determinant of the pupillary light reflex. The retinal nerve fibers that relay information for pupillary control travel through the optic nerve to the optic chiasm. Here, approximately half of the afferent (sensory) fibers decussate to the contralateral optic tract, and the remaining half continue in the ipsilateral tract. The afferent fibers follow the brachium of the respective superior colliculus to the midbrain, where they synapse with a pretectal nucleus in the hypothalamus. Here, the fibers undergo semidecussation. Each of the pretectal nuclei sends crossed and uncrossed intercalated neurons through the posterior commissure to the Edinger-Westphal nucleus, the origin of the efferent (motor) fibers that control pupil size.⁷ Therefore, the photopic information given to one eye is normally transmitted to both pupils, creating direct and consensual (indirect) responses. Both of these responses, the direct pupillary light reflex and the consensual pupillary light reflex, should be evaluated when the pupillary responses are examined.

The parasympathetic innervation to the iris, starting in the Edinger-Westphal nucleus, travels with the third cranial nerve, emerging from the brain ventrally between the cerebral peduncles. The nerve then follows a course along the posterior communicating arteries to pierce the wall of the cavernous sinus. Here it is close to the first and second divisions of the fifth nerve. The nerve enters the orbit via the superior orbital fissure. The preganglionic parasympathetic fibers deviate from the third cranial nerve and synapse at the ciliary ganglion. Postganglionic fibers reach the iris sphincter muscle via the short ciliary nerves.⁵ The overwhelming majority of

these parasympathetic fibers innervate the ciliary muscle for control of accommodation, whereas only 3% of the fibers innervate the iris sphincter muscle.¹¹

The iris dilator muscle receives innervation from the sympathetic system, which begins in the posterior hypothalamus. Efferent fibers travel to the brain stem and synapse in the intermediolateral gray matter of the spinal cord (ciliospinal center of Budge) at the T₂ level. Preganglionic efferent fibers or second-order neurons exit at this level into the thorax and then travel to synapse in the superior cervical ganglia, located at the level of the angle of the jaw. The postganglionic efferent fibers or third-order neurons follow the internal carotid arteries and reach the orbits by way of the superior orbital fissures, eventually joining the ophthalmic division of the fifth cranial nerve to innervate the iris dilator by way of the long ciliary nerve in each eye. At the carotid bifurcation, fibers that supply the sweat glands split from those that supply the iris dilator and muscle of Müller in the upper eyelid.^{5,7}

With near fixation, the pupils of both eyes constrict as part of triad response of accommodation, convergence, and miosis. The pupillary near reflex does not depend on retinal illumination and is, therefore, present in a blind eye.^{7,12} How the accommodative input reaches the third nerve nucleus is not completely understood. However, midbrain lesions affecting fibers just approaching or leaving the pretectal synapse often affect the light reflex but spare the accommodative reflex. It is believed the midbrain center for the accommodative reflex may be located slightly ventral to the center for the light reflex (pretectal nucleus).¹³ Hence, the near reflex is nearly always present when the direct light reflex is intact.

Clinical Evaluation

The pupils should be observed for their size and shape, direct and consensual responses to light, and accommodative miosis. They should be observed individually and in comparison with each other. The direct reflex is noted when a light beam is directed into one eye, and its pupil constricts. The consensual (indirect) reflex occurs when the light is shone into one eye and the pupil in the opposite eye contracts. The swinging flashlight test detects afferent defects due to anomalies in the retina or optic nerve pathway anterior to the lateral geniculate nucleus. The pupil cycle time is determined by the rapidity with which the neural and muscular components act to constrict and dilate the pupil.

Analysis of Pupil Size

Measurement of the physiological pupil diameters requires the patient to be adapted to the level of illumination in the immediate environment, typically under normal room illumination. After the patient history is completed, but before changes in room illu-

mination are made, is an ideal time to make the assessment. The room should be illuminated well enough to clearly see the pupils, but light should not be beamed directly at the eyes. The patient is asked to fixate a distance target, and the diameter of each pupil is measured with a millimeter rule held against the cheekbones and covering the lower half of the patient's pupils. The zero mark is aligned with one edge of the pupil and the point that intersects the other edge of the pupil, at its largest diameter, is the pupil size. The pupil size can also be estimated by size comparisons with a series of filled circles, having a progression of diameters from small to large, as is often performed with a pupil gauge (Figure 10-1). An alternative pupil gauge has a series of filled hemispheres, progressing in size from small to large, for which one vertical or lateral pupil margin is aligned with the edge of a filled half circle (Figure 10-2). The scale is moved to the appropriate hemisphere, at which the opposing edge of a half circle is simultaneously coincident with the corresponding edge of the pupillary margin.^{8,14} The entoptic pupillometer, a less common subjective instrument for determining pupil size using the Scheiner disc principle, is covered in the appendix of Chapter 20.

If a millimeter rule is used, the pupil size should be recorded to the nearest 0.5 mm with the right eye recorded first (i.e., 3/3). Even if graded in 1.0-mm increments, the pupil size can be interpolated to the nearest 0.5 mm when using pupil gauges of the filled circle, hemispherical, or Scheiner principle designs. Pupillary unrest is generally of a magnitude below that of 0.5 mm and should not significantly influence the results.

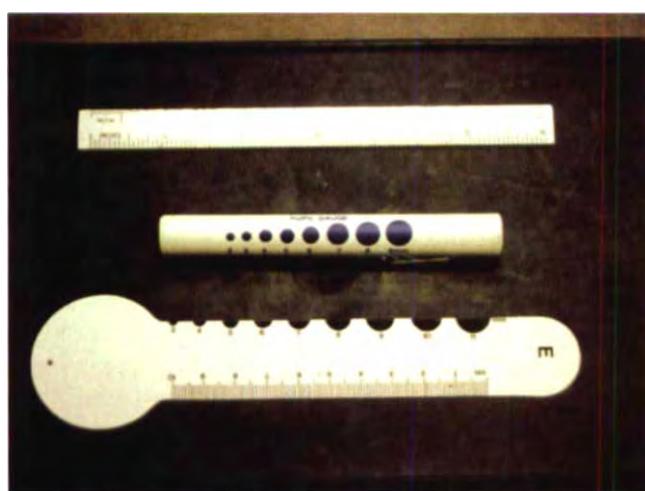


Figure 10-1

Measurement devices for pupil diameter: *Top*, a millimeter rule. *Middle*, a pupil gauge having filled circles of progressively greater diameter, printed on the side of a penlight. *Bottom*, a pupil gauge having filled hemispheres of progressively greater diameter, produced in combination with a millimeter rule.

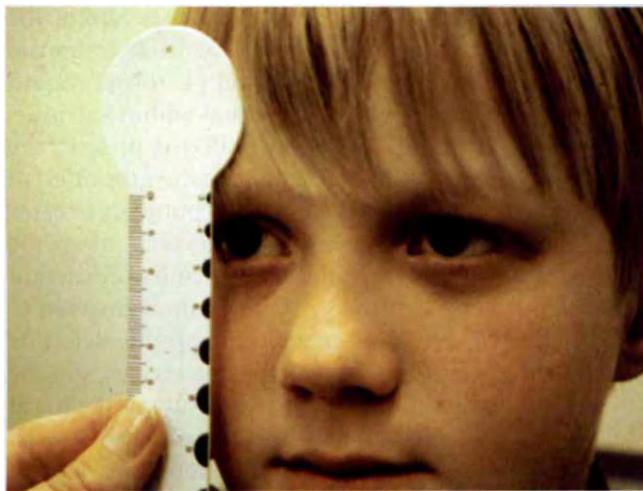


Figure 10-2

Pupil diameter of a young patient's right eye is being measured with a hemispherical pupil size gauge.

In cases of very dark irises, a heightened level of illumination may be required in order to distinguish the pupil against the surrounding iris. An ultraviolet lamp, such as the Burton lamp, can be helpful in viewing the pupils in these instances. The lamp should be held about 15 cm from the patient's face. The examiner views through the magnifying lens of the lamp while assessing the pupil sizes with a millimeter rule or pupil gauge.

If a significant difference in size is noted between the two pupils (anisocoria), the pupils should be remeasured under bright and dim conditions to note if the anisocoria varies with illumination. Normally the pupil size ranges from 2 to 4 mm in bright light and from 4 to 8 mm in dim illumination.¹⁵ To measure the pupils under "bright" conditions, the room illumination should be maximized; additionally the stand light, or other auxiliary light source, may be directed toward the patient's eyes. Care should be taken that the lighting is not so bright as to cause significant patient discomfort. The measuring procedures previously described are used on each pupil. The auxiliary lighting is then removed and the room lighting dimmed as much as possible, until the clinician is just able to see the pupils unaided. Again, the pupil diameters are measured with a rule or gauge.

Pupil sizes under bright and dim conditions may also be assessed using a direct ophthalmoscope. The examiner should be positioned directly in front of the patient in a darkened room, the only light present being a distance fixation target or chart. With the ophthalmoscope set to +1 D and turned up to its fullest intensity through the largest aperture, the light beam is directed onto the patient's face from a 1-m distance. This simulates the "bright" conditions binocularly. The practitioner views the red pupillary reflexes through the aperture of the scope and compares the sizes of the entrance pupils. The red reflexes enhance the ability to detect small differ-

ences in pupil sizes. The eye with the largest pupil should be noted and the size difference estimated or measured with a rule or gauge. To assess the pupils under "dim" conditions, the intensity is reduced until the red reflexes are barely visible.¹⁴ Again, differences in pupil size are noted and estimated or measured.

The findings for bright and dim conditions should be recorded separately. If no difference in the amount of anisocoria occurred between light and dim conditions, the examiner should record that the anisocoria was equal under bright and dim conditions. When anisocoria is present in equal amounts under both bright and dim conditions and is not accompanied by other clinical signs, it is known as simple, physiological, or essential anisocoria. Twenty percent of the population may manifest this discrepancy in the amount of 0.4 mm or more,¹⁶ which is of little or no adverse consequence. The anisocoria can often be observed in old photographs of the patient, especially if the red reflexes are present. Small amounts of anisocoria are often easier to detect in dim illumination.^{13,17} However, anisocoria that varies significantly with the amount of room illumination can be pathological in origin, and further investigation of such patients is necessary.

Pupil size, and its measurement, has become increasingly important in the outcomes of corneal and refractive lens surgery. Disturbance of vision and optical glare phenomena are produced when the pupil diameter increases, most commonly at night. A pupillometer should be used to assess the pupil size under scotopic conditions before surgery to choose the appropriate ablation zone or the phakic IOL optic size.¹⁸

Direct and Consensual (Indirect) Light Reflexes
Upon completion of the pupil size analysis, each pupil should be observed for a direct response to light. The room illumination should be semi-darkened yet sufficient for the clinician to easily view the pupils from a distance of 30 cm or less. The patient is asked to fixate a distant target, perhaps a projected 20/400 (6/120) Snellen letter, as the examiner sits or stands slightly off to one side so as not to be in the patient's direct line of sight. The beam from a handheld light source, usually a penlight or transilluminator, is directed toward the patient's right eye for 2 to 4 seconds and is then removed. The clinician notes the change in the pupil size due to the direct light reflex, in terms of the initial constriction when the penlight beam is directed into the eye, and the dilation when the penlight beam is removed. The magnitude (quantity) of change and the rapidity (quality) of the change in pupil size should be noted. The constriction will be slightly faster than the dilation, as noted previously. The direct response of the right eye should be elicited two or three times to confirm the result. The penlight is then directed toward the patient's left eye. The quantity and quality of the left

pupil's direct responses are noted two or three times and compared with the responses of the right eye. The direct responses of the two eyes should be the same in terms of magnitude and rapidity.

Repeating the process involved in assessment of the direct reflexes, the clinician next assesses the consensual (indirect) reflexes. When the penlight beam is directed into the right eye, the examiner observes the pupillary response of the left eye; similarly, the right eye is observed when the beam is directed into the left eye. Not only should the consensual responses of the two eyes be the same, but the consensual responses should be equal to the direct responses. Constriction will again be slightly faster than dilation. In routine cases, the examiner can simultaneously assess the direct and consensual responses in the same two or three exposures of each eye to the penlight beam.

In the presence of an afferent (sensory) defect, the direct and consensual responses will be weakened or absent when a light beam is directed into the affected eye. The severity of the defect can be graded on a clinical scale from 0 to 4, with 0 corresponding to no defect and 4 corresponding to an absence of the appropriate pupillary reflexes.⁸ In the presence of an efferent (motor) defect, the direct response of the affected eye will be weakened or absent, and the consensual response will be attenuated or absent when light is directed at the unaffected eye. Hence, significant anisocoria will exist only with lesions of the efferent system except in rare instances.

Swinging Flashlight Test

The swinging flashlight test compares the strength of the direct pupillary response with that of the consensual (indirect) response and is used to assess afferent (sensory) pupillary defects. The technique is the same as that described for the direct and indirect responses, with the exception that the light beam is alternated from one eye to the other, back and forth, while the practitioner observes the pupil sizes in the two eyes. The penlight is used to illuminate the right eye for 2 to 4 seconds before being quickly moved to illuminate the left eye for another 2 to 4 seconds and is swung back and forth between the eyes in this manner four or five times. The exposures of the eyes must be the same in terms of illumination and duration to avoid false-positive results,¹⁹ and movement of the light beam between the eyes is at a speed that prohibits pupil dilation during the instant that the light beam is not shining into the right or left eyes.

In the normal case, the pupils should remain equally constricted during the swinging flashlight test as the light beam is alternated between the eyes. This is because (1) the light beam is moved quickly back and forth between the eyes so as to prohibit dilation as a result of the interval during which the light beam is

between the eyes, (2) the direct responses should be equal for the two eyes, (3) the consensual responses should be equal for the two eyes, and (4) the direct and consensual responses should be equal within each eye.

If an afferent pupillary defect (APD) is present, the pupils of both eyes will dilate slightly when the affected eye is illuminated. This is known as pupillary escape. The binocular dilation should be observed when the light is alternated to the affected eye, and constriction will again occur when the penlight beam is directed to the unaffected eye. The magnitude of the defect is usually correlated to the rapidity of the escape, and the severity of the defect can be graded on a typical clinical scale from 0 to 4. Neutral density filters can also be used to grade the severity of the defect. A filter is placed in front of the unaffected eye and increased in density until the pupil responses are equal. The density of the filter that produces an equal response between the eyes is taken to be the measure of the defect's relative severity.^{8,20} In unilateral optic neuropathies, the magnitude of the APD correlates with the estimated percentage of retinal ganglion cell loss.²¹

Near Response

If the direct response in each eye is brisk and the constriction is equal relative to the other eye, the near response need not be tested. As noted previously, it is rare for the near reflex to be abnormal when the direct response is intact. However, in the presence of an abnormal direct response, near testing can be of diagnostic importance and should be performed.

In testing the near reflex, the patient is asked to view a distant target in normal room illumination and then to fixate a near target held 25 to 30 cm from the eyes. The illumination of the near target should be the same or similar to the illumination of the distant target such that the light reflex is not confused with the near reflex. Several letters on a near-point card work well as a target. Normally, both pupils will constrict when focusing on the near target, but this may be difficult to observe in those patients having small pupils. The near reflex in these cases may be easier to confirm in a semi-darkened room by observing the red reflex produced with an ophthalmoscope or retinoscope. Indeed, the retinoscopist can assess the stability of accommodation by noting pupil size changes due to accommodative fluctuations and knows if a patient alternates focus from distance to near during the procedure (see Chapter 18). However, care should be taken in the retinoscopic analysis of the near response, that the patient focuses on a near target illuminated only by room light, rather than fixating on the bright light of the retinoscope or ophthalmoscope. If the near response is present in the absence of a direct reflex, it is called a light-near dissociation. The near response can be present even in blind eyes.^{7,12}

Pupil Cycle Time

Periodic oscillations of the pupil can be observed by use of the slit lamp biomicroscope, after introduction of a focused horizontal slit of light approximately 1-mm wide on the iris at the inferior pupillary border. When the light beam is placed such that a small portion of its thickness is focused inside the pupil, the pupil should constrict such that the stationary light beam is excluded from the pupil. With the light excluded, the pupil should then dilate such that a small portion of the beam's thickness is again allowed into the pupil. Hence, the size of the pupil cycles as constriction and dilation repeatedly occur. The pupil cycle time is averaged over 30 cycles, measured with a stopwatch, and is indicative of a pupil anomaly if greater than 954 msec/cycle (just about 1 sec per cycle) for a single pupil or if the difference between the two pupils is greater than 70 msec/cycle.²² Most pupillary reflex anomalies will significantly increase the pupil cycle time.

Recording

If all pupil reflexes are normal, the acronym PERRLA is recorded. PERRLA stands for "pupils equal, round, responsive to light and accommodation." If the near reflex was not tested, the acronym can be shortened to PERRL. One could also record APD-, indicating that no afferent pupillary defect was present. Because APD defects are also called Marcus-Gunn pupils (see later), MG- is often recorded as a substitute for APD-.

If a defect in the pupillary responses was noted, the type of defect, degree of defect, and the eye should be recorded. For instance, an afferent defect in the left eye having a severity of grade 2 would be recorded: APD 2 OS. The mean pupil cycle times, if performed, should be recorded in milliseconds per cycle for each eye.

Pupil Anomalies

Pupil size and reflex anomalies may be secondary to lesions in the afferent (i.e., retina, optic nerve) or efferent (i.e., sympathetic or third cranial nerve) pathways. Many unilateral efferent anomalies of the pupillary reflexes will generate anisocoria, which should be found in the assessment of pupil size at the beginning of the pupillary examination. The more common pupillary reflex anomalies are listed in this section, below, such that the eye care clinician can be looking for them in practice. A flow chart concerning the diagnosis of pupillary anomalies is shown in Figure 10-3.

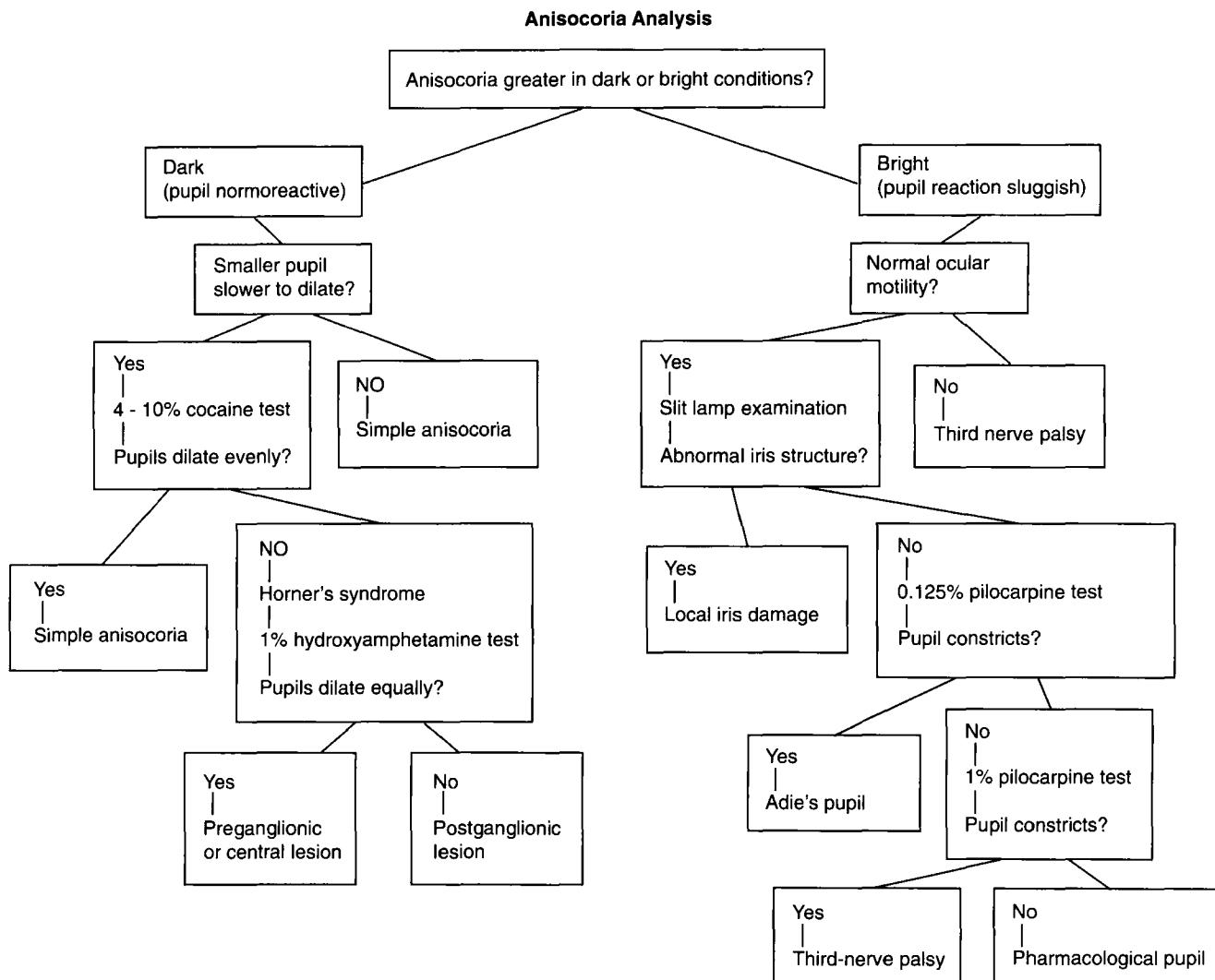
Anisocoria Greater in Bright Conditions

Adie's Tonic Pupil. A relatively common occurrence called Adie's tonic pupil is noted primarily in females in their third and fourth decades of life, yet it has been seen in all age groups and both genders. The incidence is approximately 4.7 per 100,000 in the pop-

ulation per year, and the prevalence is approximately 2 per 1000 in the population. The mean age of onset is 32 years, and the ratio of affected females to males is 2.5:1.^{23,24} These patients appear otherwise healthy but present with a unilateral semi-dilated pupil that responds minimally and slowly (sluggishly) to light. In 10% to 20% of the cases, the fellow eye also becomes involved.^{5,13,25} The unilateral defect is present directly when light is shone into the affected eye and consensually when light is shone into the unaffected eye. The near reflex will also be sluggish for the affected eye, and dilation is slower in the affected eye when switching fixation from near to far. The anisocoria is more pronounced in bright conditions than in dim conditions. These findings suggest the existence of a lesion in the efferent parasympathetic pathway on the side of the semi-dilated pupil, resulting in poor constriction of the corresponding iris sphincter muscle. The denervation often appears secondary to a mild viral infection that adversely impacts the postganglionic fibers at or distal to the ciliary ganglion on the affected side.²⁶ The affected eye's accommodative motor control is likely to be diminished simultaneously. Because the assumed lesion is distal to the deviation of the parasympathetic fibers from the third cranial nerve, there is no involvement of the extraocular muscles.

Although Adie's pupil is not associated with any ocular or nervous system disease that requires treatment,¹² other orbital and ocular conditions should be ruled out in the differential diagnosis. Proptosis or engorged conjunctival vessels, for instance, could indicate a tumor or mass behind the globe that reduces the function of the parasympathetic innervation.^{5,13} Patients often are concerned about the cosmetic appearance of the anisocoria or blurred near vision from the simultaneous involvement of the accommodative fibers. Accommodation often returns within 2 years as efferent fibers regenerate, but the anisocoria persists. This is due to the much larger number of fibers in the ciliary ganglion that are destined for the ciliary muscle (97%) than for the iris (3%).¹¹ The few pupillary fibers become lost among the many accommodative fibers and end up becoming misdirected. Indeed, the pupil often becomes controlled by regenerated accommodative fibers.²⁶ The practitioner should be concerned with a proper accommodative balance (equalization) at distance and at near during the subjective refraction (see Chapter 20) for patients having Adie's pupil. Unequal bifocal add powers may be indicated for presbyopes, a subject further covered in Chapter 20. The refractive error of the affected eye may also become slightly more hyperopic or less myopic.

Many patients with Adie's pupil show absent or reduced deep tendon reflexes, especially in the lower extremities.²⁷ The iris sphincter muscle resulting in an Adie's pupil can become hypersensitive to cholinergic

**Figure 10-3**

A flow chart for diagnosis of anisocoria.

stimulation over time. A drop of only 0.125% pilocarpine, not concentrated enough to influence the normal pupil, will cause 80% of Adie's pupils to constrict. Hence, a drop of 0.125% pilocarpine is thought to be a diagnostic test indicative of Adie's tonic pupil. In the classic literature, 2.5% methacholine hydrochloride (Mecholyl) was used in an identical fashion for the diagnosis of Adie's pupil, but pilocarpine is now generally available, whereas Mecholyl is not. Greater variability in the sensitivity of Adie's patients to methacholine also limited the usefulness of the drug in making an accurate diagnosis.²⁷

Palsy of the Third Cranial Nerve. Anisocoria greater in bright conditions is usually associated with involvement of the extraocular muscles (EOMs) during third cranial nerve palsies. Tumors, aneurysms, bone fragments, and herniated tissue can all compress the oculomotor nerve. Because the pupillary fibers travel

superficially with the third nerve, they tend to be involved early in the compressive process, resulting in a fixed and dilated pupil.^{7,13} Thus, early diagnosis of the pupillary defect can be important in the evaluation and management of an acute third-nerve palsy.¹⁷ With an ischemic lesion, such as in diabetes or arteriosclerosis, the pupil is often spared while the extraocular muscles are adversely affected.¹³ Progressive involvement of functions related to the third cranial nerve may result in accommodative insufficiency, ptosis on the affected side secondary to interruption of innervation to the levator, and exotropia secondary to paralysis of the rectus muscles (excluding the lateral rectus) and the inferior oblique. Hence, the clinician can gain insight relative to the disease progression by evaluation of the pupils, accommodation, eyelids, and any incompatibilities indicating the particular EOMs that are paretic or paralyzed. The corneal blink reflex should be tested to assess the

function of the fifth nerve, whose first and second branches pass alongside the third nerve in the cavernous sinus and through the superior orbital fissure. Orbital auscultation should be done to listen for bruits suggestive of a posterior communicating artery aneurysm.

Anomalous eyelid or eye movements with an abnormal pupil response may occur secondary to aberrant regeneration of oculomotor fibers following a compressive lesion due to trauma, tumor, or aneurysm.¹³ The pupil will most often constrict in downgaze and adduction when aberrant EOM fibers have regenerated. Abnormal accommodation may also be a result.

Pharmacological Pupil. It is simple for an atropinic substance to get on the hands and be rubbed into the eye, and some patients instill eye drops intended for other persons or purposes. A fixed and dilated pupil in an otherwise healthy and unremarkable patient should alert the practitioner to the possibility of pharmacologically induced pupil dilation. Health care workers should be questioned regarding their exposure to possible causative medications, and patients should be asked about use of topical ocular medications, especially those that were originally prescribed for another family member. Gardeners and those with outdoor interests may come into contact with plants leaving atropinic-like residues on the hands. Neurologically enlarged pupils will constrict to an application of 1% pilocarpine, but pharmacologically blocked pupils will not.¹⁷ Hence, the clinician has available a test to differentiate the two basic causes of pupil dilation. However, this test should not be used if an acute neurological lesion is strongly suspected, because the induced miosis leaves the pupil untestable in a potentially rapidly evolving neurological condition.⁷

Anisocoria Greater in Dim Conditions

Horner's Syndrome. Horner's syndrome is the name given to the condition wherein sympathetic innervation to the eye is interrupted, resulting in a miotic pupil with incomplete dilation in darkness. Because the sympathetic system also controls Müller's muscles of the eyelids and the facial sweat glands, slight ptosis and decrease in facial sweating (facial anhydrosis) may occur on the same side as the miotic pupil. Hence, the three hallmark signs of Horner's syndrome are "miosis, ptosis, and anhydrosis" on the affected side.⁷ The condition can be easily missed, because the anisocoria can be small, with less than 1-mm difference between the pupils, a result of the paretic iris dilator muscle being weaker than the sphincter. The ptosis is generally mild, because Müller's muscle is weak and controls only the tonic retraction of the upper eyelid. Faint ptosis of the lower lid may also be present, in which the lower lid rises slightly, but this is difficult or impossible to document because it is smaller in magnitude than even ptosis of the upper lid.

The efferent lesion causing a Horner syndrome can be anywhere in the long sympathetic pathway to the pupil. First-order or central lesions may be due to stroke, multiple sclerosis, cervical spinal cord trauma, syringomyelia, or neoplastic disease of the brain stem or spinal cord. Preganglionic lesions may be located in the thoracic apex or in the neck proximal to the superior cervical ganglion, such as carcinoma of the lung apex (Pancoast's tumor) and neck lesions including those resulting from trauma and thyroid enlargement. Breast carcinoma, lymphadenopathy, and thoracic aneurysms may also result in preganglionic Horner's. Postganglionic lesions may be *extracranial* with similar etiologies listed for preganglionic neck lesions and abnormalities of the internal carotid artery. They may also be *intracranial* from a cavernous sinus or middle cranial fossa lesion or cluster headaches.

The lack of sympathetic innervation in congenital cases of Horner's syndrome may cause heterochromia since the growth of pigmented melanocytic cells is modulated by the sympathetic system.²⁸ Hypopigmentation in the affected eye may occur if the onset of oculosympathetic paresis is before age 2 years.²⁹ Congenital Horner's syndrome is generally benign. Acquired Horner's syndrome can also be benign, as in trauma to the head or neck, or indicate a serious problem, as in a tumor along the sympathetic pupillary pathway or aneurysm of the carotid or subclavian arteries. Trauma is the leading cause of Horner's in patients under 20 years. In patients aged 21 to 50, however, tumors are the cause in almost half the cases. Neoplasms are an important etiology in the over 50-year age group as well.³⁰

As a general rule, the postganglionic lesions are benign and the preganglionic lesions are indicative of a serious problem.^{5,12} Hence, it is important to be able to differentiate between the two. Postganglionic lesions do not generally cause facial anhydrosis, because the sympathetic fibers supplying the sweat glands split from those innervating the iris and Müller's muscle at the carotid bifurcation.¹² A history of endarterectomy, head trauma, or thyroidectomy suggests a postganglionic problem. Cluster headaches are also associated with postganglionic lesions. Auscultation of the neck and testing of corneal and facial sensitivity should take place, because the sympathetic path closely follows the carotid artery and first division of the 5th cranial (trigeminal) nerve.

Some experts advocate the topical use of 4% to 10% cocaine in the diagnosis of Horner's syndrome.^{31,32} Cocaine prevents the reuptake of norepinephrine at the sympathetic neuromuscular junctions. Thus, the normal pupil will dilate in response to cocaine because its sympathetic innervation is capable of maintaining endogenous levels of norepinephrine at the neuromuscular junctions. Blockage of reuptake increases the concentration of norepinephrine at the neuromuscular junc-

tions, resulting in the dilation. However, most affected eyes in Horner's syndrome show minimal or no pupillary dilation to these concentrations of cocaine. In the presence of a paretic or paralyzed efferent sympathetic path, norepinephrine is not released sufficiently on the palsied side for a significant accumulation to occur even when reuptake is blocked. Cocaine is difficult to obtain and keep fresh. The pupillary reaction to cocaine, although it may help diagnose the Horner's syndrome, does not differentiate between a preganglionic or postganglionic lesion.³²

To distinguish between a postganglionic lesion and a preganglionic/central lesion, 1% hydroxyamphetamine (Paredrine) can be applied topically to each eye. An indirect-acting sympathomimetic agent, the release of norepinephrine will produce mydriasis in the unaffected eye but not in the eye affected by a postganglionic lesion.³³ This is because the fibers involved in the postganglionic lesion will have degenerated and be incapable of producing norepinephrine. However, dilation will occur in an eye affected by a preganglionic or central lesion, for the postganglionic fibers will be intact. The pneumonic device "Fail-Safe" may be used to remember that if the Horner's pupil "fails" to dilate with hydroxyamphetamine the patient is "safe" because the lesion is postganglionic and likely to be benign.³⁴

Anisocoria Not Present

Marcus-Gunn Pupil (Afferent Pupillary Defect). As noted earlier, an afferent pupillary defect causes less constriction in both the affected eye, due to a reduced direct reflex, and the unaffected eye, due to a deficient consensual reflex, when only the affected eye is illuminated. Both eyes constrict when the light beam is directed into the unaffected eye. The cause of a Marcus-Gunn pupil is generally optic nerve disease, such as neuritis or atrophy, which causes a defect in the afferent pathway in any position from the retinal ganglion cells to the pretectal area of the hypothalamus.¹² The defect diminishes the visual signal and pupillary reflex to light. Anisocoria is not present under the normal circumstances when the two eyes are equally illuminated. Afferent pupillary defects are generally unilateral.^{13,25}

Optic nerve lesions secondary to inflammatory, demyelinating, neoplastic, nutritional, or toxic conditions are possible causes of a Marcus-Gunn pupil.^{5,12,13} Extensive retinal lesions can also produce the effect but should be easy to identify as the cause by ophthalmoscopy.¹³ Postgeniculate lesions do not cause the problem, although a chiasmal lesion may show a Marcus-Gunn pupil if one eye is more affected than the other.^{5,25} The visual acuity in the affected eye may be markedly decreased, slightly decreased, or unchanged.⁵ Afferent pupillary defects are not due to refractive errors or malingering.

Amaurotic Pupil. An amaurotic pupil occurs in an eye with no light perception. The direct reflex will be absent in the affected eye but its pupil contracts because of consensual reflex when the unaffected eye is illuminated. The unaffected eye will demonstrate a direct reflex but no consensual response when the affected eye is illuminated. Both eyes will constrict to a near target. Anisocoria will not be present under normal circumstances when the two eyes are equally illuminated. The amaurotic pupil is essentially a severe afferent pupillary defect.

Light-near Dissociation

Pupils that fail to constrict to light but demonstrate a near reflex are said to have a light-near dissociation. Previously, this was frequently seen in neurosyphilis, where it was associated with the bilateral Argyll Robertson pupil, discussed separately later. A true light-near dissociation can be seen in afferent pupillary defects and amaurotic pupils, which adversely influence the light reflex but leave the efferent pathways intact. Certain lesions of the midbrain including Parinaud's syndrome affect the initial motor control of the light reflex but allow slightly more distal input for the near response through otherwise intact efferent paths.^{7,13} The Argyll Robertson pupil is likely a type of a midbrain lesion resulting in a light-near dissociation. It is important to note that the near response in a light-near dissociation is present bilaterally, even if the pupillary defect in the light response is unilateral or bilateral.

It is difficult to understand how a light-near dissociation, having a bilateral near reflex, could appear in cases of efferent pupillary defects. One would expect the near response to be unilateral: diminished or absent on the affected side. Sometimes, however, a false light-near dissociation will present on the affected side when the "near response" is accomplished through aberrant regenerated nerve fibers to the iris musculature. This often occurs in lesions of the third nerve, when fibers originally destined for the medial rectus aberrantly innervate the iris sphincter, and in lesions of the ciliary ganglion (Adie's pupil), when fibers originally destined for the ciliary muscle aberrantly innervate the iris sphincter. Light-near dissociations have been reported with several types of motor neuropathies and are most likely related to aberrant regenerations.¹⁹

Midbrain Lesions

If both pupils show little or no response to light, bilateral Adie's pupils should be considered. In these instances, the patient's vision would likely be decreased at near and the pupil cycle time large. A myopathy or neuropathy in the midbrain that affects the pretectal synapses can also present this bilateral condition, but visual acuity will be unaffected.¹² Usually the near reflex is present because the light reflex fibers in the tegmen-

tum of the midbrain are located dorsal to the near reflex fibers. A variety of central nervous system conditions infrequently cause light-near dissociations, including diabetes, chronic alcoholism, encephalitis, multiple sclerosis, central nervous system degenerative disease, and tumors of the midbrain.

Pinealomas, other tumors, trauma, infection, infarction, and arteriovenous malformations are causes of Parinaud's ophthalmoplegia, also called the Sylvian aqueduct syndrome, because the lesions are in the periaqueductal area of the midbrain. Voluntary, conjugate, upward movements of the eyes are paretic or paralyzed. Lid retraction (Collier's sign) may also manifest. The pupils are sometimes semi-dilated as a result of loss of the light reflexes, with near responses intact.^{12,13}

The classic midbrain lesion resulting in abnormal pupil reflexes is well known as the Argyll Robertson pupil. Rather than presenting with a semi-dilated pupil or pupils, the eyes are almost always bilaterally miotic, irregular, and difficult to dilate. Simultaneously, the pupillary light reactions, direct and consensual, are absent or sluggish. The classic cause of the Argyll Robertson pupil was neurosyphilis; however, this is now not as prevalent. The cause is now as likely to be general neuropathy related to diabetes or alcoholism.¹³ Loewenfeld³⁵ concluded that the causative lesion is in the rostral midbrain near the aqueduct of Sylvius, interrupting the synapses of supranuclear inhibitory fibers with the light reflex fibers as they approach the Edinger-Westphal nucleus. Because of the relative lack of inhibition from higher centers, the pupils become bilaterally constricted and relatively unresponsive to light.

THE PALPEBRAL APERTURES AND EYELID MOVEMENTS

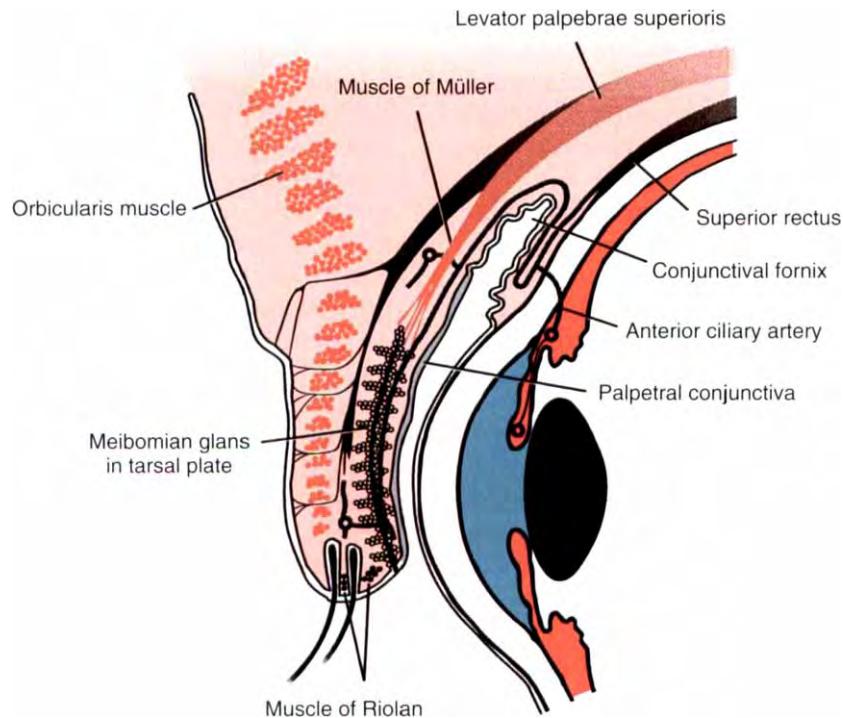
The eyelids protect the eyes and distribute the tear film to moisten the ocular surface and maintain the cornea's optical surface quality. Abnormal innervation of the musculature of the eyelids can cause visual impairment by an inability to provide excellent surface optics over the pupillary area, inability to keep all of the interpalpebral space adequately wet or moistened, inability to retract the upper eyelids such that all or part of the pupil is uncovered, or inability to blink at the appropriate times so as not to interfere with vision. Many of the aspects of an evaluation of the tear film and eyelids are concerned with the physiology of the ocular surface and logically fall into the external examination or biomicroscopy (see Chapter 13) or into the province of contact lens practice. Here, abnormal aperture size and eyelid movements resulting from neuromuscular eyelid disorders will be discussed, such that the examiner can recognize and manage these neuromuscular problems.

The position and movement of the eyelids are controlled by three separate neuromuscular systems actuating the levator palpebrae superioris, the muscle of Müller, and the orbicularis oculi. The levator palpebrae superioris is a muscle in the superior orbit that extends into the upper eyelid. The levator's tendon inserts into a large area of the skin of the upper eyelid, and some of its fibers insert into the anterior surface of the tarsal plate,³⁶ as shown in Figure 10-4. The levator is the primary muscle responsible for retraction of the upper lid following the blink and in upgaze. The fold near the top of the upper lid marks the superior boundary of the insertion of the levator into the skin covering the eyelid. This fold is not present in oriental eyelids, because the levator's tendon does not insert as completely into the overlying skin.

The facial sheath of the levator is common to that of the superior rectus muscle (see Figure 10-4), and its neural input is coordinated with that of the superior rectus, such that upgaze produces simultaneous elevation of the upper lid. The frontalis muscles of the brow help the levators to elevate the upper lids in extreme upgaze. When a person forcibly closes the eyelids, as in blepharospasm, the eyes rotate bilaterally upwards. "Bell's phenomenon" is present in 90% of persons and believed to be a protective mechanism that brings the cornea underneath the upper lid and away from potential sources of injury.³⁶ Bell's phenomenon can be observed by holding the lids open while the subject attempts to forcibly close the eyes.

The levator is supplied by the superior branch of the third cranial (oculomotor) nerve, which also innervates the iris sphincter, ciliary muscle, and four of the six EOMs on the same side. Both oculomotor nerves are supplied by fibers destined for the levator from a single nucleus located on the dorsal aspect of the oculomotor nuclear group in the mesencephalon.³⁷ Hence, the two levator muscles, one in each upper eyelid, are activated together to achieve simultaneous retractions after the blink and in upgaze.³⁸

The muscles of Müller receive innervation from branches of the sympathetic nervous system, whose course was described earlier in this chapter for the pupillary dilator muscle and facial sweat glands. There are actually four Müller's muscles: one smooth muscle for each eyelid. The muscle of Müller in a superior eyelid is anchored into the inferior facial surface of the levator and inserts into the upper edge of the superior tarsal plate.³⁸ Contraction of these muscle fibers retracts the superior tarsus and, therefore, moves the superior eyelid upward. In an inferior eyelid, the muscle of Müller is anchored into the upper facial surface of the inferior rectus and inserts into two places: the lower edge of the inferior tarsal plate and the conjunctival fornix. Contraction of these muscle fibers retracts the inferior eyelid downward. Being of smooth muscle and of relatively

**Figure 10-4**

Diagrammatic cross section of the upper eyelid. Note the insertion of the levator into the skin of the upper eyelid by fibers that pass through the orbicularis. Fibers inserting into the anterior surface of the tarsal plate are not shown.

consistent activity, the muscles of Müller supply a tonus to the open-eye retractions of the upper and lower eyelids.

The orbicularis oculi is the primary muscle that closes the eyelids. There are two major divisions of the orbicularis: the palpebral portion and the orbital portion. The palpebral portion of the orbicularis covers the tarsal plates, yet lies below the outer surfaces of the upper and lower eyelids, and extends from the eyelid margins to the orbital rim. The physiology of the palpebral orbicularis is suited for rapid movement and acceleration. It is responsible for involuntary eyelid closure during the blink and voluntary eyelid closure as in winking.³⁶ A small specialized portion of the palpebral orbicularis is called the muscle of Riolan, which is located in the margins of the eyelids and thought to help keep the margins in apposition with the ocular surface. During forced eyelid closure, as occurs in winking or blepharospasm, the orbital portion of the orbicularis and eyebrow muscles come progressively into action, depending on the force of closure involved. Another small specialized portion of the orbicularis ensnares the nasal lacrimal sac. Upon eye closure, as in blinking, this sprig of the orbicularis squeezes the sac, emptying its contents into the nasal passage. Upon eye opening, this unnamed portion of the orbicularis releases, allowing

the lacrimal sac to take in tear fluid via the canalicular/punctal drainage system.³⁹

The orbicularis is innervated by fibers from the seventh cranial (facial) nerve, which originates in the pons. The nerve enters the internal auditory canal and then passes through the facial canal in the petrous portion of the temporal bone, emerging through the stylomastoid foramen, which is inferior to and slightly posterior to the external auditory opening.⁴⁰ The two facial nerves also innervate the muscles of the corresponding sides of the face. The routes and branches of the facial nerves are highly variable among persons, such that certain people can "wiggle" one or both ears, or raise one or both eyebrows individually, whereas others cannot. Hence, some patients can voluntarily wink or close each eye by itself (monocularly), others can wink or close only one of the eyes by itself, yet a few are unable to voluntarily close or wink either eye by itself. In all cases, however, the normal patient should be able to voluntarily blink or close both eyes at the same time.

The palpebral aperture, or fissure, is 27 to 30 mm in length (horizontally) and 8- to 11-mm wide (vertically) at its widest point in adults, which is usually nasal of center by 1 to 4 mm, creating an "almond" shape. In Asians, the aperture may not be quite as wide, although

the characteristic shape is retained. In children the aperture is not as long and is relatively wider, compared with its length, whereas in infants the aperture can be nearly circular. The normal width of the palpebral aperture is the result of a competitive equilibrium of muscle tonus between the orbicularis, acting to lessen the fissure, and the combined tonus of the levator and muscles of Müller, acting to widen the fissure. The adult palpebral aperture can be made approximately 15 mm wide by voluntary lid retraction of the levator and maximally 17 or 18 mm wide by simultaneous action of the frontalis muscle of the eyebrow.³⁶

The upper eyelid normally covers the superior cornea from approximately the 10 o'clock to the 2 o'clock positions. It covers a mean of 2.1 mm of the superior cornea in Caucasians (± 0.9 mm), perhaps more in Asians representing 7% of the corneal surface area.⁴¹ In most persons the lower eyelid margin will reside at or below the lower limbus by 1 or 2 mm. In a few persons, the upper eyelid margin will normally reside at the superior limbus or 1 to 2 mm above, the inferior eyelid margin will cover a small portion (1–2 mm) of the inferior cornea, or both. It is important that the clinician assess the geometry of the patient's palpebral apertures in order to recognize the abnormal from the normal, so that underlying neuromuscular deficits can be detected.

Clinical Evaluation

In routine examinations, the width of the palpebral aperture is not actually measured, although it is assessed qualitatively by the clinician. The aperture sizes of the two eyes are compared with each other, and the anatomical locations of the upper and lower lid margins are noted relative to the corneal limbus of each eye. Should the widths of the palpebral apertures require documentation, the patient is asked to fixate a distant target under normal room illumination, and a millimeter rule is positioned vertically at the aperture's widest extent. The distance between the upper and lower lid margins is measured for the right and left eyes. Similarly, the positions of the eyelid margins with respect to the upper and lower extents of the corneal limbus can be measured.

Perfect symmetry of the right and left palpebral apertures exists only for a few patients. Typically, one eye will have a slightly wider aperture than the other, and the eyelid margins will intersect the upper and lower limbus at positions that are slightly different for the two eyes. A difference of 2 or more millimeters between the widths of the palpebral apertures is suggestive of unilateral ptosis, which results from inferior positioning of one upper lid relative to the other, superior positioning of one lower lid relative to the other, or both. However, ptosis can also be bilateral, in which both upper lids or both lower lids are insufficiently retracted. The eyelids

may also appear to be overly retracted or widened, as is common in thyroid disease.

When one or both palpebral apertures appear malformed, or a difference in lid positions is noted between the eyes, it is important to question the patient about the asymmetry without suggesting or implying initially that the appearance of the eyes is abnormal. The onset, progression over time, and variation of the asymmetry with certain actions (e.g., upgaze, blinking, eye closure) are of particular interest. Ptosis can often be observed in old photographs of the patient. The patient may be instructed to follow, with the eyes, the clinician's finger into upgaze and downgaze while the clinician observes the intersection of the eyelid margins with the corneas. In this manner the clinician may assess whether the asymmetry becomes greater or lesser in upgaze or downgaze as compared with primary gaze. The clinician notes the completeness of the blink in each eye and of voluntary eyelid closure. Blinks and voluntary closure should be assessed bilaterally because, as noted earlier, it is not possible for some persons to voluntarily wink or close an eye monocularly.

Recording

There is no standardized system for the recording of palpebral aperture widths, eyelid margin locations, or palpebral abnormalities. Indeed, these are usually recorded only if the practitioner notices an abnormality of the palpebral aperture. Documentation of the palpebral aperture width is merely a recording of the widths for the right and left eyes. The positions of the eyelid margins can be recorded relative to the corneal limbus, with positive numbers indicating coverage of the cornea and zero indicating the margin at the limbus. For instance, assume that the palpebral aperture widths are 12 mm in the right eye and 9 mm in the left eye; the upper lid margins are overlapping onto the cornea by 1 mm in the right eye and 3 mm in the left eye; and the lower lid margins are 1 mm below the lower limbus in the right eye and at the lower limbus in the left eye. A recording could be: R 12/+1/-1 mm, L 9/+3/0 mm. The clinician might also note if, for instance, ptosis exists in the left eye, proptosis in the right eye, or that the eyes are normally asymmetric to this degree, whichever is determined.

Neuromuscular Palpebral Anomalies

Ptosis

The most common neuromuscular abnormality of the palpebral aperture is ptosis, which generally manifests as an abnormal location of the superior eyelid. Ptosis of the superior lids can be documented by the extent of upper lid overhang onto the cornea in both eyes, as noted earlier, and graded using a simple 0 to 4 clinical scale of severity. Gravity usually works in favor of ptosis

of the upper eyelids. In the extreme, the low position of an upper lid can occlude all (grade 4) or part of (grade 3) the pupil, and the lid may not be retractable. This could be caused by a lesion of the levator's innervation or interruption of its function, as commonly occurs with a marked upper eyelid inflammation. The ptotic upper eyelid may not occlude any of the pupil (grade 2), and mild ptosis (grade 1) can be difficult to discern from normal asymmetry. This could be the result of a lesion that partially blocks the sympathetic innervation to the superior muscles of Müller or a small upper eyelid inflammation.

A very common cause of ptosis is an eyelid inflammation of microbial, allergenic, or traumatic nature. An inverse ptosis, also called an upside-down ptosis, is an elevation of the lower lid as a result of lower lid inflammation or interruption of innervation to the inferior muscles of Müller. Inverse ptosis is usually mild and is generally difficult to recognize, because gravity works in favor of retraction of the inferior eyelid. The positions of the lower lids can be documented relative to the lower corneal limbus as noted earlier. A mild (grade 1 or 2) to moderate (grade 3) bilateral ptosis often occurs in the aged as a result of disinsertion of the levator or reduction of retrobulbar orbital fat. The enophthalmos may bring about increased coverage of the globe by the superior and inferior eyelids. Most cases of ptosis will be more evident when the patient is sleepy or fatigued, and this can help in the diagnosis of the milder forms (grades 1 and 2). Unilateral or bilateral ptosis should be evaluated with consideration given to the pupillary examination, the function of the EOMs, and other clinical neurological signs.

Dysfunction of the Levator Palpebrae Superioris. Dysfunction of the levator can be caused by a lesion of the oculomotor nerve or by restriction of the levator's function. Head trauma, tumors, aneurysms, and thrombosis of the cavernous sinus can result in lesions of the oculomotor nerve, which are often accompanied by involvement of the EOMs supplied by the oculomotor nerve (exotropia) and an ipsilateral dilated pupil with accommodative involvement (efferent pupillary defect). Ptosis of recent onset is usually caused by an oculomotor nerve lesion and is accompanied by diplopia if the lid does not completely occlude the pupil. Mechanical restriction can result from excessive pressure on the lid from tumors or inflammation, or scar tissue can interfere with lid retraction. Trauma of the eyelid may break some or many of the tendinous fibers inserting into the eyelid, as might occur resulting in postoperative ptosis (usually unilateral). The insertion of the levator may become less effective with aging, resulting in senile ptosis (usually bilateral). Myogenic defects are caused by impaired function of the muscle or the myoneural junction as occurs in congenital ptosis, myotonic dystrophy, and myasthenia gravis.

Ptosis involving the motor route to the levator will generally be unilateral. The eyelid does not retract completely after a blink or in upgaze. Hence, the ptosis will appear to be of greater magnitude in upgaze. The motor nuclei of the superior divisions of the 3rd cranial nerves are located dorsally in the midbrain, and damage there results in bilateral ptosis. An unaffected pupil with ptosis may occur in diabetic neuropathy, accompanied by a history of diabetes, and myasthenia gravis, accompanied by complaints of unusual fatigue. The ptosis can be increased with fatigue in myasthenia gravis, and fatigue of the levator may be elicited by having the patient hold the eyes in upgaze for several minutes. The superior muscle of Müller cannot compensate for a paretic levator because the smooth muscle is anchored on the underside of the levator, which is not able to supply much support in its paretic state.⁴¹

Dysfunction of Müller's Muscle. Horner's syndrome was described earlier, in which the sympathetic pathway to the ipsilateral pupil, muscles of Müller, and facial sweat glands was interrupted. Ptosis in this syndrome is primarily the result of the paresis or paralysis of the superior muscle of Müller. Much lid retraction after the blink and in upgaze is intact, because the levator is unaffected. The ptosis does not increase in upgaze. Ptosis in Horner's syndrome is generally not as pronounced as that occurring with paralysis of the levator. In addition, the EOMs are unaffected, the ipsilateral pupil is miotic, and anhydrosis may be present on the affected side of the face.

Eyelid Retraction

When the eye is in primary gaze, the visibility of sclera between the limbus and upper lid margin may indicate the presence of an eyelid retraction. It is common for 1 to 2 mm of sclera to show between the lower eyelid margin and the limbus. As will be noted later, a slight eyelid retraction may be apparent with a 7th cranial nerve palsy. However, eyelid retraction is most commonly due to thyroid disease or midbrain lesions. It can also be the result of surgical overcorrection of ptosis, scarring from eyelid trauma, or tumors. Aberrant regeneration of nerve fibers to the levator could be one cause of the Marcus-Gunn phenomenon (jaw-winking), for which the upper eyelid unilaterally retracts when the mouth is opened, and the pseudo-Graefe phenomenon, for which the upper lid retracts upon downgaze.⁴³ Globe displacement or enlargement (as seen in axial myopia or congenital glaucoma), the use of topical sympathomimetics, or high doses of systemic steroids can produce eyelid retraction.³⁸ In addition to identifying the etiology of retraction, the clinician should monitor for a resulting exposure keratitis, which may require treatment with topical lubricants or surgical correction.

Thyroid Disease. Thyroid disease is the most common cause of lid retraction and may be present in

hyper- or hypothyroidism. Euthyroid, wherein the ocular signs are apparent in the presence of normal thyroid function tests, is also a frequent cause. Lid lag in downgaze (Graefe's sign), a staring appearance (Dalmatian's sign), and infrequent and incomplete blinking (Stellwag's sign) often occur with retraction in Grave's disease. Although ocular effects from thyroid dysfunction are generally observed bilaterally, it is not uncommon for the signs to present asymmetrically and appear as a unilateral lid retraction. In hyperthyroidism the lids often return to normal after medical treatment of the thyroid condition, but usually the lid retraction in euthyroid persists if it has been present for a year or more.⁴⁴ Ocular lubricants are often necessary for the treatment of the resulting dry eye, but any surgical correction of the lids should wait until the thyroid condition is stable.

Midbrain Disease. Lesions of the posterior third ventricle (Parinaud's ophthalmoplegia) may manifest Collier's sign, a staring appearance caused by bilateral lid retraction. Conjugate, upward movement of the eyes is restricted, and a convergence-retraction nystagmus is elicited on attempted upgaze. Abnormal pupils (light-near dissociation) are also present in Parinaud's ophthalmoplegia. Etiologies range from hydrocephalus and pinealoma in infants and teens to arteriovenous malformations, tumors, and basilar artery disease in adults.

Dysfunction of the Orbicularis Oculi. Paresis of the orbicularis may cause ineffective or incomplete lid closure, whereas outright paralysis results in no blink whatsoever. Served by the 7th cranial (facial) nerve, some or all of the facial muscles of the cheek and mouth likely will be affected by paresis or paralysis, causing the patient to lose ipsilateral facial expression. The simultaneous influence on the muscle of Riolan leaves eyelid apposition to the globe chronically affected by gravity. The upper lid may remain loosely apposed to the ocular surface and perhaps slightly retracted⁴⁰ because of the loss of orbicularis tone and the unopposed tonus of the levator and superior muscle of Müller. The lower lid may manifest ectropion. In the aged, a loss of tonus in the orbicularis is common and may contribute to incomplete blinking, lagophthalmos, and marked ectropion of the lower lid with epiphora.

A 7th cranial (facial) nerve palsy is usually unilateral, because the lesion occurs in the peripheral nerve instead of encompassing both nuclei in the pons. Bell's phenomenon, noted earlier in this chapter, will be intact in the preponderance of these cases but will be absent if the lesion damages a 7th nerve nucleus. A common paresis of the 7th cranial nerve is Bell's palsy, which is actually of unknown etiology, but inflammation around the 7th nerve inside the facial canal and trauma at the opening of the stylomastoid foramen are two suspected causes.⁴⁰

Idiosyncratic Eyelid Motions

When assessing asymmetrical aperture fissures, it is important to consider that an apparent unilateral ptosis may actually be a contralateral proptosis, lid retraction, or slack lower lid caused by a weak orbicularis muscle. Ptosis can also induce a pseudo-lid retraction in the fellow eye as the patient uses the brows to raise the upper eyelid on the affected side. Because there are equal innervations to the upper eyelids from a single nucleus, a forcible attempt to raise the affected upper lid may cause the other upper lid to rise excessively. Raised eyebrows or furrowing of the forehead indicate this maneuver.

MONOCULAR AND BINOCULAR EYE MOVEMENTS

The purpose of eye movements, actually rotations of the eye, is to initiate and maintain foveal fixation. Vertical gaze and lateral (horizontal) gaze direct the lines of sight in object space along the Y and X axes, respectively, such that combinations of vertical and lateral conjugate eye movements (or rotations) result in direction of the eyes toward targets within any of the four quadrants. Eye movements direct the lines of sight up, down, right, or left away from the primary gaze position (straight ahead). Conjugate eye movements (versions) are those in which both eyes rotate simultaneously in the same direction by equal amounts. Vergence (disconjugate) eye movements, covered in the next section of this chapter, rotate the eyes in opposite directions so as to align the eyes along the anteroposterior Z axis. Hence, the eyes can be directed toward objects located in three-dimensional space in front of the eyes by a combination of conjugate and vergence eye movements. All reflexive and voluntary eye movements are hierarchically controlled by a cortical network that involves the frontal, parietal, and occipital areas⁴⁵ that send diverse premotor signals to the nuclei of the third, fourth, and sixth cranial nerves. Generally, reflexive eye movements originate in the posterior parts of the brain and voluntary movements from frontal areas. Structures involved in horizontal gaze generation occupy the lower pons and upper medulla, and those structures important for vertical gaze reside in the rostral midbrain.⁴⁶

Torsion (torsional eye movement) twists the eyes clockwise or counterclockwise as viewed by the clinician from the front: *encyclorotation (intorsion)* is the term applied when the top of the eye rotates toward the nose, and *excyclorotation (extorsion)* is the term applied when the top of the eye rotates away from the nose. Conjugate torsion twists the eyes in the same direction, clockwise or counterclockwise in both eyes, when the head is tilted to the right or left. Vergence or disconju-

gate torsions occur in opposing directions, intorsion or extorsion in both eyes. Both types of torsional movements are necessary to maintain alignment of the meridians of the two eyes for single binocular vision.

Conjugate eye movements can be tested to determine if the neuromuscular systems controlling the movements are intact and functioning properly. The signal for ocular movements originates in the cerebral hemispheres and is transmitted to the gaze centers in the midbrain and motor nuclei in the pons. From there, the information travels through the 3rd (oculomotor), 4th (trochlear), and 6th (abducens) cranial nerves to supply the EOMs. Supranuclear neuronal pathways conduct impulses to the gaze centers, and internuclear pathways coordinate the gaze centers with the motor nuclei. The infranuclear pathways lie in the individual cranial nerves.

Abnormal conjugate eye movements can be used to help discern whether a lesion involves one or more of the three cranial nerves on each side (the infranuclear paths) or is located at the motor nuclei in the midbrain and pons, the gaze centers in the upper midbrain, or the cerebral centers where the eye movements are initiated. The internuclear or supranuclear pathways may also become dysfunctional. As in many other areas of the general eye examination, the degree of complexity becomes ever greater as more specificity is required and as more specialty topics are covered. Ocular motility can become a specialty by itself when taken to the "nth degree," wherein the border between the roles of a neurologist and an eye care practitioner is indistinct.

However, we will limit this section of our chapter to a screening for conjugate eye movement defects in the initial phases of the routine eye examination.

Monocular Eye Movements

Each eye is suspended within the bony orbit by six EOMs and a complicated system of connective tissue extending from the orbital apex, posteriorly, to the orbital rim, anteriorly. The connective tissue consists of ligaments, septa, and sheaths of the EOMs. The rectus muscles and their intermuscular septa form a "muscle cone," in which the space is filled with the optic nerve, ophthalmic artery, blood vessels to the EOMs, nerves to the EOMs, and the ciliary ganglion. The remainder of the space is filled with orbital fat. The connective tissues, fat, and the extraocular muscles actually form a larger structure that surrounds the globe, dampening movement of the eye and acting as a "fluid brake" for smooth, quick completion of eye rotations. The widest part of the orbit is located 15 mm behind the orbital rim, corresponding roughly to the position of the widest diameter of the muscle cone situated within (Figure 10-5). Hence, the orbital space has been described as being in the shape of a pear.⁴⁷

The EOMs are arranged in three planes of action, each containing a cooperative pair of muscles that act together to control rotations of the globe within the respective planes. With the exception of the superior oblique (see later), the planes contain the midpoints of the origins and scleral insertions of the respective pair

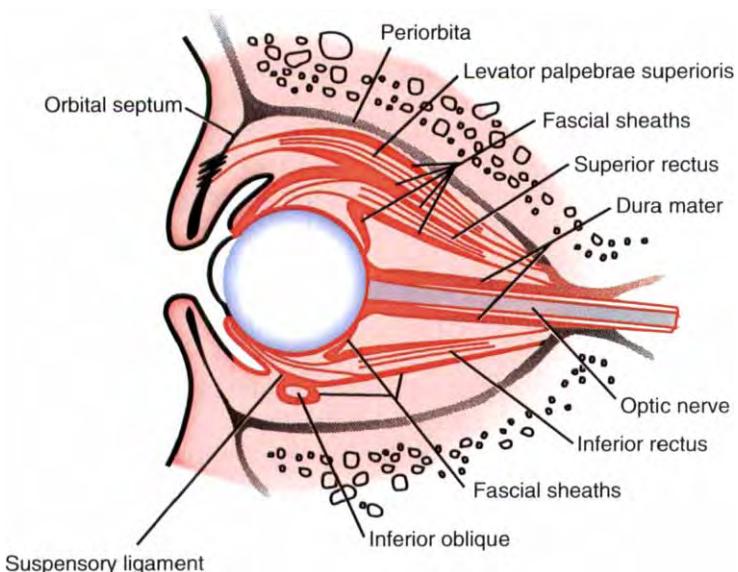


Figure 10-5

Diagrammatic sagittal cross section through the center of the globe and orbit. Note the pear-like shape of the bony orbit, the fascial connection between the levator and superior rectus, and the fascial connection of the inferior rectus and inferior oblique.

of muscles and the longitudinal axes of the muscle fibers. The lateral and medial recti are located in the horizontal plane. When the line of sight of the eye is in the horizontal plane, the actions of the lateral and medial rectus muscles are to direct the line of sight to the left or right within the horizontal plane. The superior and inferior recti are located in a vertical plane that intersects the line of sight in primary gaze (straight ahead) at an angle of 23 to 25 degrees (Figure 10-6). When the line of sight of the eye is 23 to 25 degrees temporal to that of the primary gaze position, the actions of the superior and inferior rectus muscles are to direct the line of sight up or down, respectively, within the plane of the muscles. The superior and inferior oblique muscles act in a vertical plane that intersects the primary line of sight at an angle of 51 to 53 degrees (Figure 10-7). When the line of sight of the eye is 51 to 53 degrees nasal to that of the primary gaze position, the actions of the superior and inferior oblique muscles are to direct the line of sight down or up, respectively, within the plane of action.⁴⁷

An important concept in ocular motility is that a paretic or paralyzed EOM will always have its greatest adverse effect when the line of sight is directed into the

muscle's primary action within its plane of action. Hence, the rotation of an eye will lag farthest behind that wanted or required to fixate a target when the line of sight of the eye is made to lie in the paretic muscle's plane of action, and the patient is asked to then direct the eye into the muscle's primary action. For instance, a paretic lateral rectus in the right eye will be the most obvious when the eye is directed along the horizontal to the patient's right. A paretic superior rectus in the left eye will be most obvious when the gaze is shifted 23 to 25 degrees to the patient's left and then in upgaze. When the line of sight is outside of a muscle's plane of action, the actions of the EOM become more complicated, as will be explained.

Rectus Muscles

The lateral, medial, superior, and inferior rectus muscles are anchored at the apex of the orbit in a thickened annular portion of the periosteum called the circle of Zinn and insert into the sclera anterior to the equator of the globe and posterior to the limbus. The insertion of the medial rectus is 5.5 mm from the limbus. The inferior rectus inserts 6.5 mm from the limbus, the lateral rectus 6.9 mm from the limbus, and the superior

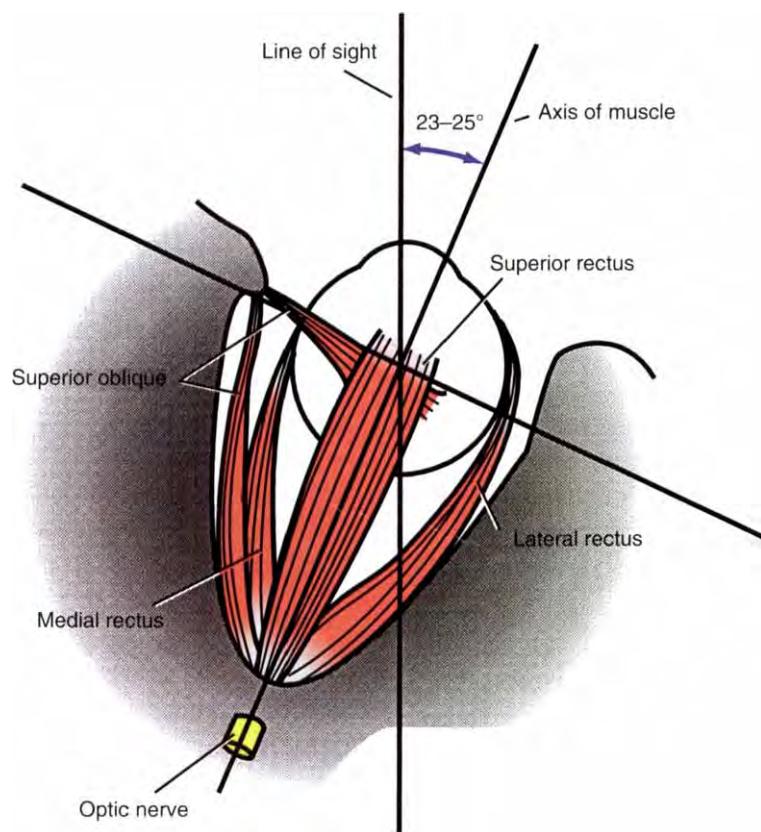


Figure 10-6

Diagram of the top of the eye, from above, showing the origin, insertion, and longitudinal axis of the superior rectus muscle, which lie in the same vertical plane as those of the inferior rectus muscle (not shown).

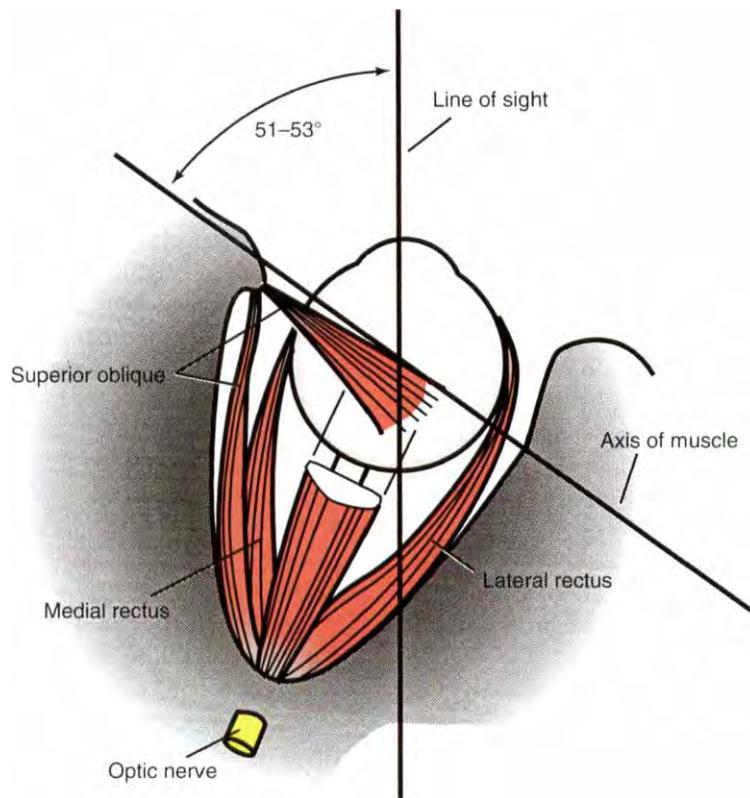
**Figure 10-7**

Diagram of the top of the eye, from above, showing the redirection of the superior oblique muscle at the trochlea, its insertion, and longitudinal axis. The superior oblique from the trochlea to the midpoint of the scleral insertion lies in the same vertical plane as the entire inferior oblique (not shown).

rectus 7.7 mm from the limbus (Figure 10-8). An imaginary spiral formed around the corneal limbus by connecting the insertions of the medial, inferior, lateral, and superior rectus muscles is called the spiral of Tilleaux.⁴⁷ A lateral check ligament is connected anteriorly to the muscle sheath of the lateral rectus and is anchored to the zygomatic bone. A medial check ligament is connected similarly to the medial rectus and is anchored to the nasal bone. The lateral and medial check ligaments limit the nasal and temporal rotations of the eye, respectively, in extreme positions of horizontal gaze. Along with the insertion of the four rectus muscles and the superior oblique at the orbital apex, the check ligaments prohibit the globe from moving forward outside of the orbit. These ligaments have no effect on normal rotations of the eye except for the limitation in extreme lateral gaze. Simultaneous contraction of all of the rectus muscles can result in retraction of the globe and apparent exophthalmos.

The lateral rectus muscle lies in the horizontal plane and is aligned with the middle of the globe as viewed from the temporal side. The lateral rectus is innervated by the 6th cranial (abducens) nerve; contraction of the lateral rectus results in temporal rotation of the globe

(abduction). The medial rectus also lies in the horizontal plane and aligns with the middle of the globe. The medial rectus is innervated by the inferior division of the 3rd cranial (oculomotor) nerve; contraction of the medial rectus results in nasal rotation of the globe (adduction). The innervations of the two muscles are coordinated, such that one is inhibited while the other is active, thus directing component rotations of the eye in the horizontal plane. It is important to note that, under ordinary circumstances in primary gaze, actions of the medial and lateral recti muscles do not result in torsion of the globe or in vertical eye rotation. However, when the eye is directed upward, contractions of the medial and lateral recti help slightly to elevate the eye; in downgaze, these rectus muscles help to slightly depress the eyes. This is because, as noted earlier, the insertions of the EOMs are anterior to the equator of the globe. The torsional movements of the eyes caused by the lateral and medial recti in upgaze and downgaze appear to be subclinical.

The superior rectus muscle is in a vertical plane having an angle of approximately 23 to 25 degrees with the line of sight in primary gaze (see Figure 10-6). Its anchorage is medial to the center of rotation of the eye,

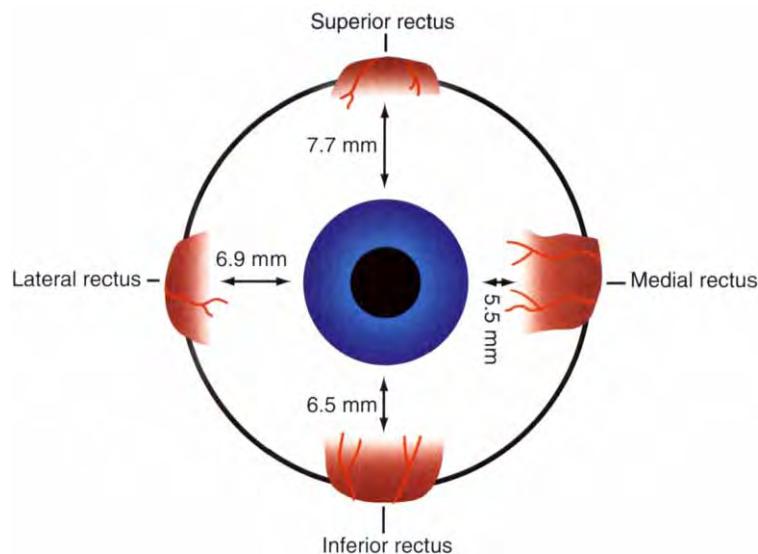


Figure 10-8

Diagram of the insertions of the four rectus muscles. An imaginary spiral connecting the insertion points is called the spiral of Tillaux.

and its insertion into the sclera is anterior to the center of rotation, superior to the corneal limbus. The muscle sheath of the superior rectus is continuous with that of the levator palpebrae superioris, as noted previously, and it is similarly innervated by the superior division of the 3rd cranial (oculomotor) nerve. Upon contraction in primary gaze, the major function of the superior rectus is to rotate the globe upward (elevation). However, because of its insertion anterior to the equator, contraction of the superior rectus in primary gaze results secondarily in a small nasal rotation of the eye (adduction) and slight encyclotorsion (intorsion). The action of the superior rectus varies significantly, depending on the horizontal rotation of the eye. When the line of sight is directed 23 to 25 degrees temporal to that of the primary gaze position, the superior rectus produces only ocular elevation. When the line of sight is 65 to 67 degrees nasal to that of primary gaze, the superior rectus produces only intorsion and adduction. As noted earlier, the overlapping innervation and physical connections between the superior recti and the levators (see Figure 10-5) are responsible for simultaneous upper eyelid retraction in upgaze and lowering of the upper eyelids in downgaze.

The inferior rectus muscle is situated in the plane of the superior rectus but inserts into the sclera below the limbus. Its muscle sheath is attached anteriorly to that of the inferior oblique muscle. The inferior rectus is innervated by the inferior division of the 3rd cranial (oculomotor) nerve, and its contraction in primary gaze results primarily in depression of the eye with, secondarily, a small amount of adduction and exyclotorsion (extorsion). As with the superior rectus, the action of the

inferior rectus varies significantly, depending on the horizontal rotation of the eye. When the line of sight is directed 23 to 25 degrees temporal to that of the primary gaze position, the inferior rectus produces only ocular depression. When the line of sight is 65 to 67 degrees nasal to that of primary gaze, the inferior rectus produces only extorsion and adduction.

The innervations of the superior and inferior rectus muscles are coordinated, such that one is inhibited while the other is active, thus directing component rotations of the eye in the vertical plane. Adduction produced by the combined actions of the superior and inferior recti is countered by the lateral rectus, and torsions are countered by action of the superior or inferior oblique muscles (see the next section).

Oblique Muscles

The distal portion of the superior oblique muscle and the entire inferior oblique muscle are located in a vertical plane that intersects the primary line of sight by an angle of 51 to 53 degrees (see Figure 10-7). The superior oblique muscle is anchored in the lesser wing of the sphenoid bone at the orbital apex above the circle of Zinn. It runs outside the rectus muscle cone, superonasally, to the trochlear fossa in the frontal bone near the superonasal orbital rim. At this point, its route is redirected by slippage through a cartilaginous "pulley," or trochlea, into the plane of action. The superior oblique then runs back under the muscle cone and inserts into the sclera of the globe behind the insertion of the superior rectus and posterior to the equator (see Figure 10-7). Unlike a rectus muscle, the superior oblique pulls its insertion forward instead of backward.

The superior oblique is innervated by the 4th cranial (trochlear) nerve; contraction of the superior oblique in primary gaze acts primarily to encyclorotate (intort) the globe. However, because of the insertion posterior to the equator, contraction of the superior oblique in primary gaze results secondarily in a slight depression and temporal rotation of the eye. When the line of sight is directed 51 to 53 degrees nasal to that of the primary gaze position, the superior oblique produces only ocular depression. When the line of sight is 37 to 39 degrees temporal to that of primary gaze, the superior oblique produces only intorsion and slight abduction.

The inferior oblique muscle also pulls the globe forward instead of backward in the same plane of action as that of the superior oblique. Although the other EOMs originate at the orbital apex, the inferior oblique is anchored in a shallow depression at the front of the anteronasal floor of the orbit near the lacrimal fossa. The inferior oblique runs back within the rectus muscle cone and above the inferior rectus, where the muscle sheaths of the two muscles become attached. The inferior oblique inserts into the sclera behind the insertion of the inferior rectus and posterior to the equator, an area that is close to the macula, ciliary vessels, and ciliary nerves. The inferior oblique is a part of the "suspensory ligament of Lockwood," which consists of (1) the inferior oblique and its muscle sheath, (2) the anterior portion of the inferior rectus and its muscle sheath, (2) intermuscular septa connecting the anterior muscle sheaths of the lateral and medial recti to that of the inferior rectus, and (3) the lateral and medial check ligaments (see Figure 10-5). It is believed that Lockwood's ligament helps support the globe from underneath so as to maintain its vertical position within the orbit.⁴⁸

The inferior oblique is innervated by the inferior division of the 3rd cranial (oculomotor) nerve; its contraction in primary gaze acts primarily to excyclorotate (extort) the globe. However, because of the insertion posterior to the equator, contraction of the inferior oblique in primary gaze results secondarily in a small elevation and temporal rotation (abduction) of the eye. When the line of sight is directed 51 to 53 degrees nasal to that of the primary gaze position, the inferior oblique produces only ocular elevation. When the line of sight is 37 to 39 degrees temporal to that of primary gaze, the inferior oblique produces only extorsion and slight abduction.

The innervations of the superior and inferior oblique muscles are coordinated, such that one is inhibited while the other is active, thus establishing the torsional position of the eye. Vertical rotations or abduction produced by the combined actions of the superior and inferior oblique muscles are countered by action of the other two EOM pairs. The tendinous fibers that insert the oblique muscles into the sclera are spread out in a fan shape (see Figure 10-7), unlike the relatively straight

insertion fibers of the rectus fibers (see Figure 10-6). Hence, the medial fibers in the fan are shortened by adduction and the temporal fibers are elongated; the opposite occurs during abduction. This tends to allow the contractile force of the oblique muscles to remain concentrated in the same plane of action for various horizontal positions of gaze. As a result, the primary action of an oblique muscle is to intort (superior oblique) and extort (inferior oblique) the globe through most of the lateral excursion of the line of sight. This leaves the superior and inferior rectus muscles as the primary muscles controlling vertical eye rotations throughout most of the lateral range of eye excursion. The secondary actions of the oblique muscles, noted earlier, are less powerful in primary gaze than are the secondary actions of the superior and inferior rectus muscles.⁴⁸

Cranial Nerves III, IV, and VI

It is important to review the neuroanatomy of the motor controls for the EOMs, because lesions of the nerves or at the central origins of the nerves will have consequences directly linked to the resulting lack of innervation.^{38,40,49}

Emerging ventrally from the midbrain (mesencephalon) between the cerebral peduncles, near the midline, the two 3rd cranial (oculomotor) nerves pass between the ipsilateral superior cerebellar and posterior cerebral arteries. Each 3rd nerve then follows a course forward and downward along the ipsilateral posterior communicating artery, and pierces the wall of the cavernous sinus on that side. Here, the 3rd nerve is close to the 4th and 6th cranial nerves, as well as the ophthalmic division of the 5th cranial (trigeminal) nerve. The 3rd cranial nerve divides and enters the orbit as the superior and inferior branches via the superior orbital fissure. The branches go through the circle of Zinn into the muscle cone. The preganglionic parasympathetic fibers exit the inferior branch of the 3rd nerve and synapse within the ciliary ganglion, which is normally attached to the outer surface of the inferior branch. As was noted earlier, postganglionic fibers from the ciliary ganglion innervate the ipsilateral pupillary sphincter and the ciliary muscle. The superior branch of the 3rd cranial (oculomotor) nerve is of smaller caliber than the inferior branch, because it serves only the ipsilateral superior rectus and levator palpebrae superioris. The inferior branch, of larger caliber, serves all of the remaining ocular muscles except the iris dilator, superior oblique, and lateral rectus.

Fibers in the 3rd cranial nerve are supplied by the oculomotor complex, which is located near the central gray matter of the midbrain at the level of the superior colliculi. The oculomotor complex consists of several coordinated nuclei and motor cell column pairs (the dorsal cell columns, intermediate cell columns, ventral

cell columns, and dorsal median cell columns). Most of the fibers in the 3rd cranial nerve are uncrossed, but some are crossed. The dorsal cell column supplies uncrossed fibers destined for the inferior rectus. Similarly, the intermediate cell column and ventral cell column supply uncrossed fibers for the inferior oblique and the medial rectus, respectively. The dorsal median column provides crossed fibers to the superior rectus. The paired Edinger-Westphal nuclei and anterior median nuclei supply uncrossed preganglionic parasympathetic fibers for the ciliary ganglia. As a result, the columns on the right side of the midbrain send fibers destined for the right inferior rectus, right inferior oblique, right medial rectus, *left* superior rectus, right pupillary sphincter, and right ciliary muscle. The single caudal central nucleus gives rise to fibers destined for the two levator palpebrae superioris muscles that are equally crossed and uncrossed. The existence of a central nucleus of Perlia, which has been said to control convergence and divergence, has been postulated but has been difficult to substantiate. Smaller accessory nuclei exist, which are thought to be involved in torsional eye movements and reflex movements of the head and neck.

The pair of trochlear nuclei lie in the midbrain (mesencephalon) at the level of the inferior colliculi, in the peri-aqueductal gray matter, caudal (below) and adjacent to the oculomotor complex. Each trochlear nucleus supplies originally uncrossed fibers to its respective 4th cranial (trochlear) nerve. However, the two slender nerves emerge behind the midbrain (dorsally), in a downward direction, and decussate completely behind the brain stem in what is called the superior medullary velum. Each 4th cranial nerve then curves around the brain stem to attain a ventral direction, then inward and directly forward, to pass between the superior cerebellar and posterior cerebral arteries. Here, the 4th nerve is significantly inferior and lateral to the 3rd cranial nerve as the nerves follow the posterior communicating artery. Their vertical separation reduces as the 3rd nerve drops to nearly meet the 4th nerve prior to entering the cavernous sinus. The 4th nerve slips above the 3rd nerve in the cavernous sinus and escapes the circle of Zinn to innervate the superior oblique muscle.

The 4th cranial nerves have the longest intracranial course of any of the cranial nerves (75 mm) and are the only completely crossed cranial nerves. They are also the only nerves to emerge dorsally from the central nervous system and are the thinnest of the cranial nerves. As a result, the somewhat fragile 4th nerve supplies innervation to the superior oblique muscle on the contralateral side of its nucleus and is more likely to be injured as it runs most of its long course on the side ipsilateral to the superior oblique.

The pair of abducens nuclei lie in the very dorsal (back) portion of the pons next to the floor of the 4th ventricle, well below (caudal to) the trochlear nuclei

and oculomotor complex. Each abducens nucleus is partially encircled by the root of a 7th cranial (facial) nerve as the complicated root loops behind and around the nucleus. The abducens nucleus supplies uncrossed fibers to the root of its respective 6th cranial (abducens) nerve. The 6th nerve root emanates ventrally from the nucleus and travels across nearly the entire width of the pons before emerging ventrally in the furrow between the pons and medulla, immediately next to the midline, as a slender 6th cranial nerve. The thin nerve runs a long course steeply up and over the petrous tip of the temporal bone, to which it is bound, then a less inclined route up to the cavernous sinus where it is adjacent to the other cranial nerves destined for the orbit. The 6th cranial nerve enters the orbit via the superior orbital fissure with the other ocular cranial nerves, and goes through the circle of Zinn to innervate the lateral rectus muscle. Because of its fragility and long course through the cranium, over the apex of the temporal bone, the 6th cranial nerve is vulnerable to injury and increased intracranial pressure.

Binocular (Conjugate) Eye Movements

The actions of the EOMs are coordinated between the two eyes, with bifoveal fixation as the goal. This is achieved by gaze centers in the midbrain and pons, which are responsible for the appropriate excitatory and inhibitory innervations to the individual ocular muscles in order to achieve the amount and direction of conjugate eye movement required. The vertical gaze center is a single nucleus in the posterior commissure of the midbrain above the level of the superior colliculi, which disseminates input to the nuclei of the oculomotor complex and the trochlear nuclei, such that the proper signals are sent along the cranial nerves to both eyes. The horizontal gaze center is also known as the paramedian pontine reticular formation (PPRF). The PPRF is a pair of sites in the lower pons, ventral to the nuclei of the 6th cranial nerves, which are connected to each other and the motor nuclei of both eyes by the medial longitudinal fasciculus (MLF). Therefore, a lesion in the upper midbrain may reduce the ability to rotate the eyes vertically, whereas a defect in the lower pons may reduce the ability to rotate the eyes horizontally.

Hering's Law

Under normal binocular circumstances, the direction, speed, and magnitude of rotation will be equal between the two eyes during conjugate movements. The EOMs of the two eyes are yoked together, with identical excitatory or inhibitory innervation supplied to corresponding yoked muscles (Table 10-2). This is the basis of Hering's law, which concludes that equal and simultaneous innervation is sent to the corresponding EOMs of the two eyes for all voluntary conjugate eye movements.

TABLE 10-2 Yoked Pairs of Ocular Muscles

Right Eye	Left Eye
Lateral rectus	Medial rectus
Medial rectus	Lateral rectus
Superior rectus	Inferior oblique
Inferior oblique	Superior rectus
Inferior rectus	Superior oblique
Superior oblique	Inferior rectus

Hering's law applies whether the eyes are fixating binocularly or monocularly. For instance, the covered left eye will normally follow the right eye when the right eye fixates in different positions of gaze. However, Hering's law does not imply that the corresponding muscles actually *receive* the innervation that is intended, or that the muscles will equally react to the innervation that reaches them. This is because syndromes or lesions of the neural routes may reduce the actual innervations that arrive at the EOMs, or muscular defects and local physical abnormalities may reduce the ability of the muscles to carry out their functions. In these cases, the paretic muscle will induce two phenomena that can be recognized by the clinician when the line of sight is in or near the paretic muscle's plane of action. First, when fixating with the nonparetic eye, the line of sight of the eye with the paretic muscle will lag behind that of the nonparetic eye when the patient is asked to fixate into the direction of action of the paretic muscle. This is called the primary deviation, or undershooting. Second, when fixating with the paretic eye, the nonparetic eye will overshoot into the paretic muscle's direction of action. This is called the secondary deviation, or overshooting. Overshooting is more pronounced and noticeable to the clinician than is undershooting. Hence, it is often easier to recognize that an EOM paresis exists and to identify the paretic muscles using the secondary deviation in comparison with using the primary deviation.

Types of Conjugate Eye Movements

There are three primary types of conjugate eye movements: saccades, pursuits, and vestibular eye movements. Saccades and pursuits are each generated in different cerebral areas and may be mediated through different supranuclear pathways. Vestibular movements are reflex actions initiated by the ear canal and mediated by the cerebellum and brain stem. However, they use the same gaze centers, motor nuclei, and motor nerves, which together constitute the "final common pathway" to the EOMs. All of the movements result from the coordinated action of the 12 EOMs (six EOMs per eye).

Saccades are rapid, voluntary or reflex fixational conjugate eye movements stimulated by alternation of the object of regard in the X, Y object plane. They can be elicited by asking the patient to look around the examination room at different distant targets. The fixations and refixations depend on the integrity of the fovea and cooperation of the patient. The cerebral origin of saccades appears to be in the two areas 8 of the frontal lobes (the frontal eye fields) and the posterior parietal cortex. Supranuclear fibers course from these areas to the midbrain (superior colliculus) and cross to the other side. The saccadic gaze center is most likely in the PPRF in the lower pons, which receives the supranuclear inputs from the frontal eye fields (FEF) and the superior colliculus. The gaze center is, in turn, responsible for the appropriate excitatory and inhibitory influences given to the motor nuclei of the 3rd, 4th, and 6th cranial nerves, such that a saccade is made to the proper approximate X, Y position in object space. Undershoots and overshoots are then corrected by subsequent additional saccadic movements.

Each FEF directs saccadic eye movements to the contralateral side. Thus, stimulation of the right FEF results in conjugate eye movements to the left side. The contralateral eye movements can be strictly lateral, or they may be also up or down to various degrees, depending on the location of the stimulus within area 8. Strictly vertical saccades are elicited by simultaneous and equal stimulation of both sides of the FEF. Saccadic dysfunction can be a result of cortical disease in the frontal lobes.

Pursuits are slow, smooth tracking conjugate eye movements stimulated by target motion, which maintain fixation at the foveas. They can be elicited by asking the patient to follow a slow moving target. In the absence of target motion, patients who attempt to move the eyes smoothly will produce a series of small saccades. Should a target be moving too fast or slow for a pursuit to keep the line of sight on target, a saccadic movement is made to regain fixation before the system again pursues the target.

The cerebral origin of pursuits appears to be in the striate visual cortex at the parieto-temporo-occipital junction. From the visual cortex the signal is relayed to the FEF, which projects to horizontal gaze center (PPRF) in the lower pons by supranuclear fibers that cross in the midbrain. The visual cortex directs pursuit movements to the ipsilateral side. Stimulation of the right area striate visual cortex results in conjugate eye movements to the right side. Vertical pursuit movements and component movements are elicited by simultaneous stimulation of both sides.

During self-motion or motion in the environment, retinal images are stabilized by a reflex system consisting of the vestibulo-ocular and the optokinetic reflexes.⁴⁹ The optokinetic response is simulated by

retinal image slippage and adapts eye velocity to the velocity of the retinal image.⁵¹ It complements the vestibulo-ocular reflex to generate compensating gaze-stabilizing eye movements.

Vestibular eye movements are reflex, smooth, pursuit-like conjugate eye rotations that counteract head movements during locomotion. They are initiated by angular acceleration of the head sensed by the three semicircular canals or by head tilt sensed by the utricle and saccule. The former are often associated with the term *vestibular nystagmus* and the latter with the term *doll's eye movements*. These sensory organs are located adjacently in the vestibular apparatus of the inner ear.

Lateral eye movement is driven by the ampulla of a horizontal semicircular canal, whose fibers connect to vestibular nuclei in the pons and are relayed to the contralateral PPRF. Stimulation of a horizontal ampulla results in a pursuit-like conjugate eye movement to the contralateral side, within the plane of the canal (horizontally). Similarly, stimulation of the ampulla of an anterior or posterior vertical semicircular canal results in a pursuit-like eye movement in the respective plane of the stimulated canal. Hence, pursuit-like eye movements in the plane of the angular acceleration will occur because of the component eye movements produced by the three pairs of semicircular canals. These movements compose the "slow phase" of vestibular nystagmus. The "fast phase" of vestibular nystagmus is a corrective saccadic movement driven by the frontal eye fields. Dysfunction of the semicircular canals or of their afferent fibers can result in abnormal vestibular nystagmus that occurs without angular acceleration of the head.⁵²

Doll's eye movements, also called counter-rolling, are reflex pursuit-like compensatory eye rotations that help to maintain fixation when the head is tilted forward or backward or turned to the left or right. The eyes rotate up reflexly, the upper lids are raised as the head is tilted forward, and the eyes rotate down as the head is tilted back. Torsional eye movements are made in response to head tilt left or right, and are the basis for the Bielschowsky head tilt test, to be covered later in this chapter.

These eye movements are the result of the oculocephalic reflex. The utricle and saccule in each inner ear contain hair cells that sense the weight of small crystals of calcium carbonate (otoliths). The hair cells in the utricle are situated parallel to the horizontal plane and the hair cells in the saccule are parallel to a vertical plane. Hence, the tilt of the head forward or backward and to the left or right is coded and sent to the vestibular nuclei in the pons. The codes for head position are applied to vertical and torsional eye movements. Similar horizontal compensatory eye movements, which help maintain fixation during head rotation to the right or left (not head tilt), are doll's eye movements originating at the semicircular canals. Doll's

eye movements become more evident when the patient's other conjugate eye movements have been incapacitated at the cerebral or supranuclear levels—for instance, after a stroke. Being reflexly driven at a lower level, counter-rolling is produced when the patient's head is tilted or turned by the examiner or another person.

Optokinetic nystagmus (OKN) is a phenomenon that is unrelated to vestibular nystagmus, except that the same final common pathways are likely. It probably results from intercortical connections between the frontal and occipital eye fields,⁵³ activates the same network as saccades and pursuits,⁴⁵ and is generated in response to sustained rotations. It is often elicited clinically by use of a vertically striped drum that is rotated before the patient's eyes. A particular stripe is fixated and followed by conjugate pursuit as the stripe travels in one direction across the field of vision. Once the stripe becomes no longer visible, a saccade is made in the opposite direction, such that another moving stripe is fixated and followed. The process repeats itself as long as the patient directs his or her attention to the moving stripes, resulting in an alternation of slow, smooth pursuit movements consistent with the direction and speed of the drum rotation and fast saccadic movements in the opposite direction. OKN is the basis for the well-known "railroad car nystagmus."

OKN is a strong involuntary reflex in the horizontal plane but is relatively weak vertically. The reflex is involuntary and can be induced in all persons with a normal visual system, if sufficient visual acuity is present to recognize the stripes. It can be used to document the function of both the saccadic and pursuit systems. OKN cannot be suppressed for long periods. As a result, malingeringers and hysterical patients, who are expressing visual acuity much lower than the capability of their visual systems, can often be identified by use of OKN.⁵³

Clinical Evaluation

During the initial phases of the eye examination, the clinician observes the patient's ability to fixate and change fixation from one target to another (saccades). The patient is asked to maintain fixation while following a moving target into different gaze positions (pursuits). While the patient is fixating a target, the head can be tilted forward or back and to the right or left by the examiner and maintenance of fixation observed (doll's eye movements). If fixation is poor, the clinician should rule out poor vision, poor attention, and poor motivation as causes, before concluding that an abnormality is present. The clinician should be alert for abnormal fixation, saccades, or pursuits: nystagmus, head movements substituting for eye movements, drifts of fixation, delays of initiating eye movements, deviations (differences) between the rotations of the two eyes, under-

shooting, and overshooting. Abnormal "cogwheel" eye movements appear as jerky, erratic pursuits with frequent refixation attempts.⁵⁴

In the routine case, much of the necessary observation for saccades and vestibular eye movements can be performed during the case history, as the patient looks over the examination room, or in concert with an examination of the external ocular structures. Although the broad H test, noted immediately following, is used to screen the pursuit system, it is rare when a lesion involves the supranuclear connections or the occipital eye fields. Hence, in nearly all examinations, the broad H test allows the clinician to assess specifically the final common pathway to the EOMs. If a neuromuscular abnormality is identified, the red lens test or the Parks three-step procedure may be used to inspect and perhaps identify the EOMs that are not operating appropriately. With this information, the clinician may diagnose the probable or potential neuromuscular deficiency and apply this knowledge to the management of the patient.

Versions and Ductions: Six Cardinal or Secondary Positions of Gaze

When the neuromuscular systems controlling eye movements are operating properly, each eye is able to fixate a distant target in all positions of gaze. The angle of deviation that exists between the lines of sight of the two eyes is zero in all gaze directions for those patients achieving binocularly or is constant in all gaze directions for patients with a nonparetic strabismus. These deviations are called comitant (also known as concomitant). When one or more EOMs are paretic or paralyzed, however, the angle of deviation between the eyes varies in different positions of gaze. These deviations are called incomitant. According to the anatomy of the EOMs described earlier, it is apparent that the lines of sight will be at their greatest misalignments from that required for bifoveal fixation when the patient is gazing in one or more of the following six positions, called

the six cardinal or secondary positions of gaze (Figure 10-9):

1. To the patient's immediate left, in the direction and plane of primary action of the medial rectus of the right eye and the lateral rectus of the left eye. These EOMs are primarily responsible for rotation of the respective eye directly to the left.
2. To the patient's immediate right, in the direction and plane of primary action of the medial rectus of the left eye and the lateral rectus of the right eye. These EOMs are primarily responsible for rotation of the respective eye directly to the right.
3. To the patient's left and then up, in the general direction and approximate plane of action of the inferior oblique of the right eye and the superior rectus of the left eye. These EOMs are primarily responsible for rotation of the respective eye up when gazing to the left.
4. To the patient's right and then up, in the general direction and approximate plane of action of the inferior oblique of the left eye and the superior rectus of the right eye. These EOMs are primarily responsible for rotation of the respective eye up when gazing to the right.
5. To the patient's left and then down, in the general direction and approximate plane of action of the superior oblique of the right eye and the inferior rectus of the left eye. These EOMs are primarily responsible for rotation of the respective eye down when gazing to the left.
6. To the patient's right and then down, in the general direction and approximate plane of action of the superior oblique of the left eye and the inferior rectus of the right eye. These EOMs are primarily responsible for rotation of the respective eye down when gazing to the right.

The broad H test is used to perform screenings of pursuit eye movements and the final common pathways in the six cardinal positions of gaze. The "H" refers to the path that the lines of sight follow in the X, Y plane

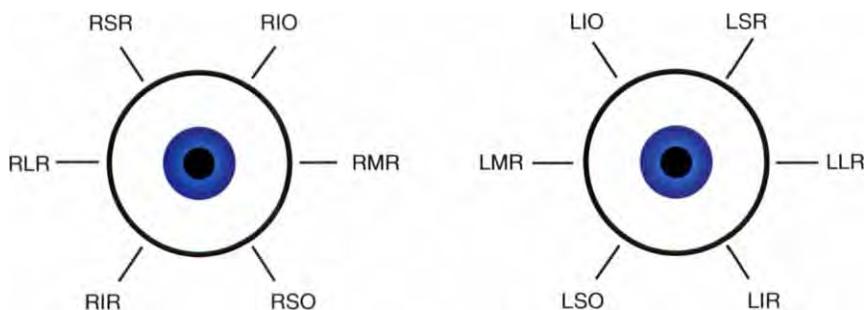


Figure 10-9

Diagram of the six cardinal positions of gaze and the extraocular muscles of each eye that are involved primarily with maintenance of gaze in these positions. *R*, Right; *L*, left; *SR*, superior rectus; *IR*, inferior rectus; *MR*, medial rectus; *LR*, lateral rectus; *SO*, superior oblique; *IO*, inferior oblique.

in object space, because the patient is asked to fixate a small target in the examiner's hand that traces out an imaginary "H" pattern in front of the patient. The two positions at which the horizontal bar intersect the legs of the "H" are meant to correspond to cardinal gaze positions 1 and 2, noted earlier. The upper ends of the two legs are indicative of cardinal positions 3 and 4, whereas the lower ends indicate positions 5 and 6. By directing the two eyes to these six gaze positions, the clinician may observe deviations in eye movements, manifested as underactions or overactions, caused by neuromuscular EOM deficiencies.

The patient should remove his or her spectacles so the examiner can easily view the eyes and their alignment. The examiner stands or sits facing the patient at a distance of approximately 1 m or less in a fully illuminated room. The patient is instructed to fixate a small target, often a penlight or the examiner's finger, held approximately 40 cm in front of the midpoint between the patient's eyes. For children, a small finger puppet or other colorful target may be used. The patient is asked to follow the movement of the target with the eyes, without moving the head, as an "H" pattern is traced out in front of the patient (Figure 10-10). The ends of the legs of the "H" correspond to extreme gaze positions at the four corners of the possible field of fixation. Lid retraction may be necessary in downgaze to allow the examiner to note the extent of eye rotation. It is normal to see a slight nystagmus, called end-point nystagmus, when the eyes are in an extreme gaze position. However, end-point nystagmus may be accentuated by paresis of one or more of the EOMs.

Versions (binocular pursuits) are assessed first, because both eyes are observed at the same time for sim-

ilarity in movements. If a penlight is used as the fixation target, the corneal reflections can be used to note changes in position of the two eyes relative to each other. If any overactions or underactions occur in either eye, the testing is repeated monocularly for each eye. These are ductions, and are conducted in the same manner as versions with the exception that the contralateral eye is occluded while the ipsilateral eye undergoes the broad H test.

In the routine eye examination, versions are screened first, because it is usually easier to identify a relative discrepancy between the sighting of the eyes than it is to observe the same discrepancy monocularly. The patient may report diplopia when gazing into the direction of action of the paretic muscle, yet single binocular vision may result when gazing in the opposite direction. If the patient can follow the target around the H pattern in the routine manner, with both eyes fixating the target, the broad H test result is negative. If an incomitant strabismus is encountered such that either or both eyes are unable to properly follow the target into one or more of the cardinal positions of gaze, the test result is positive, and the clinician will need to follow-up this finding with additional testing.

It is possible that the patient may appear to have comitant strabismus that is manifested in all of the six cardinal positions. Incomitancies are more apparent immediately after a lesion occurs, in the acute stage, because the visual system compensates for them over time. The incomitancies that are paretic in origin become minimized and are often difficult or impossible to diagnose in the chronic stages. Inspection of comitancy is somewhat inexact with the H test because any incomitancy would be necessarily large if it could be viewed by the practitioner in this manner. When a strabismus appears comitant, the clinician should perform a more exacting procedure to rule out lesser incomitancies, such as the red lens test or the cover test (in nine positions of gaze), noted later.

It is often difficult to tell an overaction of one eye from an underaction of the other. Having suspected or found an EOM deficiency, then, one observes ductions of each eye critically to identify which eye has the paretic EOMs. A paretic EOM will cause the eye to lag behind or undershoot when the patient is asked to gaze monocularly into the direction of action of the EOM. However, the eye rotation may appear normal when the patient is asked to gaze in the opposite direction. Ductions are usually not tested when the versions are unremarkable. The results of version and duction testing in cases of abnormal EOM function will be consistent with those discussed later under the neuromuscular EOM anomalies.

Inspection of Incomitancy

The red lens test is a *subjective* determination of the binocular ocular deviations in nine positions of gaze,

Figure 10-10

Testing of versions with the broad "H" pattern. Here, the patient is looking at the top of one of the legs of the "H" to his upper left.

and it is performed using the red accessory lens from the trial lens set. The nine positions include the six cardinal positions, the primary gaze position (straight ahead), upgaze, and downgaze. The test is used to identify and categorize incomitancies in cases of strabismus. The red lens is placed in front of the patient's right eye initially, and a penlight is directed toward the midpoint between the patient's eyes from a distance of 40 cm to 1 m. The strabismic patient should be able to see two lights: one is white as viewed by the left eye, and the other is red as viewed by the right eye. Diplopia is induced by reduced clues to fusion in the presence of a binocular system that is unable to fuse because of strabismus or that is on the edge of binocularity because of EOM paresis. The test is of little use for patients who are fully binocular such that the diplopia does not occur. Likewise, the test is inadequate for patients having abnormal retinal correspondence or who strongly suppress one eye as a result of longstanding strabismus. An alternative for these latter patients is to neutralize the deviations with an alternating cover test using loose prisms (a procedure described later) in the nine positions of gaze, which can serve as an *objective* measurement of the deviations for any strabismic patient.

The penlight is moved into the nine positions of gaze, and the patient is required to fixate the light without moving the head. At each position, the patient is asked whether one or two lights are seen. If two lights are seen, the clinician questions the patient about the relative position of the lights to each other and their degree of separation. The relative positions and degree of separation of the two images are recorded at each of the nine positions, as shown in Figure 10-11. The most peripheral image of the two is that viewed by the undershooting or lagging eye.

Should the red and white images appear aligned side by side, a horizontal deviation is present. Similarly, a vertical deviation is present when the red and white images appear to be aligned vertically. Most of the time, however, the total deviation will be the result of addition of its horizontal and vertical components. If the deviation is comitant, the separation between the red and white images will be equal in all positions of gaze. If the deviation is incomitant, the separation between the lights will be greatest in the field of gaze of the affected muscle or muscles. The separation will be the least or zero in the field of gaze opposite to that of the affected muscle or muscles. The separation will increase as the gaze position changes over the field of fixation from minimum to maximum.

The patient will generally fixate with the eye that is not covered by the red lens, because the lens produces a darker visual field. The condition of the affected eye can be substantiated by repeating the test after switching the red lens to the left eye. Because overactions are generally larger than underactions, greater deviations

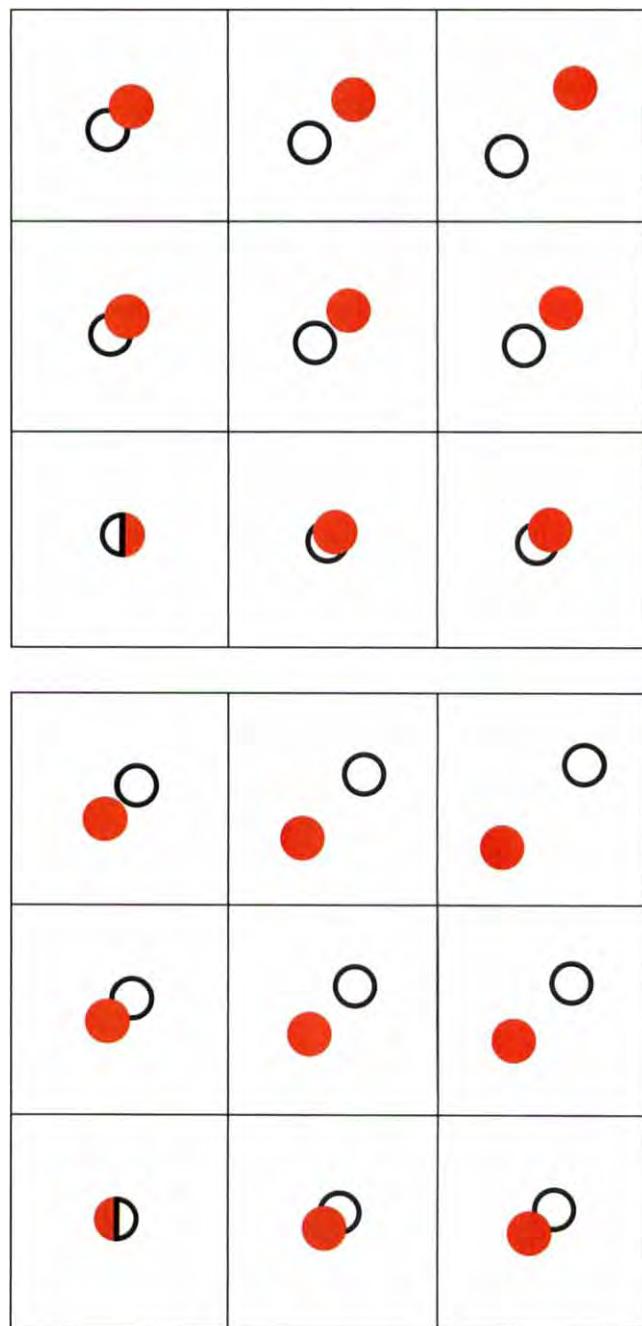


Figure 10-11

Results of the red lens test for a classic paresis of the right superior rectus: (above) with the red lens over the right eye so that the left eye is fixating (primary deviation), and (below) with the red lens over the left eye so that the right eye is fixating (secondary deviation). The filled red circles represent the perceived position of the red spot relative to the unfilled white circles representing the perceived location of the white spot. This could occur, for instance, with an isolated lesion of the superior branch of the 3rd cranial nerve. The symbol at the lower left of each diagram is intended to indicate bifoveal fixation or superimposition of the two images.

should be noted when fixating with the paretic eye (see Figure 10-11). The results of red lens tests in cases of abnormal EOM function will be consistent with those discussed later under neuromuscular EOM anomalies. The Hess–Lancaster screen is a similar, more elaborate method for the analysis of incomitancies, which requires special instrumentation and is reserved for specialty practice.

The Parks three-step procedure is a specific series of three tests designed to isolate the paretic muscle in cases of vertical deviations (Figure 10-12). The three-step procedure was described by Hagedoorn,⁵⁵ popularized by Parks,⁵⁶ and included as its third step the Bielschowsky head tilt test. When a vertical strabismus is identified or suspected, the clinician may observe the hypertropia or, more accurately, measure objectively the hypertropia by use of the alternating cover test and loose prisms in the following three steps (Table 10-3):

1. The clinician determines whether the hypertropia is present in the right eye or the left eye in primary gaze, which limits the determination of the paretic muscle to a possible four EOMs. In right hypertropia (left hypotropia), the paretic muscle

could be the right inferior rectus, right superior oblique, left superior rectus, or left inferior oblique. In left hypertropia (right hypotropia), the paretic muscle could be the left inferior rectus, left superior oblique, right superior rectus, or right inferior oblique.

2. The clinician determines whether the hypertropia increases in gaze directly to the patient's right or left. Right gaze is produced by a head turn to the left as the patient maintains fixation on a distant object straight ahead. Similarly, left gaze is produced by a head turn to the right as the patient fixates the same object. This step eliminates two of the four possible muscles remaining from step 1. If right hypertropia increases in right gaze (left head turn), the potentially paretic EOMs remaining are the right inferior rectus and the left inferior oblique; if the increase is in left gaze (right head turn), the remaining EOMs are the right superior oblique and the left superior rectus. If left hypertropia increases in left gaze (right head turn), the potentially paretic EOMs remaining are the left inferior rectus and the right inferior oblique; if the

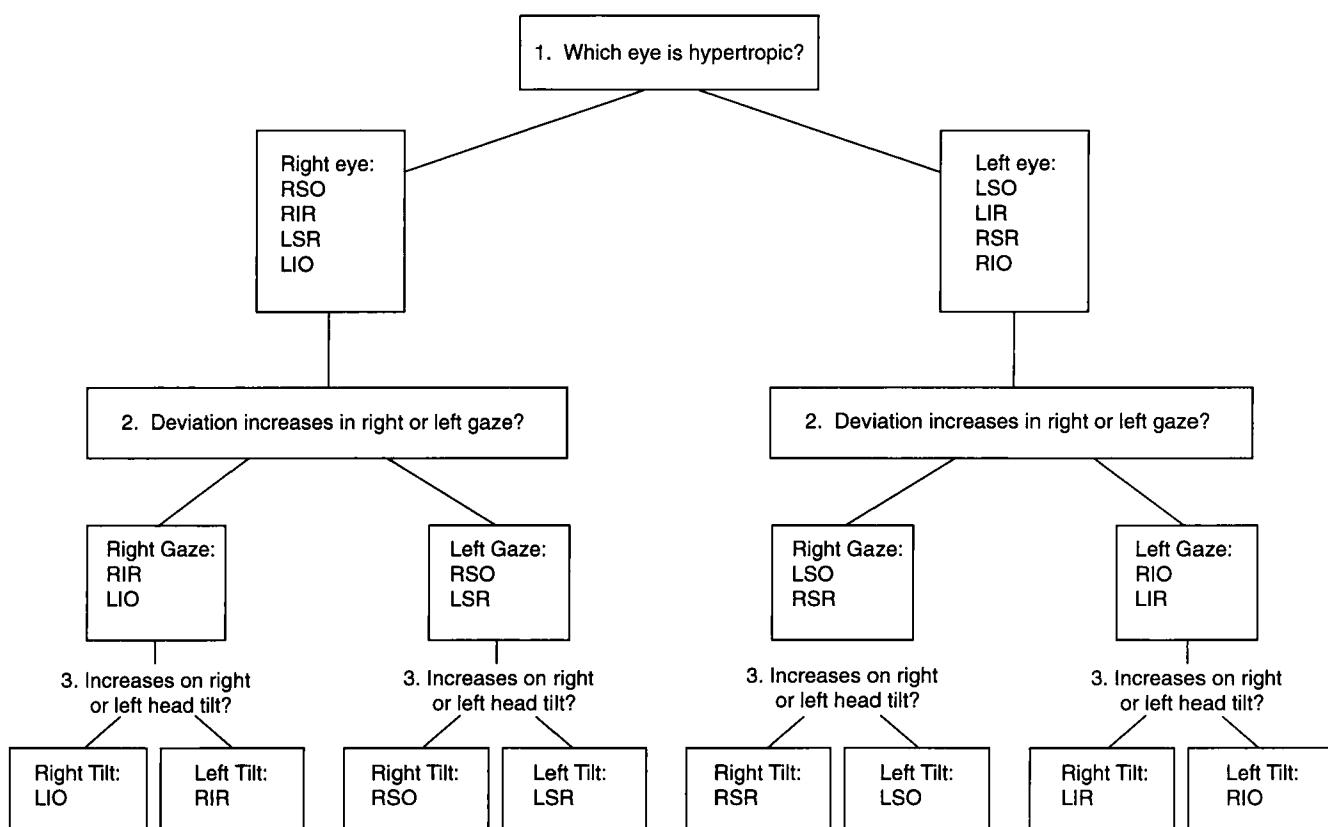


Figure 10-12

Flow chart for the Parks three-step procedure. *R*, Right; *L*, left; *SR*, superior rectus; *IR*, inferior rectus; *MR*, medial rectus; *LR*, lateral rectus; *SO*, superior oblique; *IO*, inferior oblique.

TABLE 10-3 Diagnosis of a Paretic Muscle for a Vertical Deviation

INCREASED HYPERDEVIATION			
Vertical Deviation	Head Turn	Head Tilt	Paretic Muscle
Right hyper	to Right	to Right	LIO
	to Right	to Left	RIR
	to Left	to Right	RSO
	to Left	to Left	LSR
Left hyper	to Right	to Right	RSR
	to Right	to Left	LSO
	to Left	to Right	LIR
	to Left	to Left	RIO

increase is in the right gaze (left head turn), the remaining EOMs are the left superior oblique and the right superior rectus.

- The clinician determines whether the hypertropia is greater when the patient's head is tilted to the right or to the left. This step eliminates one of the two possible muscles remaining from step 2 and thus isolates the paretic muscle. The head tilts induce reflex conjugate torsions as a result of the vestibular apparatus, which attempt to keep the horizontal meridians of the eyes aligned with the horizon. Head tilt to the patient's right shoulder creates a clockwise rotation of both eyes as viewed by the clinician (intorsion of the right eye and extorsion of the left eye), and head tilt to the left shoulder creates a counterclockwise rotation of both eyes (extorsion of the right eye and intorsion of the left eye). Intorsion is the primary action of the superior oblique and extorsion of the inferior oblique. Vertical eye rotations are secondary for these muscles. Therefore, the induction of torsion by head tilt also creates input for vertical eye movement, which is countered by the superior and inferior rectus muscles.

When the head is tilted toward the side of a paretic superior oblique, the compensatory upward action of the superior rectus on that side is unopposed, and the vertical deviation becomes greater than if the head is tilted away from the affected eye. Had the paretic muscle been the contralateral superior rectus, the head tilt toward that affected side would create intorsion of that eye's superior oblique, the secondary downward input unopposed by the paretic superior rectus. Hence, the head tilt produces greater hypertropia when it is toward the paretic superior oblique of one eye or the paretic superior rectus of the other eye, and the effect of either is isolated.

Similarly, head tilt away from the side of a paretic inferior oblique results in unopposed downward

action of the inferior rectus on that side, and the vertical deviation becomes greater than if the head were tilted toward the affected eye. Had the paretic muscle been the contralateral inferior rectus, the head tilt away from that affected side would create extorsion of that eye's inferior oblique, the secondary upward input unopposed by the paretic inferior rectus. Hence, the head tilt produces greater hypertropia when it is away from the paretic inferior oblique of one eye or the paretic inferior rectus of the other eye, and the effect of either is isolated.

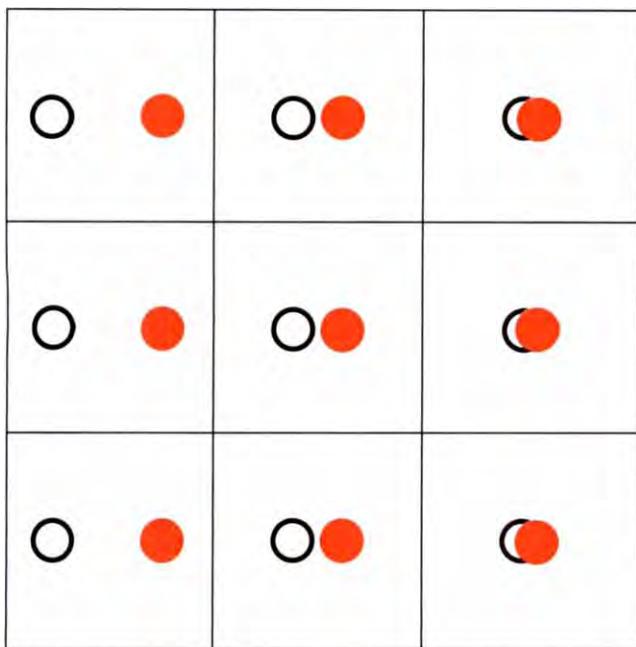
The three-step procedure assumes that only a single paretic muscle is present and so is of particular use in the diagnosis of palsies of the 4th cranial (trochlear) nerve or the superior division of the 3rd cranial (oculomotor) nerve. It is of little help when the entire 3rd cranial nerve is affected or in the diagnosis of 6th cranial (abducens) nerve palsies because paresis of the lateral rectus produces a horizontal instead of vertical deviation.

Neuromuscular Extraocular Muscle Anomalies

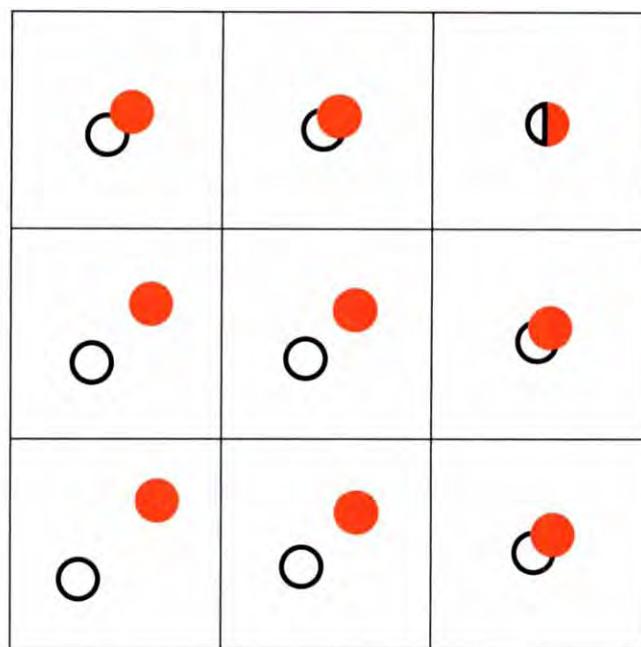
Symptoms of oculomotor problems include decreased ability to fixate an object, eye fatigue, and headaches associated with the need for critical vision or near work. Paresis of the oblique muscles is often accompanied by head tilts to the left or right, and paresis of the vertical rectus muscles by chin elevation or depression. Paretic horizontal rectus muscles are often similarly accompanied by head turns to the left or right. These compensatory head postures allow the gaze position of minimum deviation to occupy the straight-ahead position. Many patients are able to achieve single binocular vision by tilting or turning the head toward the primary actions of the involved extraocular musculature, which permits the eyes to be rotated away from the primary actions. An acute episode of strabismus may result in complaints of diplopia, especially in the gaze position where an affected muscle has its greatest action, which may recede as the visual system compensates by suppressing the vision of the deviating eye.

Sixth Cranial (Abducens) Nerve Palsy and the Lateral Rectus

The long external course of the slender 6th nerve through the cranium, particularly over the apex of the temporal bone, makes it especially susceptible to injury and increased intracranial pressure. Lesions of the nerve, its root, and its nucleus will cause ipsilateral paresis of the lateral rectus, convergent strabismus increasing in temporal gaze, and lateral diplopia (Figure 10-13). Nuclear lesions will most likely be accompanied by ipsilateral paresis or paralysis of the facial muscles, includ-

**Figure 10-13**

Results of the red lens test for a classic paresis of the left lateral rectus, with the red lens over the right eye so that the left eye is fixating (secondary deviation). The filled red circles represent the perceived position of the red spot relative to the unfilled white circles representing the perceived location of the white spot. This could occur, for instance, with an isolated lesion of the 6th cranial nerve. The symbols on the right are intended to indicate a partial superimposition of the two images in those gaze positions.

**Figure 10-14**

Results of the red lens test for a classic paresis of the right superior oblique, with the red lens over the left eye so that the right eye is fixating (secondary deviation). The filled red circles represent the perceived position of the red spot relative to the unfilled white circles representing the perceived location of the white spot. This could occur, for instance, with an isolated lesion of the 4th cranial nerve. The symbol at the upper right is intended to indicate bifoveal fixation or superimposition of the two images.

ing the orbicularis, due to simultaneous involvement of the root of the 7th cranial (facial) nerve, which encircles the 6th nerve nucleus. Sixth nerve palsies are also associated with involvement of the 5th and 8th cranial nerves. Unlike the other rectus muscles, which are supplied by two anterior ciliary arteries originating from muscular branches of the ophthalmic artery, the lateral rectus is supplied by only a single anterior ciliary artery³⁹ (see Figure 10-8). As a result, it is possible that the lateral rectus is more frequently and adversely affected by ischemia than are the other EOMs.

In Duane's retraction syndrome, the globe of the affected eye is retracted and the palpebral fissure narrowed when adduction is attempted. The affected eye is also unable to abduct in most cases. The traditional explanation has been that the lateral rectus is fibrotic, such that it cannot contract or stretch properly. Hence, the lateral rectus is unable to abduct the eye properly and is not elastic enough to allow much adduction by the medial rectus. An alternative explanation is a miswiring of the innervation to the lateral rectus, resulting in cocontraction of the lateral and medial rectus

muscles.⁵⁷ It has been shown that the abducens nucleus may be hypoplastic in cases of Duane's syndrome, possibly because of disuse, and that innervation of the lateral rectus is supplied by 3rd nerve fibers similar to those innervating the medial rectus.^{58,59} Despite the inability to abduct the eye, patients with Duane's syndrome do not appear esotropic in the primary gaze position. Approximately 90% of the cases are unilateral and 10% bilateral.

Fourth Cranial (Trocklear) Nerve Palsy and the Superior Oblique

The thinness and long course of the 4th nerve, crossing in back of the brain stem and partially encircling the midbrain, makes it also especially susceptible to injury. Lesions of the nerve distal to its decussation will cause an ipsilateral paresis of the superior oblique and hypertropia with vertical diplopia increasing in inferonasal gaze (Figure 10-14). There will be significant torsional diplopia existing in other gaze positions, especially temporally. The Parks three-step procedure will reveal increased ipsilateral hypertropia with lateral gaze in the contralateral direction and with head tilt toward the side

of the lesion. Nuclear lesions will be contralateral, as will lesions of the nerve root prior to decussation, whereas a lesion at the site of decussation can result in bilateral oblique paresis.

Palsy of the 4th cranial nerve often occurs simultaneously with paresis of the 3rd cranial nerve because of their close proximity along the posterior communicating arteries, within the cavernous sinus, and through the superior orbital fissure. The presence or absence of a 4th nerve palsy in conjunction with a palsy of the 3rd nerve is a special diagnostic problem, because the eye cannot be directed into the adducted gaze position from which vertical eye movements can be assessed. If the clinician can have the patient direct the eye to the temporal side (with 3rd nerve palsies, the affected eye is usually exotropic) and attempt to move the eye up and down, torsion of the eye as a result of a functioning superior oblique can be positively identified. This can be most easily observed by watching a landmark, such as a conjunctival or limbal blood vessel.

Third Cranial (Oculomotor) Nerve Palsy and the Other Extraocular Muscles

The parasympathetic effects of 3rd nerve paresis on the pupillary sphincter muscle and the ciliary muscle were described earlier in this chapter, as was the impact of 3rd nerve paresis on the levator palpebrae superioris. The 3rd nerve is considerably thicker than the 4th and 6th cranial nerves, and though significant lesions can interfere with the function of the entire nerve, compressive and traumatic lesions of lesser impact may disrupt only a proportion of fibers in the nerve—those on the side of the nerve affected by the lesion. Function of the pupil, for instance, may be spared in some lesions and not in others. In addition, the 3rd nerve splits into two branches before the superior orbital fissure. Hence, diagnosis of 3rd nerve lesions can be more difficult as one attempts to ascertain the location of the lesion along the 3rd nerve.

A total block of the 3rd nerve before it divides will, of course, lead to ipsilateral paralysis of the medial rectus, inferior rectus, inferior oblique, superior rectus, levator, pupillary sphincter, and ciliary muscle. Third nerve lesions can leave the eye mydriatic, with dysfunctional accommodation (cycloplegia), ptosis, and divergent strabismus. The patient is unable to move the eye down, up, or in. Incomplete paralysis (palsy or paresis) of the 3rd nerve can result in combinations of these signs and symptoms, as can lesions distal to the branching of the nerve. The ipsilateral superior rectus and levator, for instance, may be paretic because of a lesion of the superior branch (see Figure 10-11) of the 3rd nerve, yet the inferior oblique, inferior rectus, medial rectus, and the motor root to the ciliary ganglion can be compromised by a lesion to the inferior branch. Lesions in the oculomotor complex of the 3rd nerve can

adversely influence some motor nuclei and leave others intact, resulting in paresis or paralysis of the ipsilateral medial rectus, inferior rectus, inferior oblique, and the contralateral superior rectus, of variable collective involvement and severity.

Internuclear, Gaze Center, Supranuclear, and Cortex Lesions

Having studied the control of the ocular musculature presented earlier in this chapter, one must realize that lesions in various areas of the cortex and supranuclear connections can bring about specific conjugate dysfunctions in saccades, pursuits, or vestibular eye movements. Lower down, in the midbrain, interference with the gaze centers and internuclear connections can result in inability to produce conjugate eye movements of any particular type, because they each require the same or similar gaze centers to coordinate the movements of all of the EOMs for both eyes. It is only at the level of the motor nuclei to or the individual pathways of the cranial nerves that the ill effects of nervous damage is present in only one eye or the other, with adverse function of a specific EOM or small set of EOMs.

MONOCULAR AND BINOCULAR EYE ALIGNMENT

Vergences are binocular eye movements that are not conjugate. Indeed, they are often called disconjugate eye movements, because the lines of sight are rotated toward or away from each other—not in the same direction as occurs for conjugate eye movements. The function of lateral (horizontal) vergences is to maintain bifoveal fixation of targets at various distances, and their control was extensively discussed in Chapter 5. Therefore, lateral phorias and vergences are evaluated during fixation of a distant target and a near target during the typical eye examination. There are vertical vergences, in which one eye rotates up or down in the direction opposite to that of the other eye, and torsional vergences, in which an eye cyclorotates relative to the other eye in order to achieve corresponding meridians. All three vergence motions are necessary for attainment and maintenance of bifoveal fixation. Because vertical and torsional vergences do not normally depend on target distance, they are usually tested only at a single distance, whichever is easier to accomplish or gives the most information with the particular method being used.

The signal for vergences begins in area 19 of the occipital cortex and is relayed to the oculomotor complex by supranuclear fibers. Apparently, the exact center for distribution of fibers to the nuclei of the 3rd and 6th cranial nerves, thought necessary for coordination of the ocular muscles when vergences are desired,

has not been found. It is known that the vergence system does not use the horizontal gaze center in the PPRF as the beginning of its final pathway to the EOMs. Therefore, a "nucleus of Perlia" in the oculomotor complex has been postulated but not substantiated. Nearly all pareses and paralyses of vergence eye movements can be explained on the basis of lesions involving the other known ocular neurological sites within the cortex and midbrain.

In the initial phases of the ocular examination, the ability to fixate and the alignments of the lines of sight are assessed monocularly (angles lambda) and binocularly (the Hirschberg test) in primary gaze by observation of the corneal reflex with respect to the center of the pupil. These assessments enable the clinician to estimate the angle of deviation for a large strabismus or tropia, which is evaluated for comitancy or incomitancy as noted in the previous section. Even if a phoria is present in primary gaze, a tropia may manifest at one or more of the cardinal directions, should an EOM be slightly paretic. The interpupillary distance (IPD) is measured so that the eyes can be later aligned with the optical centers of lenses in the phoropter or trial frame. These tests can be performed without the patient's spectacle correction in place, so that the examiner can obtain an excellent view of the eyes. Contact lenses may be worn when they do not interfere with the examiner's view. These tests enable the clinician to roughly gauge the fixational ability of the patient before more refined testing is conducted.

The angle of deviation (phoria or tropia) in primary gaze at distance and at near can be critically assessed via loose prisms with the cover test, an objective evaluation that is one of the more underrated of all diagnostic eye procedures. Lateral and vertical ranges of vergence ability, respectively, can be tested using a series of horizontal or vertical prisms arranged in a prism bar. Hence, the name *bar vergences*. The Maddox rod is an efficient and more accurate method for measurement of vertical deviations and vergences using loose prisms or a rotary prism. If necessary, two Maddox rods can be used to measure cyclodeviations and their associated ranges of cyclovergence. The closest distance from the spectacle plane to which the eyes can converge and maintain single binocular vision is measured by testing the near of point convergence (NPC).

The results of these tests through the habitual optical correction allow the practitioner to modify the procedures of the objective and subjective refractions (see Chapters 18 and 20) in order to achieve the most accurate refractive analysis possible. During phorometry (see Chapter 21), the data from these tests can be refined even further to the point that the final optical correction including refractive and prismatic components can be prescribed. The cover test and, perhaps, bar vergences are sometimes repeated through the predicted new

optical prescription toward the end of the examination, so as to confirm the potential correction's expected effects on the deviation and associated vergences at near or distance.

Observation of the Corneal Reflections

Angles Lambda (or Kappa)

Clinicians must objectively verify that the patient's eyes are looking in the direction that they are supposed to. One might conclude falsely that this is an easy assignment, given that the eye should be pointed directly toward the object of regard. Upon more critical inspection, however, the globe generally appears to be viewing at an angle temporal to the object of regard. This is because the optical components of the eye are not aligned with the line of sight, but along an optical axis at an angle temporal to it. The pupil, in particular, may not be centered exactly on the optical axis of the eye. Because the center of the pupil is an easy landmark to locate by visual inspection, a special "axis" has been assigned to it: The imaginary line normal to the cornea and containing the center of the pupil is called the pupillary axis. To identify the position of the line of sight, which has no anatomical landmark that is available to the clinician, its angular position is noted relative to the pupillary axis. The angle between the pupillary axis and the line of sight normally averages +5 degrees, ranging from +3 to +7 degrees, and is known as angle lambda. The angle is plus (+) when the line of sight is nasal to the pupillary axis (the usual situation) and negative (-) when temporal.

The route of the line of sight through the pupil can be located by observing the corneal reflex of the object of regard, which is in approximately the same plane as the entrance pupil. With the left eye occluded, typically using a handheld occluder, the patient is asked to fixate a penlight held in front of one of the clinician's eyes. The penlight is directed at the midpoint between the patient's two eyes from a distance of approximately 40 to 50 cm (Figure 10-15). While sighting just over the penlight, the clinician notes the position of the corneal reflection with respect to the center of the entrance pupil of the patient's right eye. The corneal reflex usually appears approximately 0.4 mm nasal to the center of the entrance pupil, because it has been shown that 1.0 mm of displacement corresponds to an eye rotation of 22°, or 12.5 degrees.⁶⁰ Switching the occlusion to the right eye, the position of the corneal reflection is noted with respect to the center of the entrance pupil of the left eye. Hence, the angle lambda is assessed for each eye according to the lateral (almost always nasal) displacement of the corneal reflex relative to the pupillary center.

Although the average position of the corneal reflex will be 0.4 mm nasal of center (+), there is some individual variability accounting for normal displacement



Figure 10-15

Assessment of angle lambda of a young patient's right eye, with the left eye occluded.

of the reflex from +0.25 mm to +0.6 mm, corresponding to angle lambdas of +3 to +7 degrees. Seldom will the angles be negative or zero (at the pupillary center) or equal to +1.0 mm or greater in eyes that are not fixating eccentrically. Angles lambda of the two normal eyes are rarely significantly different, such that the monocular reflex positions of the two eyes should be identical. If the location of the monocular reflex in one eye is significantly different than in the other, the clinician should suspect strabismus (see Chapter 31), which can be accompanied by reduced monocular visual acuity in the deviating eye. Angle kappa (between the visual and pupillary axes) was confused with angle lambda in the early literature, and this test is even today sometimes called a test for angle kappa. This distinction is clinically inconsequential.

The Hirschberg Test and Krimsky Method

With both eyes unoccluded and the patient still fixating the penlight, the clinician may note the positions of the corneal reflexes in both eyes under binocular conditions, and compare these with the corresponding positions noted under monocular conditions. This method of assessing the presence or absence of strabismus is called the Hirschberg test. When the eyes are unoccluded, the corneal reflexes should remain in their monocular positions unless a strabismus is present. In a strabismus one reflex will move away from its monocular position in evidence of a strabismic deviation. The magnitude of the deviation can be estimated by the amount of movement of the reflex in millimeters (1.0 mm = 22^{Δ}), or loose prisms of increasing power can be placed in front of the fixating eye until the reflex of the deviating eye has matched its monocular position relative to the pupillary center. The latter is the Krimsky method for measurement of the strabismic angle of deviation.

The Hirschberg test and the Krimsky method are relatively inaccurate in comparison with the cover test, noted later, but in certain situations they are the best methods available for identification and measurement of the strabismic angle of deviation. The tests are particularly suitable for infants and young children, or for those adults who cannot or will not respond or cooperate appropriately. Corneal reflexes are recorded on film during certain photorefractive screening techniques, and a binocular photograph is shown in Chapter 18 (see Figure 18-46). In the examination of a cooperating adult, the Krimsky method is of less value, because the cover test will ultimately measure the deviation within $\pm 2^{\Delta}$, but the Hirschberg test can serve to forewarn the clinician of a strabismic deviation prior to the cover test, and takes only a little time and effort in conjunction with the assessment of angles lambda.

Interpupillary Distance (IPD)

The entrance pupils determine the size and location of the bundles of light that enter the eyes and stimulate the retinas. The horizontal distance between the centers of the entrance pupils is called the interpupillary distance (IPD), or merely pupillary distance (PD). The IPD can be measured for distance fixation and also for near fixation. The "distance IPD" is useful for the horizontal placement of the optical centers of spectacle lenses before the entrance pupils of the eyes in primary gaze, such that the appropriate amounts of lateral prism are located before the eyes (see Chapters 23 and 24). Similarly, the distance IPD is used to separate the optical centers of the interchangeable lenses in the refractor during the subjective distance refraction (see Chapter 20). It is, thus, important for the clinician to accurately measure the IPD to later assess the visual system using other tests and to properly prescribe the optical correction. The distance IPD is related to the amount of binocular convergence required for bifoveal fixation of a target by the equation

$$\text{Convergence}(\Delta) = \frac{\text{IPD (in cm)}}{d'' (\text{in cm})}$$

where IPD is the interpupillary distance in centimeters (cm) for bifoveal fixation of a distant target, and d'' is the distance in meters (m) of the target plane from the midpoint between the centers of rotation of the eyes (Figure 10-16). The distance (d'') is, therefore, 0.013 m (13 mm) longer than the distance from the anterior corneal surface to the target plane (d'). The clinician should know that patients with extremely large distance IPDs have increased demands for convergence to a near target and that patients with small IPDs have reduced convergence demands at near. The mean distance IPD

for adults is 64 mm, and for children the IPD ranges from 50 to 60 mm.

The "near IPD" is the distance between the pupillary axes where they pierce the spectacle plane as the patient fixates a near target bifoveally. The near IPD is important in determining the nasal decentration of multifocal segments and the near zones of progressive-addition lenses (PALs) into their proper positions inferiorly before the eyes (see Chapter 23). Although the near IPD can be measured, there is a simple trigonometric relationship among the distance IPD, near IPD, vertex distance (vd), and distance (d) of the target plane from the spectacle plane (see Figure 10-16). By similar triangles, the following equation can be derived:

$$\text{near IPD} = \frac{d}{d''} \times \text{distance IPD}$$

where d'' is the distance of the target plane from the midpoint between the centers of rotation of the eyes (see Figure 10-16), which is longer than the distance from the spectacle plane to the target plane (d) by an amount equal to 13 mm plus the vertex distance. These above equations treat the pupillary axes as if they were lines of sight, but the relevance of the discrepancy is subclinical. The near IPDs for the average male and female patient are approximately 3.7 mm less than the corresponding distance IPDs, noted earlier. The difference between near and far IPDs does not reach 4.5 mm until the distance IPD is greater than 75 mm, and the difference does not fall to 3.0 mm until the distance IPD is 50 mm or less. Hence, multifocal segments are usually

decentered nasally by a standard 2.0 mm in each eye. The interpatient variation of the differences between near and far IPDs is generally subclinical and ignored.

Clinical Evaluation

The centers of the entrance pupils are thought to be difficult to precisely locate in the clinical situation, which is the primary reason why the Krimsky method is felt to be inaccurate. Hence, the identical distance between two anatomical landmarks on the eyes, usually the temporal pupillary margin in one eye and the nasal pupillary margin in the other, is most often used to assess the IPD. The distance between these landmarks is measured with a millimeter rule and should be equal to the distance between the pupillary centers if the pupils are symmetric. Alternatively, the temporal limbus of one eye and the nasal limbus of the other might be used, for instance, when anisocoria is present. The IPD can be determined for fixation at distance and at near.

To measure the distance IPD, the examiner should be standing or sitting directly in front of the patient at a distance approximating 40 cm. The examiner's eyes should be in the same horizontal plane as those of the patient. Room illumination should be sufficient for the examiner to identify the landmarks to be used for the measurement (i.e., the pupillary margins or limbi) and to read the inscriptions on a millimeter rule. The ruler, often called a "PD stick" in clinical parlance, is held horizontally in the patient's spectacle plane by one hand of the examiner, slightly below the pupils. The examiner closes the right eye and, with the other hand, points to his or her open left eye. The patient is instructed to fixate

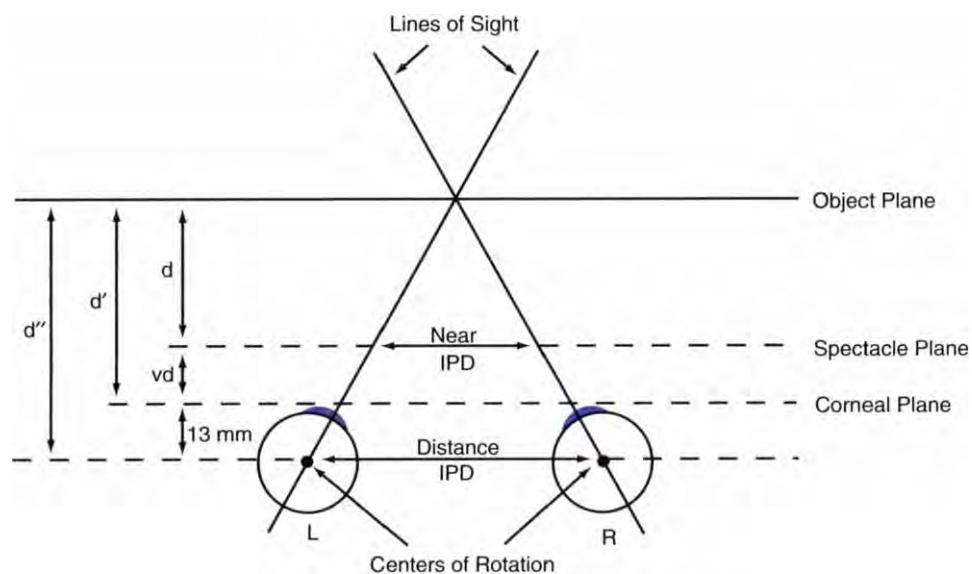


Figure 10-16

Relationship of the interpupillary distance (IPD) to convergence demand for a near target, and of the distance IPD to the near IPD.

bifoveally on the examiner's open left eye, while the examiner's left eye sights along the line of sight of the patient's right eye and aligns the zero mark of the ruler with the temporal pupillary margin or limbus of the patient's right eye (Figure 10-17). The examiner opens the right eye and closes the left, switching the pointing hand to delineate his or her right eye. The patient is asked to fixate bifoveally the examiner's open right eye. The examiner's right eye sights along the line of sight of the patient's left eye, and the distance IPD is specified on the ruler where it intersects the corresponding nasal pupillary margin or limbus of the patient's left eye.

The patient does not actually fixate a distant target, but the difference between the examiner's IPD and the patient's IPD is not enough to deviate the patient's eyes from viewing straight ahead by an amount that would alter the measurement significantly. The IPDs of patients who are strabismic can be measured by occluding the patient's left eye while the PD stick is being aligned with respect to the patient's right eye, and then by occlusion of the patient's right eye when the reading is determined on the left eye. In this way, both eyes are viewing straight ahead at the appropriate times during the measurement procedure.

The near IPD can be measured immediately at the end of the distance IPD procedure by keeping the ruler in the same position. The patient is instructed to refixate on the examiner's reopened left eye as the examiner closes the right eye and points to the left eye. The examiner confirms that the zero position on the ruler still marks the temporal pupillary margin or limbus of the patient's right eye. With his or her left eye, the examiner then sights across to the patient's left eye and notes the distance to the corresponding nasal landmark of the

patient's left eye. This is the patient's near IPD, which can be measured at any working distance used by the patient so long as the examiner's eyes are placed at that working distance. The IPDs are recorded in millimeters with the distance IPD listed first, followed by a slash, and then the near IPD (i.e., 62/58).

It is sometimes assumed that half of the distance IPD (the "split IPD") should be the horizontal distance from the center of the bridge of the nose to the center of the pupil on either side. However, the eyes are seldom located symmetrically relative to the nose, such that the true distance of the left eye from the center of the bridge is usually different than for the right eye of the same patient. This distance is better called monocular PD and should be specified for each eye instead of the distance IPD or split IPD when the clinical situation warrants a more accurate description of the positions of the pupillary centers. Precise monocular PDs are necessary for the prescription of highly powered spectacle prescriptions (see Chapter 33) and especially for PALs (see Chapter 24). Use of the split IPD persists because, as an approximation, it is often "close enough to get the job done" without having to measure the monocular PDs separately.

Monocular PDs can be measured using a PD rule to the center of each of the patient's pupils, in a manner similar to that described earlier. The patient fixates on the examiner's left eye, the examiner's right eye being closed, and the zero mark of the PD rule is aligned with the center of the patient's right pupil. The distance to the center of the patient's bridge is noted and becomes the right monocular PD. The patient then fixates the examiner's opened right eye, the examiner's left eye being closed, and the zero mark of the rule is moved to the center of the patient's bridge. The distance to the center of the patient's left pupil is noted and becomes the left monocular PD. When precise monocular PDs are required, this method may not be as accurate as necessary, because the exact positions of the pupillary centers and the center of the bridge of the nose are difficult to isolate. The monocular PDs are recorded in millimeters with the right PD listed first, followed by a slash, and then the left PD (i.e., 33/31).

Some practitioners prefer to use the corneal reflexes as landmarks for measurement in these procedures. Instead of using the eyes of the examiner as fixation targets for the patient, the examiner's pointing hand is used to hold a penlight as a fixation target immediately below the examiner's left and right eyes during the appropriate times during the measurements. Use of the corneal reflex eliminates the need to estimate the position of the center of the pupil. However, the corneal reflexes mark the positions of the lines of sight and, as has been noted, average 0.4 mm nasal to the center of the entrance pupil. Therefore, measurement by corneal reflection will usually underestimate the monocular PD by 0.2 to 0.6 mm in each eye or the IPDs by a total of



Figure 10-17

The millimeter ruler is zeroed on the temporal limbus of a young patient's right eye during the measurement of the distance IPD. The patient is fixating the examiner's left eye.

0.4 to 1.2 mm, depending on the individual patient's angles lambda.

A special instrument called a pupillometer is recommended when monocular PDs are a necessity. Several pupillometers are available that measure to either the centers of the pupils or the corneal reflexes, depending on the instrument, as reviewed by Brown.⁶¹ These are optical devices that each have a spectacle-like padded bridge that rests on the bridge and upper sides of the nose during measurement. Thus, the center of the bridge of the nose is precisely located with respect to the two optical pathways of the instrument. The devices can be battery-operated and present a fixation light directly in front of each eye of the patient along the sighting paths of the examiner. A reticle projected at the plane of the patient's pupil allows measurement of the monocular PD by alignment of the reticle with the center of the corneal reflex or the center of the pupil. The distance IPD is a simple addition of the two monocular PDs. Some pupillometers allow adjustment of the two optical pathways to ascertain the near IPD. A pupillometer is shown in Chapter 24 (see Figure 24-40).

The Cover Test

The "cover test" is an objective method of evaluating and measuring the deviation of the lines of sight from those directions necessary for bifoveal fixation of a target. It is a test that is unnecessary in the monocular patient but of much importance in the binocular patient. The test is accomplished in three segments: (1) observation of fixation, (2) the unilateral cover test, and (3) the alternating cover test, with measurement of the deviation using loose (Figure 10-18) or bar prisms. The other equipment necessary for the cover test are an

occluder, or "cover paddle" (Figure 10-19), and targets for distance and near viewing. The clinician is seated beside the patient, and in front of the patient by a short distance of perhaps 25 to 40 cm (Figure 10-20). The clinician must be close enough to be able to critically note movements made by the patient's eyes yet not block the vision of the patient. The room must be well lighted to promote visual inspection of the eyes.

The cover test is performed with the patient wearing his or her habitual optical correction (spectacles or contact lenses) in the early phases of the eye examination (see Figure 10-20). The clinician may wish occasionally to do a cover test without the correction, or with an updated correction in the trial frame nearer the



Figure 10-19

A typical occluder or "cover paddle," above in this photograph, which is often accompanied by a Maddox rod at the other (lower) end.



Figure 10-18

A set of loose prisms used in the cover test. Note the red lens at the left, which can be used in the red lens test for incomitancy and the Maddox rod test for vertical phoria.



Figure 10-20

Performance of the unilateral cover test with the patient fixating a distant target. Note that the spectacle correction is being worn and that the clinician is in a position to closely monitor the movements of the eyes.

end of the examination. The patient fixates a target at distance, which can be an isolated letter a few lines above threshold in the poorer eye, usually 20/25 (6/7.5) to 20/40 (6/12), or an equivalent target. The patient should be asked to follow the target with the eyes if it appears to move.

Observation of Fixation

The ability to fixate should have already been assessed by the clinician earlier in the eye examination (see discussions of angles lambda and the Hirschberg test). Nearly all patients except for the very young, anxious, hyperactive, or inattentive should be able to sustain steady fixation for 10 seconds or more, monocularly and binocularly, which is sufficient to conduct the cover test. At the beginning of the cover test, the clinician should watch the right eye to see if it can maintain steady fixation at distance while the left eye is covered with an occluder for several seconds. Upon removal of the occluder, the right eye should maintain fixation. Switching occlusion to the right eye, the left eye should attain fixation at distance and maintain it when the contralateral occlusion is removed.

If latent nystagmus or reduced central vision keeps monocular fixation from being steady, it becomes more difficult or impossible (depending on the severity) to assess the necessary eye movements in the upcoming segments of the cover test. Unsteady monocular fixation can also be an indication of eccentric fixation. Eye movement in the fixating eyes should not occur immediately after removal of the contralateral occlusion in patients capable of bifoveal fixation. The formerly occluded eyes, showing heterophoria (or merely "phoria") will move to take up fixation after the occlusion is ended unless the eyes have zero phoria (orthophoria). If movement of either or both of the fixating eyes is detected immediately after removal of the contralateral occlusion, strabismus may be present. The strabismus, also called heterotropia (or merely "tropia") will be categorized and measured during the rest of the cover test. Hence, the initial segment of the cover test determines if fixation is sufficient to conduct the rest of the segments and tentatively establishes whether phoria or tropia exists.

Unilateral Cover Test

The unilateral cover test confirms the presence of a phoria or tropia and defines its component directions (eso, exo, hyper, or hypo). The knowledgeable practitioner can also obtain a general idea about the deviation's magnitude. In the case of a strabismus, the unilateral cover test further classifies the tropia as alternating or unilateral and, for the latter, in which eye the deviation is manifested. The strabismus may also be characterized as constant or intermittent. The cover test can be performed in different directions of gaze for

assessment of incomitancy, but such testing is not a part of the routine cover test. Testing for incomitancy should be performed in all cases of strabismus, as noted earlier in this chapter.

The unilateral cover test is accomplished by observing the movement of the fixating eye when the other eye is first covered with the occluder for 2 or 3 seconds and then by observing the movement of both eyes immediately after the contralateral occluder is quickly removed (see Figure 10-20). The eye movements are assessed while the right eye fixates the distant target, covering and uncovering the left eye, and then while the left eye fixates, covering and uncovering the right eye. It is important to allow the eyes to fuse the target, if possible, before switching the cover paddle from one eye to the other. In practice, it is not possible to pay attention to both eyes at the same time when uncovering an eye. Hence, the clinician covers and uncovers an eye through several cycles, first watching the fixating eye for a few cycles, then the other for a few cycles, in order to evaluate the movements of both.

When fusion is broken by the occluder, the occluded eye will assume its normal tonic vergence position while the unoccluded eye will either: (1) remain fixating the target or (2) take up fixation of the target. The movement of the occluded eye will not be visible to the clinician. When the contralateral occlusion is removed, the fixating eye then either (3) remains fixating the target or (4) gives up fixation to the formerly occluded eye. Simultaneously, the formerly occluded eye (5) will take up fixation of the target, (6) will remain deviated, or (7) may not move if orthophoric. On the basis of these eye movements, or the lack thereof, the clinician reaches a diagnosis of the type of phoria or tropia present and deduces the direction in which the occluded eye's line of sight was pointed during the occlusion.

In phorias, only the eye that is covered moves. It is obvious that, in orthophoria, neither eye will move after the contralateral eye is occluded or unoccluded. In esophoria, the occluded eye will adduct to its tonic vergence position, and once occlusion is removed, it will be seen to abduct (move temporally) as it takes up fixation along with the other eye. In exophoria the occluded eye will abduct to its tonic vergence position and will be seen to adduct (move nasally) when the occlusion is removed. The same response will be generated regardless of the eye that is occluded. Hence, a phoria is in the direction opposite to the movement seen upon removal of the eye's occlusion. The experienced practitioner will be able to assess the relative magnitude of the phoria by the amount of eye movement that is observed. However, definitive measurement of the phoria's magnitude will be performed later during the alternating cover test.

The eye with hyperphoria will move down as it is uncovered, and the other eye will appear to have

hypophoria, because it will move upward when uncovered. Vertical phorias will most likely be much less in magnitude than horizontal phorias, and the eye having the hyper or hypo deviation must be specified. A hyperphoria in one eye is indistinguishable from a hypophoria in the other eye. A combined phoria, with both lateral and vertical components, may also be present. No matter the direction of the phoria, however, the eye not being covered should remain stationary as the other eye is covered and uncovered.

In a unilateral strabismus, when the deviating eye is covered and uncovered, the fixating nonstrabismic eye will remain stationary, as will the strabismic (deviating) eye. If the nonstrabismic fixating eye is covered, the strabismic eye will move to take up fixation. Upon removing the cover from the nonstrabismic eye, the strabismic eye will give up fixation to the formerly occluded eye, and both will move: The nonstrabismic eye reassumes fixation, and the strabismic eye regains its deviated position. The clinician, then, is able to identify the strabismic eye, the direction of the deviation, and the relative magnitude of the deviation. Hence, the deviation can be classified as left or right, esotropia or exotropia, and hypertropia or hypotropia. Unlike phorias, unilateral hypertropia in one eye is different than unilateral hypotropia in the other eye.

In an alternating strabismus, the eyes are able to each assume the role of fixation under binocular conditions. When a deviating eye is covered and uncovered, the eyes remain stationary. When a nondeviating eye is covered, the other eye moves to assume fixation. The difference between the alternating strabismus and a unilateral strabismus is that when the formerly nondeviating eye is now uncovered, the eyes will both remain stationary. Based on the movements of the eyes when the deviating eyes are covered, the strabismus can be classified as an esotropia or exotropia, hypertropia or hypotropia. Alternating tropias are not specified for the left or right eyes unless a vertical deviation is present. Like phorias, an alternating hypertropia in one eye is indistinguishable from an alternating hypotropia in the other eye.

An alternating strabismus can fool the practitioner if the patient is one who anticipates the eye that will be used for fixation. It is common for an alternating strabismic to switch fixation from the eye that appears about to be covered, to the eye that will not be covered. This is usually not malingering, but a mechanism that allows the patient to maximally use his or her visual capabilities in normal life. The result is that the unilateral cover test will falsely appear to indicate orthophoria, unless the clinician perceives that an eye is deviating (e.g., during the Hirschberg test) or recognizes that the eyes are both moving as the cover paddle is brought toward a deviating eye.

Preparation for the covering of an eye is easily accomplished by the alternating strabismic when the clinician

brings the cover paddle toward the eye to be covered from the temporal side. One way of minimizing this problem is to center the cover paddle over the forehead of the patient and to cover the appropriate eye from above by moving the paddle down and out. Or, the clinician could center the cover paddle over the nose and suddenly cover one eye or the other by moving the paddle up and out. In these manners, the patient may not predict which eye will be occluded. If the clinician ever reaches the alternating cover test (see later), believing an orthophoria to exist, and then measures an unexpectedly large deviation, the chances are that an alternating tropia was missed earlier. The clinician will need to go back and more critically perform the unilateral segment of the cover test.

A constant tropia appears always as a tropia when tested at the same distance. If the deviation appears to be tropia in some instances during the cover test and appears to be phoria on other instances or occasions, the tropia is intermittent.

The expert clinician should be aware of a few conditions, other than phorias and tropias, that can cause eye movements during the unilateral cover test. A small flick of an eye may occur, prior to taking up fixation, when a large phoria is broken by occlusion of the other eye. In these cases the phoria may actually be an intermittent tropia or a small-angle strabismus called a microstrabismus or microtropia. Eccentric fixation and unsteady fixation can also cause movement, but these should have been detected previously. Uncorrected or residual anisometropia can create the illusion of esophoria or esotropia because of greater accommodative convergence initiated by the more hyperopic eye.

Alternating Cover Test

The alternating cover test confirms the direction and measures the magnitude of a phoria or tropia. The alternating cover test begins immediately after the unilateral cover test has ended, using the same occluder, target, and patient/clinician relationship. Fusion is broken by covering the right eye for 2 to 3 seconds and allowing the left eye to take up fixation, if necessary. The paddle is then quickly moved, without pausing between the eyes, to cover the left eye for 2 or 3 seconds while the right eye takes up fixation. Binocular fixation is not permitted as in the unilateral cover test. The paddle is repeatedly alternated from one eye to the other, pausing 2 or 3 seconds over each eye to allow for alternation of fixation. The clinician observes the direction and relative magnitudes of the eye movements as each eye is alternately uncovered and takes up fixation of the target.

Regardless of whether a phoria or tropia exists, the eyes will alternately each move temporally to take up fixation as occlusion is alternately removed if a deviation exists in the "eso" direction, and the eyes will move nasally in the case of an "exo" deviation. An eye will



Figure 10-21

Performance of the alternating cover test with loose prisms to neutralize the deviation, with the patient fixating a near target held in her left hand. This photograph shows the cover paddle after it has been moved to cover the left eye from its initial position covering the loose prism and right eye.

move downward for a "hyper" deviation, in which case the other eye will move upward, indicating a corresponding "hypo" deviation. These results should confirm the directions of the deviations that were diagnosed as a result of the unilateral cover test. The clinician should have an estimate of the magnitude of the deviation on the basis of the amount of eye movement seen during the first portions of the cover test. The clinician then proceeds to the measurement of the deviations using loose prisms or a prism bar (see Figure 21-11).

The occluder is left in place before one of the eyes and a loose prism (or a bar prism as shown in Figure 21-11) of the estimated magnitude and direction is placed before the occluded eye (under the cover paddle). The flat posterior surface of the prism should be placed in the spectacle plane of the eye or immediately in front of a spectacle lens worn by the patient. The magnitude of the deviation is measured by neutralization of the eye movements during the alternating cover test with prisms of increasing power until the residual deviation is zero. This amount of prism is noted, then the prism is increased until the deviation is reversed and bracketed (Figure 10-21). An "eso" deviation is neutralized with base out (BO) prism, an "exo" deviation with base in (BI) prism, a "hyper" deviation with base down (BD) prism, and "hypo" deviation with base up (BU) prism. Prismatic power is always increased incrementally underneath the cover of the occluder, which is kept before one eye as loose prisms are exchanged. The reason that unilateral occlusion is maintained is to discourage any prior latency from redeveloping should bifoveal fixation be allowed.

The accuracy of this technique is influenced by the fact that the minimum deviation recognizable by the clinician is about 2^{Δ} . The manifest deviation is that neutralized when eye movement is first eliminated. Between the last minimum detectable eye movement in one direction and the first recognition of reversal, there is usually a range of 2 to 4^{Δ} over which no eye movement is observed. The amount of the latent deviation is taken to be the midpoint of the bracketed range. If orthophoria is apparent, reversal should be achieved in both lateral directions with BI and BO prism.

Some patients have both lateral and vertical component deviations. To assess both magnitudes, BO or BI loose prisms are used to first neutralize the eso or exo deviation. Loose vertical prisms can then be held over the horizontal prism (or the horizontal prism can be held by the patient over the other eye) for neutralization of the remaining vertical deviation. Although the precision of prismatic neutralization is adequate for lateral deviations ($\pm 2^{\Delta}$), it is not at this point generally acceptable for vertical deviations. Hence, some methods of enhancing the precision of the alternating cover test are to be described.

Small eye movements during the alternating cover test are often difficult to discern. A low power (approximately 5^{Δ}) loose prism can be used to find and measure small angle (3^{Δ} or less) deviations and is particularly useful when orthophoria is suspected or when a small vertical deviation requires more accurate measurement. The prism is inserted BI over one eye during the alternating cover test, and the magnitude of the residual esophoric deviation is observed. The prism is then inserted BO over the same eye, and the magnitude of the residual exophoric deviation is observed. If the eye movements appear to be of the same magnitude in the two prismatic orientations, but opposite in direction, lateral orthophoria truly exists. Exophoria exists if the eye movements appear lesser when the prism is oriented BI, and esophoria exists if the movements are lesser when the prism is BO. The experienced practitioner can estimate the magnitude of the small lateral deviation from the relative amounts of eye movements when the prism is placed BI versus BO. Similarly, the prism can be oriented BU and BD to rule out vertical orthophoria or to measure a small vertical deviation.

It is also possible in such a situation that the patient's manifest phoria could be from 3 to 7^{Δ} or be latent to an even larger extent. The practitioner might not see eye movement when the 5^{Δ} prism is placed before the eye in a manner that will correct the deviation. This could occur, for instance, if the patient is 3 to 7^{Δ} esophoric and the 5^{Δ} prism is placed over the right eye BI and then BO to observe the direction and magnitude of eye movement. The clinician would likely see a large amount of eye movement through BI prism and no movement with BO prism. The large amount of movement in one direction and the lack of movement in the other direction

should tip the clinician that the deviation is approximately 5^{Δ} and that a prism of larger magnitude will be necessary to achieve reversal in the latter.

It is important that the movements of the eyes be reversed when the BO (or BU) prism is used in comparison with the BI (or BD) prism. If reversal does not occur, the angle of deviation is likely to be latent and larger than the correcting prism. The clinician should proceed by increasing the amount of prism in the orientation of nonreversal until a reversal has occurred. The latent phoria in some cases may be significantly larger than the manifest phoria, especially for lateral deviations. A common error in the measurement of a phoria deviation, seen so often by one of the authors (WJB), is to omit the reversal of the deviation. A lateral orthophoria is not proven to be orthophoria until reversed in both horizontal directions.

Deviations can also be more accurately detected subjectively by asking the patient to identify the perceived movement of the target as the cover paddle is moved from one eye to the other. Immediately after the cover paddle is moved from the right to the left eye, and before a fixational eye movement has been made, the target previously seen by the fovea in the left eye is now imaged at a point on the retina of the right eye. If the eye is deviated in an eso direction, the image is located on the nasal retina and is projected into the patient's temporal visual field. If deviated in an exo direction, the image is located on the temporal retina and is projected into the nasal visual field. Hence, the patient will perceive that the target moves "with" the cover paddle when there is exophoria and "against" the paddle when there is esophoria. The clinician can neutralize the perceived movement of the target by addition of the appropriate prisms before the eyes in a manner identical to that described earlier. Vertical phorias may be identified and neutralized subjectively in this manner as well, allowing for the enhanced precision of measurement necessary for small vertical phorias ($\pm 1^{\Delta}$). Alternatively, even more precise vertical measurements may be achieved using the Maddox rod ($\pm 0.5^{\Delta}$), a technique especially recommended by the authors and explained later in this chapter.

After the entire cover test has been performed at distance, the procedure is repeated at near. The patient should fixate a single letter, one line above the threshold of the poorer eye, or an equivalent target at his or her habitual working distance. The common working distance (40 cm in front of the spectacle plane) is used in many instances. For children, a picture may be used as long as it contains enough detail to attract the attention of the patient and stimulate accommodation. It is beneficial to have the patient hold the target at the appropriate working distance so that both of the clinician's hands are free to manipulate the cover paddle and prisms (see Figure 10-21). A better stimulus to accommodation and fusion will also be achieved when the

near target is held by the patient. An overhead lamp should be directed to increase the illumination of the near target. The patient should be asked to follow the target with the eyes if it appears to move and be reminded during the procedure to keep the near target clear, such that accommodation is kept relatively stable. Otherwise, the procedure at near is the same as the procedure at distance.

Recording

Results of the cover test should be reported for distance and near. The magnitude in prism diopters, direction (eso, exo, hyper, hypo), and type of deviation (phoria or tropia) should be expressed. If a vertical deviation or unilateral tropia, the laterality of the deviation must be signified. The word "alternating" must be inserted if the condition is an alternating tropia. The frequency of a tropia (i.e., "constant" or "intermittent") and the comitancy of a tropia should be indicated (i.e., "comitant" or "incomitant"). Some examples: 4^{Δ} exophoria (4^{Δ} XP); 2^{Δ} right hyperphoria (2^{Δ} RHyperP); 3^{Δ} eso + 1^{Δ} left hyperphoria (3^{Δ} EP + 1^{Δ} LHyperP); 8^{Δ} constant comitant alternating esotropia (constant comitant 8^{Δ} Alt ET); 12^{Δ} right intermittent incomitant exotropia (intermittent incomitant 12^{Δ} RXT); 6^{Δ} left constant comitant alternating hyper-tropia (constant comitant 6^{Δ} Alt LHyperT); 8^{Δ} exo + 4^{Δ} right constant incomitant hypotropia (constant incomitant 8^{Δ} XT + 4^{Δ} RHypoT).

Cyclophorias and Cyclotropias

The reader may note that the methods of measurement of cyclodeviations have not been mentioned previously in this chapter. It is generally believed that cyclophorias seldom alone contribute to muscle balance problems at distance or at near. It is apparent that a significant cyclophoria must accompany a simultaneous vertical deviation and is almost always associated with a paretic superior oblique. Unilateral paresis of a superior oblique may result in a cyclodeviation of 3 or 4 degrees, whereas bilateral paralysis can triple or quadruple that amount. Therefore, a critical screening for a vertical deviation using the cover test or the Maddox rod (see later) also serves to screen for a cyclodeviation. No optical devices can be reasonably prescribed for the alleviation of a cyclophoria. Hence, the reader is referred to Chapter 20 for a discussion of how fusional torsions may affect the astigmatic axis during the monocular and binocular refractions at distance and at near, and to Chapter 21, in which a detailed procedure for cyclophoria measurement with dual Maddox rods is presented.

Bar Vergences

Bar vergences are performed to measure the fusional (disparity) vergence reserves that the patient has available to obtain and maintain bifoveal fixation in the presence of a binocular deviation. The technique is

covered in Chapter 21, and a photograph is included as Figure 21-11. The basic relevance of the blur, break, and recovery findings was discussed in Chapter 5. The expected clinical findings, specific evaluations, and overall patient assessments are covered in detail in Chapters 21 and 22. Blur, break, and recovery findings are determined in the horizontal plane at distance and at near. Break and recovery findings may be determined in the vertical (sagittal) plane, but vertical vergences are usually only pursued at a single distance.

Bar vergences are performed "outside of" a phoropter and are analogous to vergences performed with rotary prisms with the phoropter, which are also described in Chapter 21. The effects of proximal convergence on the horizontal vergence amplitudes are reduced for bar vergences in comparison with rotary prism vergences and, as a result, bar vergences are thought by many practitioners to better represent the natural viewing situation. On the other hand, bar vergences require stepped increments of prism to be placed before a single eye, whereas rotary prisms split continuous increases between the two eyes and are less likely to disrupt vision during the prismatic alterations.

The Maddox Rod

The Maddox rod can be used to evaluate lateral phorias and cyclophorias as described in Chapter 21. However, it is especially well suited for the measurement of vertical deviations at this stage of the eye examination. A Maddox rod is usually present on the opposite end of a cover paddle (see Figure 10-19) and is placed before one eye with its baffles oriented vertically (Figure 10-22, A) as the patient fixates on a penlight at 40 cm in a darkened room. The Maddox rod is usually placed over the

right eye. A red lens may or may not be placed before the left eye to equalize the color and luminance of its view. The test can be performed at distance by shining a bright spot on the wall in a dark room, but it is usually only performed at near because this is more convenient and because vertical phorias should not significantly alter from distance to near.

The Maddox rod will break fusion, and the nonsuppressing patient should perceive a red horizontal streak (Figure 10-22, B) in the right eye covered by the Maddox rod and a bright white spot of light with the other (left) eye. The spot of light will appear red if a red lens was placed before the left eye. If the eyes are vertically orthophoric, the patient will perceive the bright spot of light to be directly in the middle of the red streak. If the streak runs above (right hypo = left hyper) or below (right hyper = left hypo) the bright spot, loose prisms or a rotary prism (Figure 10-23) can be used to neutralize the deviation by bracketing of residual orthophoria. When the streak apparently touches the bright spot the magnitude of the vertical phoria is approximately 1.0^{Δ} and when overlapping but not centered the phoria is approximately 0.5^{Δ} . A detailed procedure is discussed in Chapter 21.

The precision of this technique ($\pm 0.5^{\Delta}$) is such that misadjustment of the typical spectacle correction interferes with accurate measurement of the vertical phoria. It is best to measure the phoria without the habitual spectacle correction in place (contact lenses are okay) if the true vertical phoria is the end point desired. Residual vertical phorias induced by poor frame fit or optical center placement can be ruled out or verified by comparing the vertical phorias measured with and without wearing of the suspect spectacles. If verified, the clinician can then go about eliminating the problem by adjustment or re-order of the optical correction.

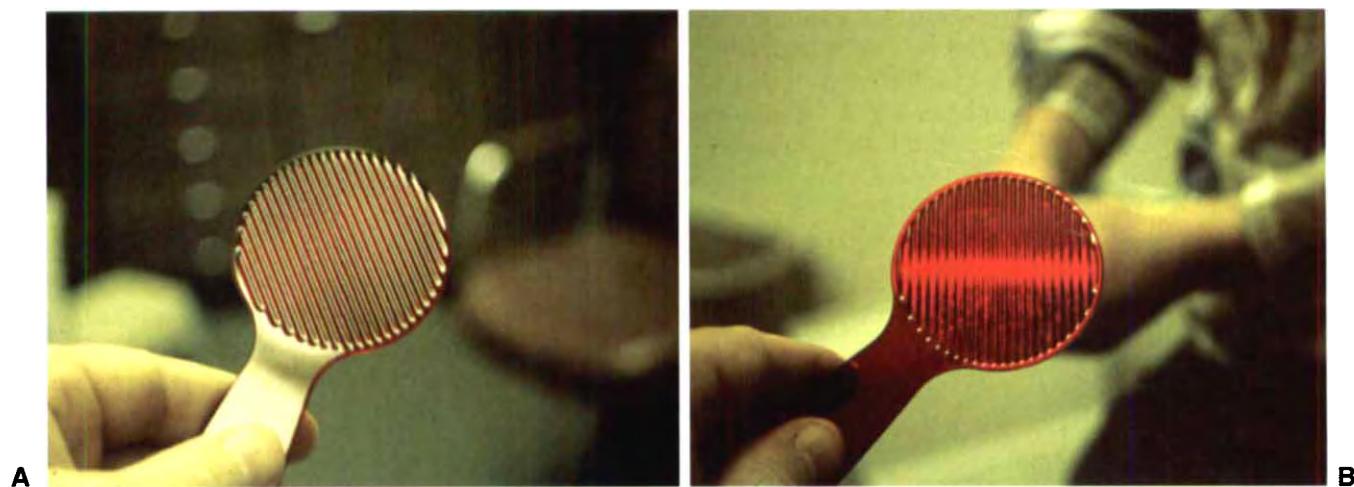


Figure 10-22

The baffles of a red Maddox rod present on one end of a cover paddle (A). When placed over the patient's eye with the baffles vertical, the penlight is seen as a horizontal streak perpendicular to the baffles (B).

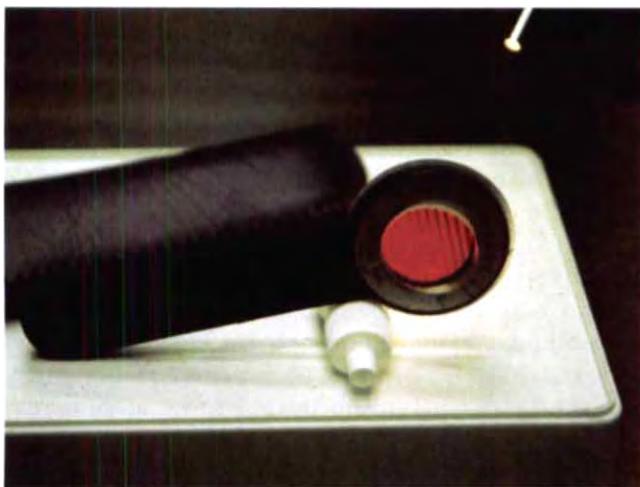


Figure 10-23

An efficient device for testing of vertical phorias, this pocket-size Maddox rod has incorporated an adjustable rotary prism and is accompanied by a handy leatherette case.

Near Point of Convergence

The near point of convergence (NPC) is the point of intersection of the lines of sight when maximum fusional (disparity) convergence is used. The distance to this point from the spectacle plane, typically taken to coincide with the middle of the forehead, is the "NPC finding." The NPC is related to the patient's ability to converge the eyes while simultaneously maintaining bifoveal fixation. Binocular vision problems, eyestrain (asthenopia), discomfort in performing near work, and reading difficulties may occur in persons with inadequate NPC findings. As a result, the NPC finding is used as a screening test for obvious convergence insufficiencies.

The NPC should be found with a nondescript target, such as a penlight or another simple target on a tongue depressor (Figure 10-24), to help isolate the fusional (disparity) vergence response from accommodative convergence. The accommodative and vergence subsystems are tightly cross-coupled⁶² with an accommodative response normally accompanied by vergence eye movement. Using an accommodative target stimulates accommodative demand and accommodative convergence, which will affect (lower) the expected values for the NPC and recovery.⁶³

The examination room should be fully illuminated, and the patient should be wearing the habitual optical correction. The examiner should be positioned slightly off to one side of the patient but in a manner that allows observation of the patient's eye movements. The target is held approximately 50 cm away at the patient's eye level and is brought slowly along the midline toward the midpoint between the patient's eyes. The patient is



Figure 10-24

Finding of a young patient's near point of convergence (NPC) by pushing up a near target until one eye deviates or the patient reports diplopia. With experience the clinician can accurately estimate the distance from the plane of the target to the midforehead without having to actually measure the distance each time.

instructed to follow the target inward with the eyes, as closely as possible, and to report if the target doubles or "breaks into two." The NPC is that point reached when the patient reports diplopia or when the examiner first observes loss of bifoveal fixation by the outward turning of one eye (see Figure 10-24). The target is then backed away from the eyes and the clinician notes the distance at which the deviating eye regains fixation. This is called the NPC recovery finding.

The patient should be instructed to ignore any blurring of the target and to continue fixating the target even though considerable blur may result as the maximal accommodative output is overcome. Indeed, the NPC finding is obtained in a similar fashion to that of the binocular accommodative amplitude (discussed later), except that the NPC finding uses a target without fine detail to lessen the stimulation of accommodation, and the end point is loss of single binocular vision instead of the introduction of blur (see Chapter 21).

The patient may suppress the deviated eye when the NPC is passed. Fusion is lost and, in these instances, diplopia will not be reported. As the target is brought closer, it assumes a position in the nasal visual field of the fixating eye. To continue fixation, that eye turns in and the suppressing eye concomitantly turns out as the vergence becomes a version. Hence, the clinician must be vigilant in monitoring the eyes for movement as the target is brought closer and closer to the top of the nose. The anteroposterior distance from the middle of the forehead to the plane of the target is measured with a ruler and recorded in centimeters to within 0.5 cm. If the patient is able to converge to the point that the target

comes in contact with the patient's nose, the NPC finding is often recorded as "nose."

The mean NPC is 3 cm (± 4 cm) from the spectacle plane (midforehead), and the recovery finding is 2 or 3 cm larger. NPC findings greater than 7 cm and recovery findings greater than 10 cm are generally regarded as inadequate and could be signs of a convergence insufficiency. Further testing and analysis would be warranted at near in the area of muscle balance and accommodation (see Chapters 21 and 22). The test may be repeated several times to check for fatigue, which is indicated by an increase of 3 cm or more between the first and last repetitive NPC findings.

ACCOMMODATIVE AMPLITUDES AND FACILITY

Accommodative Amplitudes

Accommodative insufficiencies (see Chapter 4) can occur with normal and abnormal nervous innervation. Autonomic parasympathetic fibers from the Edinger-Westphal nucleus innervate the ciliary muscle which controls accommodation, but cortical processes preceding and monitoring the ciliary motor command signals are poorly understood.⁶⁴ Screening tests for accommodative insufficiencies are typically addressed after the distance refractive correction has been determined, and their procedures are covered in detail in Chapter 21. However, some tests can be performed during the assessment of ocular motility in the early stages of the eye examination, if the refractive correction is not expected to alter considerably from that of the habitual correction. The accommodative amplitude, in particular, is measured with a technique that closely parallels the method used to assess the NPC.

The measurement of monocular and binocular accommodative amplitudes is discussed in Chapter 21, and the reader is referred there for the detailed procedures. The accommodative test uses a more finely detailed target to better stimulate accommodation in the finding of the near point of accommodation, and the end point is first sustained blur instead of diplopia (see Chapter 21). The reciprocal of the distance from the midforehead (approximately the spectacle plane) to the near point of accommodation, in meters, is the amplitude of accommodation, in diopters.

Monocular and binocular results should be recorded in the order and number of times they are measured (i.e., OD 7,7,6; OS 8,7,8; OU 7.5). The monocular amplitudes are measured along the line of sight in primary gaze, with the contralateral eye occluded, and the binocular amplitude is measured along the midline. This ensures that the binocular amplitude will normally be artifactually greater than the monocular amplitudes,

because the distance to the target will be shorter in the monocular measurement for a given dioptric amplitude value (see Figure 10-16). Accounting for this artifact of measurement, however, the binocular amplitude is only slightly greater than the monocular amplitudes by a fraction of a diopter. This minimal effect is thought to be the result of the lack of convergence accommodation in the monocular situation. The difference between monocular and binocular amplitudes of accommodation is sub-clinical unless an accommodative abnormality exists. Accommodation is consensual and equal in the two eyes, so a difference of 1 D or more between the eyes may indicate a unilateral insufficiency. Further accommodative testing would be indicated (see Chapter 21).

Hofstetter⁶⁵ derived formulas for the expected maximum, mean, and minimum accommodative amplitudes in the population from the normative data of Duane⁶⁶ and Donders,⁶⁷ a topic covered in great detail by Borish.⁶⁸ The formulas were based on age. Accommodative insufficiency should be suspected in patients with amplitudes less than the values calculated from Hofstetter's formula for the *minimum* accommodative amplitude (Table 10-4):

Expected *minimum* amplitude =

$$15.0 \text{ D} - [0.25 \text{ D} \times (\text{age in years})]$$

Expected *mean* amplitude =

$$18.5 \text{ D} - [0.30 \text{ D} \times (\text{age in years})]$$

Expected *maximum* amplitude =

$$25.0 \text{ D} - [0.40 \times (\text{age in years})]$$

The clinical measurement of accommodative amplitudes may be affected by visual acuity, target size and detail, depth of focus, patient effort, blur interpretation, ability to converge, refractive state, spectacle lens effects, and examiner technique. Although full room lighting is desired, excessive light should be avoided because of pupil constriction with resulting increased depth of

TABLE 10-4 Expected Accommodative Amplitudes vs. Age, as Determined with the Use of Hofstetter's Formulas

Age	EXPECTED AMPLITUDES (D)		
	Minimum	Mean	Maximum
10	12.5	15.0	21.0
20	10.0	12.5	17.0
30	7.5	9.5	13.0
40	5.0	6.5	9.0
50	2.5	3.5	5.0
60	0	0.5	1.0

focus, which can increase the measured amplitude. Uncorrected refractive errors will alter the location of the near point of accommodation: Uncorrected hyperopes will have erroneously low amplitudes, and uncorrected myopes will appear to have greater amplitudes, than would be the case with the proper refractive correction.

In addition to the effect of age, amplitudes may also be reduced by disease, drug reactions, or functional problems. Illnesses such as mumps, measles, influenza, anemia, and encephalitis may reduce amplitudes. Multiple sclerosis and myotonic dystrophy can have a similar effect. Transient accommodative paresis may occur in diabetics. Atrophy of the ciliary body in some glaucomas may produce accommodative problems. A lesion in the Edinger-Westphal nucleus or pineal tumors can cause reduced accommodation. Iridocyclitis, sinus problems, focal infections, dental caries, or injections may be suspected in unilateral deficiencies. Trauma to the craniocervical region, often seen in whiplash, may also be responsible for bilateral problems, whereas trauma, in the form of a tear in the iris sphincter or the zonules of Zinn, might reduce a monocular measurement.^{69,70} Systemic drugs such as alcohol, central nervous system stimulants and tranquilizers, antihistamines, tricyclic antidepressants, and phenothiazines may lead to bilateral accommodative insufficiencies.⁶ Topical agents such as cycloplegics may have unilateral or bilateral effects, depending on their administration. If a unilateral decrease in accommodation is noted in conjunction with a dilated pupil, Adie's tonic pupil and 3rd cranial nerve problems need to be ruled out.

Accommodative Facility

The ability to alter accommodation rapidly and accurately is called accommodative facility. The evaluation of accommodative facility is discussed in Chapter 21, and the reader is referred there for discussion of the detailed procedure. Briefly, the clinician asks the patient to repeatedly alternate vision between a distant and a near (40 cm) target, both being slightly above the acuity threshold of the patient (approximately 20/30 or 6/9), making each target clear before the ocular focus is immediately changed to the next target. With both targets well illuminated, the patient verbally indicates when a target becomes clear after alternating focus from one target to the other, then reverses focus to the original target and reports when it again becomes clear. Alternatively, flipper bars of lenses +2.00 OU and -2.00 OU can be used to alternate the accommodative demands required to clear binocularly a near target.

The clinician notes the number of full cycles per minute (cpm) completed in 60 seconds binocularly and monocularly in each eye and identifies with which target (distance or near) the patient may be having a problem. Is there an accommodative facility insuffi-

ciency and, if so, is the problem with relaxing accommodation or with increasing accommodation? The minimum value for adults is 12 cpm binocularly. Monocular findings should be approximately 2 or 3 cpm faster (higher) than the binocular findings. The actual number of cycles completed by the patient should be recorded in cycles per minute (i.e., facility: 6 cpm OU, 5 cpm OD, 6 cpm OS).

Facility problems may result in difficulty focusing from distance to near, or vice versa. Students may complain about not being able to see the blackboard after near-point activities. Older patients report blur of distance objects and extensive time needed to clear near targets. Asthenopia, eye rubbing, and blinking are common with infacility. Aging of the crystalline lens will decrease facility, as will diabetes, Grave's disease, measles, and chronic alcoholism. Patient's with Adie's tonic pupil may show unilateral facility problems. Systemic medications with cycloplegic side effects can negatively influence facility results.

SUMMARY

The eye examination includes an assessment of ocular motility, usually performed in the early stages of the examination with handheld instruments or devices, and before the accommodative system and pupils are topically paralyzed by a mydriatic. Most clinicians develop routines for the series of ocular motility tests, such that they can be done in an efficient manner. During their performance, the astute practitioner may simultaneously conduct a gross external examination of the eyes, overall physical examination, and/or a friendly and engaging verbal case history. Confrontation visual fields (Chapter 15) can be skillfully performed immediately after observation of versions with the "H pattern" since the position of the clinician, visual target, and general format of these two tests are similar.

The ocular motility tests are seemingly uncomplicated, yet they must be accomplished in an exacting manner, observed with a critical eye, and interpreted with caution. The practitioner must have a detailed approach to distinguish normal from abnormal and to follow up abnormal findings with more complicated motility testing. The ocular motility findings have a direct bearing on the way subsequent portions of the eye examination are conducted. Their apparent simplicity belies a tremendous diagnostic value that is supported by a great deal of clinically relevant underlying knowledge.

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