

**Ear, Nose, and Throat Diseases** With Head and Neck Surgery

3rd edition

Founding authors:

Walter Becker, Hans Heinz Naumann, Carl Rudolf Pfaltz Authors of the 3rd edition:

Hans Behrbohm, MD

Professor and Director

Department of Otorhinolaryngology, Head and Neck Surgery, Facial Plastic Surgery Park Hospital Weissensee, Berlin

Medical Faculty, Humboldt University, Berlin Germany

In cooperation with the Institute of Medical Development and Further Education Berlin e.V.

Oliver Kaschke, MD

Professor and Director

Department of Otorhinolaryngology, Head and Neck Surgery, Facial Plastic Surgery St. Gertrauden Hospital, Berlin

Medical Faculty, Humboldt University, Berlin Germany

With a contribution by Thomas Verse, MD 780 illustrations

Foreword by Professor H. Stammberger

Thieme

Stuttgart New York

Tadeus Nawka, MD

Professor

Department of Audiology and Phoniatrics Charité Hospital

Berlin, Germany

Andrew Swift, ChM, FRCS, FRCSEd Consultant ENT Surgeon

University Hospital Aintree

Liverpool, UK

**IV**

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Illustrator: Katja Dalkowski MD, Buckenhof, Germany

Contributor:

Thomas Verse, MD

Professor and Director

Department of Otorhinolaryngology,

Head and Neck Surgery

Asklepios Hospital Harburg

Hamburg, Germany

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**Foreword**

After two decades of ever-increasing subspecializa tion, it is very encouraging and rewarding to see that otorhinolaryngology, head and neck surgery still can and should be seen as an entity. In the new edition of this marvelous textbook, Professor Hans Behrbohm and his colleagues, who have taken over authorship from Professors Walter Becker, Hans Heinz Naumann, and Carl Rudolf Pfaltz, pro vide all the anatomical, physiological, diagnostic, and therapeutic evidence for this, should anyone be in doubt.

This brilliantly illustrated textbook addresses ad vanced medical students and doctors alike, and provides a modern overview of the complexity of our fascinating specialty. As such, it may serve as both a learning and a teaching reference, summa rizing our present knowledge of the different as pects of all components contributing to otorhino laryngology, head and neck surgery.

It adds to the value and beauty of this book that not only the connections between and interdepen

dence of various subspecialties are displayed, but the interdisciplinarity with neighboring specialties is also addressed, e.g., with neurosurgery and neu rology for skull base and intracranial structures, with chest medicine specialties for tracheal and pulmonary disorders, or with gastroenterology for disorders of the upper digestive tract.

Thieme Publishers have to be congratulated for providing the plentiful and outstanding colored anatomical and schematic illustrations, in addition to the photographic images.This is a truly interna tional textbook that will be of great benefit to all of its readers!

H. Stammberger, MD, FRCSEd(Hon),

FRCSEng(Hon), FACS(Hon)

Professor and Head

Department of General Otorhinolaryngology, Head and Neck Surgery

Medical University of Graz

Graz, Austria

**VI**

**Preface**

The first edition of *Ear, Nose, and Throat Diseases* was written by Professors Walter Becker (1920 1990), Hans Heinz Naumann (1919 2001), and Carl R. Pfaltz (1922 2003) and published in 1988. Since then, and over several English and Ger man editions, this book has presented the essential knowledge of otorhinolaryngology, head and neck surgery in a concise and highly accessible format. In addition to this, it also provided advanced informa tion to facilitate a better understanding of the diag nostic and therapeutic issues and challenges of the specialty. The scope and didactic presentation of its content made this book attractive to medical stu dents, specialist trainees, residents, interested practitioners and specialists, both as a textbook and reference source. Because of its continuing suc cess, a new edition of *Ear, Nose, and Throat Diseases* was very much in demand, and we are grateful to Thieme Medical Publishers for the opportunity to prepare this new volume.

We have been sensitive to the fact that the book has been important on an international scale and we have strived to maintain this status, recognizing that the range of presenting conditions and their management will vary between various countries. As a surgical specialty, otorhinolaryngology head and neck surgery has shown a fast-paced, progres sive development with regard to diagnosis and treatment and the increased understanding of pathophysiological principles. It was therefore nec essary to restructure some chapters completely and to revise and update the remaining ones in order to bring the new edition up to the present standard of scientific and technical knowledge and practice in this specialty.

As in the previous editions, basic information is presented in normal type; supplementary and ad vanced information is given in a smaller type. The figures supplementing the text have received particular attention, as they are immensely impor tant and key to understanding the text. Numerous new drawings have been prepared; all of the exist ing drawings have been recreated in full color as well as revised and updated where necessary. Vis ual findings are often the key to diagnosis for the otorhinolaryngologist, so all of the previous clinical photographs have been replaced with new images,

and the number of images has been greatly in creased.

Our medical illustrator, Ms Katja Dalkowski MD, has made a substantial contribution to the visual ap pearance of the book, and we would like to express our gratitude for her invaluable assistance and commitment to the project. Our constant contact with Mr Stephan Konnry, our editor at Thieme Medical Publishers, his thorough editorial work and clearing up of many, many details and ques tions made an extremely valuable and beneficial contribution to the book.

The book project was generously supported by Dr h. c. mult. Sybill Storz, to whom we are extremely grateful. We are also grateful to the patients who were willing to allow photographs of their some times serious diseases to be published for the pur pose of medical education.

It is our hope that this new edition will continue to be a valuable practical guide to the vast range of otorhinolaryngology, head and neck surgery for its readers the students of medicine and dental med icine, as well as physicians and surgeons who are either in training or practicing as established spe cialists in various disciplines within the field.

*Hans Behrbohm*

*Oliver Kaschke*

*Tadeus Nawka*

*Andrew Swift*

We would like to express our warm thanks here to our former head of department and teacher, H.-J. Gerhardt now Professor Emeritus at the Depart ment of Otorhinolaryngology at the Charité Hospi tal in Berlin (where he held the chair from 1973 to 1994) for providing rare pictures of disease con ditions from his image archive.

The *Berlin Diagnostic Workshops* have provided an important stimulus for the work on this book. We have been involved in obtaining and assessing vis ual and auditory organ findings for more than 10 years and have had the opportunity to discuss these discoveries with many physicians.

*Hans Behrbohm*

*Oliver Kaschke*

*Tadeus Nawka*

**VII**

**Contents**

**1 Ear** .................................... 1

**Applied Anatomy and Physiology** ............ 1 Embryology . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 1 Basic Anatomy. . . . . . . . . . . . . . . . . . . . . . . . . . . . . 1

External Ear . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 2 Middle Ear and Pneumatic System. . . . . . . . . . . 3 Inner Ear, Peripheral Hearing, and Balance Organs . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 8

Central Connections of the Organ of Corti . . 11 Central Connections of the Organ of the Balance Mech anism. . . . . . . . . . . . . . . . . . . . . 11

Facial Nerve . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 13 Physiology and Pathophysiology of Hearing and Balance . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 14

Physiology of Hearing: Middle and Internal Ear. 14 Physiology of Hearing: Retrocochlear

Analysis of Acoustic Information . . . . . . . . . . . . 17 Pathophysiologic Basis of Hearing Disorders. . . 17 Ph ysiology of th e Balance System . . . . . . . . . . . 18 Pathophysiologic Basis of Functional Vestibular

Disorders . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 20 **Methods of Investigation** . . . . . . . . . . . . . . . . . . . 21 Inspection, Palpation, Otoscopy, Microscopy . . . . 21

Inspection of th e External Ear . . . . . . . . . . . . . . 21 Diagnostic Imaging. . . . . . . . . . . . . . . . . . . . . . . . . 24 Conventional Radiograph y . . . . . . . . . . . . . . . . . 24 Computed Tomograph y . . . . . . . . . . . . . . . . . . . 24 Angiograph y . . . . . . . . . . . . . . . . . . . . . . . . . . . . 24 Magnetic Resonance Imaging . . . . . . . . . . . . . . 26 Functional Assessment of the Eustachian Tube. . . 26 Qualitative Assessment of Tubal Function. . . . . 26 Hearing Investigations . . . . . . . . . . . . . . . . . . . . . . 27 Testing Hearing with out an Audiometer . . . . . . 27 Audiometry: Fundamental Physical and

Acoustic Concepts. . . . . . . . . . . . . . . . . . . . . . . . 27 Pure-tone Audiometry . . . . . . . . . . . . . . . . . . . . 27 Speech Audiometry . . . . . . . . . . . . . . . . . . . . . . 32 Objective Hearing Tests . . . . . . . . . . . . . . . . . . . 35 Hearing Tests in Infants and Young Children. . . 40

Vestibular Function Tests . . . . . . . . . . . . . . . . . . . . 41 Case History . . . . . . . . . . . . . . . . . . . . . . . . . . . . 41 Vestibulospinal Reflexes . . . . . . . . . . . . . . . . . . . 42 Spontaneous and Provoked Nystagmus . . . . . . 42 Experimental Tests of the Vestibular System. . . 45 Investigation of th e Facial Nerve . . . . . . . . . . . . . . 47

**Clinical Aspects of Diseases of the External Ear** 49 Congenital Anomalies. . . . . . . . . . . . . . . . . . . . . . . 49 Reconstructive Operations on th e Auricle . . . . . 49 Inflammations of th e External Ear . . . . . . . . . . . . . 50 Nonspecific Inflammation. . . . . . . . . . . . . . . . . . 50 Specific Forms of Inflammation of the

External Ear . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 51 Ch ronic Inflammation . . . . . . . . . . . . . . . . . . . . . 53 Otomycosis and Eczema. . . . . . . . . . . . . . . . . . . . . 53 Trauma. . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 54 Wax and Foreign Bodies . . . . . . . . . . . . . . . . . . . . . 55 Tumors. . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 56 Benign Tumors. . . . . . . . . . . . . . . . . . . . . . . . . . . 56 Precancerous and Malignant Tumors. . . . . . . . . 56

**Clinical Aspects of Diseases of the Middle and Internal Ear** . . . . . . . . . . . . . . . . . . . . . . . . . . . 58 Disorders of Ventilation and Drainage of the Middle Ear Spaces . . . . . . . . . . . . . . . . . . . . . . . . . . 58 Nonspecific Inflammation of the Middle Ear and Mastoid. . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 61 Specific Types of Inflammation of the Middle Ear and Mastoid. . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 64 Otogenic Infective Complications . . . . . . . . . . . . . 76 Specific Diseases of the Middle Ear and

Mastoid Process. . . . . . . . . . . . . . . . . . . . . . . . . . . . 81 Noninflammatory Diseases of the

Labyrinth ine Capsule. . . . . . . . . . . . . . . . . . . . . . . . 81 Trauma of th e Middle and Inner Ear . . . . . . . . . . . 84 Tumors of the Middle and Internal Ear,

Vestibulococh lear Nerve, and Facial Nerve . . . . . . 89 Congenital Anomalies of the Middle and

Internal Ear . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 94 **Clinical Aspects of Cochleovestibular Disorders** 95 Toxic Damage to the Hearing and

Balance Apparatus. . . . . . . . . . . . . . . . . . . . . . . . . . 95 Ototoxic Drugs . . . . . . . . . . . . . . . . . . . . . . . . . . 95 Ototoxic Occupational Toxins. . . . . . . . . . . . . . . 96

Inflammatory Lesions of the Hearing and Balance Apparatus. . . . . . . . . . . . . . . . . . . . . . . . . . 96 Immunologic Diseases of th e Inner Ear . . . . . . . . . 97 Trauma. . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 97 Vestibular Disorders . . . . . . . . . . . . . . . . . . . . . . . . 97 Hearing Disorders . . . . . . . . . . . . . . . . . . . . . . . . . . 101

**VIII Contents**

**Clinical Aspects of Central Hearing Disorders** . 104 **Rehabilitation of Hearing Disorders with Hearing Aids** . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 106 **Pediatric Hearing Disorders**

**(Pediatric Audiology)** . . . . . . . . . . . . . . . . . . . . . . 107 **Clinical Aspects of Disorders of the**

**Facial Nerve** . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 111 **Synopsis of Ear Symptoms** . . . . . . . . . . . . . . . . . 114

**2 Nose, Nasal Sinuses, and Face**. . . . . . . . . . . . . 116

**Applied Anatomy and Physiology** . . . . . . . . . . . 116 Basic Anatomy . . . . . . . . . . . . . . . . . . . . . . . . . . . . 116 External Nose . . . . . . . . . . . . . . . . . . . . . . . . . . . 116 Nasal Cavity . . . . . . . . . . . . . . . . . . . . . . . . . . . . 117 Paranasal Sinuses . . . . . . . . . . . . . . . . . . . . . . . . 120 Basic Ph ysiology and Path oph ysiology . . . . . . . . . 124 Th e Nose as an Olfactory Organ . . . . . . . . . . . . 124 Th e Nose as a Respiratory Organ . . . . . . . . . . . 126 Th e Nasal Mucosa as a Protective Organ . . . . . 127 Th e Nose as a Reflex Organ . . . . . . . . . . . . . . . . 127 Influence of th e Nose on Speech . . . . . . . . . . . . 128 Function of th e Nasal Sinuses . . . . . . . . . . . . . . 128 **Methods of Examining the Nose,**

**Paranasal Sinuses, and Face** . . . . . . . . . . . . . . . . 129 External Inspection and Palpation . . . . . . . . . . . . . 129 Anterior Rh inoscopy. . . . . . . . . . . . . . . . . . . . . . . . 129 Posterior Rh inoscopy . . . . . . . . . . . . . . . . . . . . . . . 131 Nasal Endoscopy. . . . . . . . . . . . . . . . . . . . . . . . . . . 132 Assessment of Nasal Patency. . . . . . . . . . . . . . . . . 135 Olfactometry . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 136 Diagnostic Imaging of th e Nose and Sinuses . . . . 136 Lavage of th e Sinuses. . . . . . . . . . . . . . . . . . . . . . . 138 Specific Diagnostic Meth ods . . . . . . . . . . . . . . . . . 140 **Dermatologic Principles for the**

**Otolaryngologist**. . . . . . . . . . . . . . . . . . . . . . . . . . 140 Skin Type . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 140 Types of Lesion . . . . . . . . . . . . . . . . . . . . . . . . . . 141 Basics of Topical Dermatologic Th erapy . . . . . . 141

**Clinical Aspects of Diseases of the Nose, Sinuses, and Face** . . . . . . . . . . . . . . . . . . . . . . . . . 144 Inflammatory Diseases of the Nose and

Paranasal Sinuses . . . . . . . . . . . . . . . . . . . . . . . . . . 145 Inflammations Confined Mainly to the

External Nose . . . . . . . . . . . . . . . . . . . . . . . . . . . 145 Acute and Chronic Inflammations Localized Mainly in th e Nasal Cavity . . . . . . . . . . . . . . . . . 149 Local Conservative Treatment in the Upper Respiratory and Digestive Tracts . . . . . . . . . . . . 158 Acute and Ch ronic Rh inosinusitis . . . . . . . . . . . 159 Rh inogenous Headach e . . . . . . . . . . . . . . . . . . . 170

Facial Neuralgias . . . . . . . . . . . . . . . . . . . . . . . . . 171 Principles of Surgery of the Paranasal Sinuses . 171 Rh inosinusitis in Ch ildren . . . . . . . . . . . . . . . . . . 180 Fungal Diseases of th e Paranasal Sinuses . . . . . 182 Pathophysiologic Relationship between the

Sinuses and th e Rest of th e Body . . . . . . . . . . . 184 Mucoceles and Cysts . . . . . . . . . . . . . . . . . . . . . 184 Complications of Sinus Infections . . . . . . . . . . . 186

Epistaxis . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 191 Diseases of th e Septum . . . . . . . . . . . . . . . . . . . . . 197 Trauma to the Nose, Paranasal Sinuses, and Facial Skeleton . . . . . . . . . . . . . . . . . . . . . . . . . . . . 201 Trauma of th e Middle Th ird of th e Face

and th e Sinuses. . . . . . . . . . . . . . . . . . . . . . . . . . 203 Congenital Anomalies and Deformities

of th e Nose . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 210 Congenital Anomalies of th e Nose . . . . . . . . . . 210 Disorders of Sh ape of th e External Nose. . . . . . 212 Basic Plastic Reconstruction Procedures

in th e Head and Neck. . . . . . . . . . . . . . . . . . . . . 215 Tumors of th e Nose and Sinuses . . . . . . . . . . . . . . 218 Benign Tumors . . . . . . . . . . . . . . . . . . . . . . . . . . 218 Malignant Tumors of th e External Nose . . . . . . 222

Malignant Tumors of the Nasal Cavity and Nasal Sinuses . . . . . . . . . . . . . . . . . . . . . . . . . . . 225 Principles of Management of Malignant

Tumors of th e Nose and Paranasal Sinuses. . . . 226

**3 Mouth and Pharynx** . . . . . . . . . . . . . . . . . . . . . 228

**Applied Anatomy and Physiology** . . . . . . . . . . . 228 Basic Anatomy . . . . . . . . . . . . . . . . . . . . . . . . . . . . 228 Oral Cavity . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 228 Nasopharynx, Oropharynx, and Hypopharynx . 231 Lymph oepith elial System of th e Ph arynx . . . . . 233 Ph ysiologic and Path oph ysiologic Principles. . . . . 235 Eating, Preparation of Food, and Swallowing . . 235 Taste . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 237 Function of th e Tonsils. . . . . . . . . . . . . . . . . . . . 237 Formation of Sound and Speech . . . . . . . . . . . . 239 **Methods of Investigation** . . . . . . . . . . . . . . . . . . 239

Inspection, Palpation, and Examination with th e Mirror . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 239 Endoscopy. . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 241 Imaging Studies . . . . . . . . . . . . . . . . . . . . . . . . . . . 241 Examination of th e Saliva. . . . . . . . . . . . . . . . . . . . 242 Gustometry. . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 242 Specific Diagnostic Procedures . . . . . . . . . . . . . . . 243 **Clinical Aspects of Diseases of the Mouth and Pharynx** . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 243 Hyperplasia of th e Lymph oepith elial Organs . . . . 243

Dysph agia . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 245 Inflammatory Diseases . . . . . . . . . . . . . . . . . . . . . . 247 Labial and Oral Mucosa. . . . . . . . . . . . . . . . . . . . 247 Tongue. . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 256 Pharyngeal Lymphatic Ring (Waldeyer Ring). . . 260 Oth er Ph aryngeal Inflammations . . . . . . . . . . . . 273 Oth er Ph aryngeal Diseases . . . . . . . . . . . . . . . . . 276 Basic Conservative Treatment of th e Mucosa . . . . 277 Trauma in th e Mouth and Ph arynx. . . . . . . . . . . . . 277 Neurogenic Disorders . . . . . . . . . . . . . . . . . . . . . . . 279 Hypopharyngeal Diverticulum

(Zenker Diverticulum). . . . . . . . . . . . . . . . . . . . . . . 279 Anomalies of th e Mouth and Ph arynx . . . . . . . . . . 280 Tumors of th e Mouth and Ph arynx . . . . . . . . . . . . 282 Benign Tumors of the Oral Cavity,

Including th e Tongue and Oroph arynx . . . . . . . 282 Malignant Tumors of the Oral Cavity,

Including Lip, Tongue, and Oroph arynx. . . . . . . 282 Benign Tumors of th e Nasoph arynx. . . . . . . . . . 288 Malignant Tumors of th e Nasoph arynx . . . . . . . 289 Tumors of th e Hypoph arynx. . . . . . . . . . . . . . . . 290 Obstructive Sleep-related Breathing Disorders . . . 290

**4 Larynx and Hypopharynx**. . . . . . . . . . . . . . . . . 293

**Larynx Applied Anatomy and Physiology** . . . . 293 Basic Anatomy and Ph ysiology. . . . . . . . . . . . . . . . 293 Embryology . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 293 Anatomy . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 293 Ph ysiology . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 296 **Methods of Examination** . . . . . . . . . . . . . . . . . . . 297 Inspection . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 297 Palpation . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 297 Laryngoscopy . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 298 Indirect Laryngoscopy. . . . . . . . . . . . . . . . . . . . . 298 Flexible Nasendoscopy . . . . . . . . . . . . . . . . . . . . 298 Rigid Endoscopy of th e Larynx . . . . . . . . . . . . . . 299 Microlaryngoscopy . . . . . . . . . . . . . . . . . . . . . . . 299 Diagnostic Imaging. . . . . . . . . . . . . . . . . . . . . . . . . 300 Stroboscopy. . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 300 Oth er Special Tech niques . . . . . . . . . . . . . . . . . . . . 301 **Clinical Aspects** . . . . . . . . . . . . . . . . . . . . . . . . . . . 301 Congenital Anomalies. . . . . . . . . . . . . . . . . . . . . . . 301 Organic Functional Disorders . . . . . . . . . . . . . . . . . 302 Trauma. . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 308 Inflammation. . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 313 Tumors. . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 318 Benign Tumors. . . . . . . . . . . . . . . . . . . . . . . . . . . 318 Malignant Tumors . . . . . . . . . . . . . . . . . . . . . . . . 321 **Hypopharynx** . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 328 Hypoph aryngeal Carcinoma. . . . . . . . . . . . . . . . 328

*Contents* **IX**

**5 Voice, Speech, and Language** . . . . . . . . . . . . . 330

**Voice** . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 330 Voice Production. . . . . . . . . . . . . . . . . . . . . . . . . . . 330 Glottal Sound Generation. . . . . . . . . . . . . . . . . . 330 Body-cover Model . . . . . . . . . . . . . . . . . . . . . . . . 330 Source-filter Th eory. . . . . . . . . . . . . . . . . . . . . . . 331 Voice Diagnosis . . . . . . . . . . . . . . . . . . . . . . . . . . . . 332 Functional Voice Disorders . . . . . . . . . . . . . . . . . . . 334 **Speech**. . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 336 **Language** . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 338

**6 Tracheobronchial Tree** . . . . . . . . . . . . . . . . . . . 344

**Applied Anatomy and Physiology** . . . . . . . . . . . . 344 Basic Anatomy. . . . . . . . . . . . . . . . . . . . . . . . . . . . . 344 Basic Ph ysiology . . . . . . . . . . . . . . . . . . . . . . . . . . . 344 **Methods of Investigation** . . . . . . . . . . . . . . . . . . . 345 Trach eobronch oscopy. . . . . . . . . . . . . . . . . . . . . . . 345 **Clinical Aspects** . . . . . . . . . . . . . . . . . . . . . . . . . . . 347 Stenoses. . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 347 Tracheotomy, Cricothyrotomy, and Intubation . . . 351 Foreign Bodies and Trauma . . . . . . . . . . . . . . . . . . 357 Infections. . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 358 Congenital and Hereditary Anomalies . . . . . . . . . . 358 Tumors. . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 359

Benign Trach eal Tumors . . . . . . . . . . . . . . . . . . . 359 Malignant Trach eal Tumors . . . . . . . . . . . . . . . . 359

**7 Esophagus** . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 361

**Applied Anatomy**. . . . . . . . . . . . . . . . . . . . . . . . . . 361 **Physiology and Pathophysiology** . . . . . . . . . . . . 362 **Investigation Methods** . . . . . . . . . . . . . . . . . . . . . 362 Clinical Examination . . . . . . . . . . . . . . . . . . . . . . . . 362 Diagnostic Imaging. . . . . . . . . . . . . . . . . . . . . . . . . 362 Esoph agoscopy . . . . . . . . . . . . . . . . . . . . . . . . . . . . 363 Manometry . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 364 **Clinical Aspects** . . . . . . . . . . . . . . . . . . . . . . . . . . . 365 Trauma. . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 365 Esoph ageal Diverticulum . . . . . . . . . . . . . . . . . . . . 368 Inflammations . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 370 Motility Disorders . . . . . . . . . . . . . . . . . . . . . . . . . . 370 Esophageal Involvement in Diseases of

Neigh boring Organs . . . . . . . . . . . . . . . . . . . . . . . . 372 Congenital Anomalies and Fistulas . . . . . . . . . . . . 372 Tumors of th e Esoph agus. . . . . . . . . . . . . . . . . . . . 373

Benign Tumors. . . . . . . . . . . . . . . . . . . . . . . . . . . 373 Malignant Tumors . . . . . . . . . . . . . . . . . . . . . . . . 373

**Contents**

**8 Neck (Including the Thyroid Gland)** . . . . . . . . 375

**Applied Anatomy and Physiology** . . . . . . . . . . . 375 Basic Anatomy and Ph ysiology . . . . . . . . . . . . . . . 375 Regions . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 375 Fascia. . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 375 Spaces . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 377 Blood Vessels . . . . . . . . . . . . . . . . . . . . . . . . . . . 378 Cervical Lymph atic System . . . . . . . . . . . . . . . . 380 Nerves . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 382 Basic Ph ysiology . . . . . . . . . . . . . . . . . . . . . . . . . 384 **Methods of Investigation** . . . . . . . . . . . . . . . . . . 385 Specific History. . . . . . . . . . . . . . . . . . . . . . . . . . . . 385 Inspection of th e Neck Region . . . . . . . . . . . . . . . 385 Palpation . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 385 Diagnostic Imaging . . . . . . . . . . . . . . . . . . . . . . . . 386 Main Tech niques. . . . . . . . . . . . . . . . . . . . . . . . . 386 Special Tech niques . . . . . . . . . . . . . . . . . . . . . . . 387 Biopsy . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 387 **Clinical Aspects** . . . . . . . . . . . . . . . . . . . . . . . . . . . 387 Inflammation of th e Cervical Soft Tissues . . . . . . 387 Inflammatory Cervical Lymph adenopath y . . . . . . 388 Cervical Spine Syndrome . . . . . . . . . . . . . . . . . . . . 391 Trauma . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 392 Congenital Anomalies . . . . . . . . . . . . . . . . . . . . . . 393 Th yroglossal Duct Cysts and Fistulas. . . . . . . . . 393 Branch ial Cysts and Fistulas . . . . . . . . . . . . . . . . 393 Musculoskeletal Defects. . . . . . . . . . . . . . . . . . . 395 Vascular Malformations . . . . . . . . . . . . . . . . . . . 396 Tumors . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 398 Benign Tumors . . . . . . . . . . . . . . . . . . . . . . . . . . 398 Malignant Tumors of the Cervical

Lymph Nodes . . . . . . . . . . . . . . . . . . . . . . . . . . . 398 CUP Syndrome . . . . . . . . . . . . . . . . . . . . . . . . . . 402 Principles of Surgery. . . . . . . . . . . . . . . . . . . . . . . . 404 Prescalene Node Biopsy . . . . . . . . . . . . . . . . . . . 404 Mediastinoscopy. . . . . . . . . . . . . . . . . . . . . . . . . 404 Neck Dissection . . . . . . . . . . . . . . . . . . . . . . . . . 405 Th yroid Gland and Otorh inolaryngology. . . . . . . . 407 Topograph ic Anatomy . . . . . . . . . . . . . . . . . . . . 407 Diagnostic Procedures in Thyroid Disorders . . . 408 Specific Conditions. . . . . . . . . . . . . . . . . . . . . . . 408

**9 Salivary Glands** . . . . . . . . . . . . . . . . . . . . . . . . . 413

**Embryology, Structure, and Congenital Anomalies** . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 413

**Anatomy and Physiology of the Major**

**and Minor Salivary Glands**. . . . . . . . . . . . . . . . . . 414 Parotid Gland . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 414 Submandibular Gland. . . . . . . . . . . . . . . . . . . . . . . 415 Sublingual Gland. . . . . . . . . . . . . . . . . . . . . . . . . . . 415 Minor Salivary Glands. . . . . . . . . . . . . . . . . . . . . . . 415 **Formation and Function of Saliva**. . . . . . . . . . . . 415 **Methods of Investigation** . . . . . . . . . . . . . . . . . . 416 Diagnostic Imaging . . . . . . . . . . . . . . . . . . . . . . . . 417

Sonograph y. . . . . . . . . . . . . . . . . . . . . . . . . . . . . 417 Computed Tomograph y, Spiral CT . . . . . . . . . . 417 Magnetic Resonance Imaging . . . . . . . . . . . . . . 417 Sialograph y . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 417 Positron-Emission Tomograph y . . . . . . . . . . . . . 418 Plain Radiograph s . . . . . . . . . . . . . . . . . . . . . . . . 418

Function Studies . . . . . . . . . . . . . . . . . . . . . . . . . . . 418 Meth ods . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 418 Biopsy . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 419 **Clinical Aspects** . . . . . . . . . . . . . . . . . . . . . . . . . . . 420 Inflammatory Diseases. . . . . . . . . . . . . . . . . . . . . . 420 Acute Bacterial Infections . . . . . . . . . . . . . . . . . 420 Viral Infections . . . . . . . . . . . . . . . . . . . . . . . . . . 421 Ch ronic Inflammation . . . . . . . . . . . . . . . . . . . . 422 Sialolith iasis. . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 425 Cysts . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 426 Sialadenosis. . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 427 Trauma . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 427 Injury to the Nerves or Ducts and

Salivary Fistulas. . . . . . . . . . . . . . . . . . . . . . . . . . 427 Salivary Tumors . . . . . . . . . . . . . . . . . . . . . . . . . . . 428 Benign Tumors . . . . . . . . . . . . . . . . . . . . . . . . . . 428 Malignant Tumors. . . . . . . . . . . . . . . . . . . . . . . . 432 Basic Principles in the Treatment of

Salivary Tumors . . . . . . . . . . . . . . . . . . . . . . . . . 435

**Appendix** . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 438

**Emergencies in ENT and First Aid Procedures**. . 439 **Essential Information for Infection Control** . . . 440 Introduction . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 440 Infection Control Response in th e UK . . . . . . . . . . 440 *C. difficile* Infection . . . . . . . . . . . . . . . . . . . . . . . . . 441 MRSA Infection . . . . . . . . . . . . . . . . . . . . . . . . . . . . 442 **References and Further Reading**. . . . . . . . . . . . . 443 **Subject Index**. . . . . . . . . . . . . . . . . . . . . . . . . . . . . 445

**1 Ear**

**Applied Anatomy and Physiology Embryology**

**Inner ear.** The sensory organs for hearing and bal ance develop from ectoderm. The *membranous lab yrinth* develops from the ectodermal otic placode. *Embryonic mesenchymal tissue* surrounding the *membranous labyrinth* is converted into cartilage and also, by a process of vacuolization, into a fine reticular network that forms the inner layer of the *perilymphatic space.* The outer layer of the cartilage forms the *labyrinthine capsule.*

**Middle ear.** The eustachian tube and the mucosa of the middle ear arise from a diverticulum of the first pharyngeal pouch (endoderm).

The malleus and incus develop from Meckel car tilage, which emerges from the first branchial arch and is supplied by the trigeminal nerve. The stapes develops from the second branchial arch and is supplied by the facial nerve.

Myxomatous embryonic connective tissue lies between the ectodermal and endodermal in growths and makes a preformed middle ear cavity. If this myxomatous tissue does not involute prop erly after birth, the epitympanic recess remains as a narrow cleft. This is easily occluded by inflamma tion and creates a predisposition for chronic ear disease to develop.

**External ear.** The external meatus and the tym panic membrane develop from an ectodermal di verticulum between the first and second branchial arches. Developmental disorders may therefore cause deformities of both the external and middle ears. Bilateral lesions causing severe conductive deafness or a psychologically unacceptable deform ity should be corrected, for both esthetic and func tional reasons (see pp. 49 and 94) (**Figs. 1.1, 1.2**).

**Basic Anatomy**

The hearing and balance systems consist of the *peripheral receptor apparatus* (i. e., the ear in the strict sense), *neurological pathways,* and *centers in the central nervous system.* Two main subdivisions can therefore be distinguished:

*Peripheral part:*

The external, middle, and inner ear.

Vestibulocochlear nerve with its two parts, the cochlear and the vestibular divisions.

*Central part:*

Central auditory pathways.

Subcortical and cortical auditory centers. Central balance mechanism.

| 10  345 6  2  1  **a**  7 8 9  3  3  3  4  4  2  4  2  2  5  ~~5~~  6~~1~~  1  5  ~~6~~ 1  6  **b** |
| --- |

**Fig. 1.1a, b** Development of the external ear. An 11-mm embryo, lateral view.

Development of the outer ear from six hillocks arising from the first and second branchial arches.

, Tragus; , crus helicis; , helix; , crus anthelicis; , antihelix; , antitragus; , first branchial arch; , branchial cleft; , second branchial arch; **10**, auricular plate.

**1 Ear**

| 268  1  4  2  5  7  3  **a b** |
| --- |

**Fig. 1.2a, b** Developmental stages of the external auditory canal, middle ear, and labyrinth. The epithelial auditory canal pouch with the tympanic plate opens through epithelial necrolysis (apoptosis) in the seventh month. The mesen chyme of the stapes develops from the second visceral arch;the remaining structures of the middle ear develop from the first visceral arch.

**Fig. 1.3** Topography of 

the external ear structures.

, Helix; , antihelix ( : in

ferior crus, : superior

crus); , scaphoid fossa;

, cymba conchae;

, cavum conchae;

, tragus; , antitragus;

, triangular fossa;

earlobe.

The *anatomic boundary* between the *peripheral* and *central* parts is the point of entry of the eighth cranial nerve into the brain stem (the cerebellopon tine angle), at which point the peripheral part of the vestibulocochlear nerve passes into the central part, interspersed with glial cells. In functional terms, however, the peripheral neurons end in the primary centers.

**External Ear**

The *auricle* consists of a framework of elastic carti lage covered by skin (**Fig. 1.3**), located between the temporomandibular joint anteriorly and the mas toid process posteriorly. The skin adheres tightly to

Approx. 8th week. , Otic vesicle; , primary auditory canal; , tubotympanic recess; , mesenchymal condensa tion; , acousticofacial ganglion.

Approx. 7th month. , Primary auditory canal; , primor dium of the auditory ossicles; tympanic cavity; , primordium for the labyrinth.

the perichondrium on the anterior surface, but is more loosely attached posteriorly. For this reason, contusions of the anterior surface often lead to detachment of the skin perichondrial layer and to the formation of a hematoma (see p. 55).

The *external meatus* is 3 cm long, consisting of an outer cartilaginous part and an inner bony part. The cartilaginous meatus is curved and lies at an angle to the bony part. The tympanic membrane and the middle ear lying beyond it are thus pro tected from direct trauma.

Note: For an otoscope to be introduced accurately, the curved cartilaginous mobile part of the external auditory meatus has to be drawn upward and posteriorly to bring it into the same axis as the bony part.

The cartilaginous part is attached firmly to the rim of the *bony meatus* by connective tissue. The bony canal is covered by a thin layer of skin that adheres to the periosteum. It contains no accessory struc tures, in contrast to the cartilaginous part of the meatus, which has numerous hair follicles and ce ruminous glands that form wax(epidermis scale, sebaceous matter, pigment) (see p. 55).

The external meatus narrows medially. *Foreign bodies* may therefore become impacted at the junc tion of the cartilaginous and bony meatus. The meatal cartilage does not form a closed tube, but

*Applied Anatomy and Physiology*

| Inner ear External ear  7 6 3 2 1  8  9  10  11 12 5 4  Middle ear |
| --- |

**Fig. 1.4** Overview of the three sections of the ear. *External ear:* , auricle; , external ear canal;

, tympanic membrane. *Middle ear:* , tympanic cavity; , eu stachian tube. *Inner ear:*

labyrinth with internal ear canal and vestibulocochlear nerve; , internal carotid artery;

, cartilage of eustachian tube; **10**, levator veli palatini muscle; **11**, tensor veli palati muscle; **12**, tensor tympani muscle (Toynbee muscle).

rather a channel closed superiorly by fibrous tissue. The cartilage contains several fissures (Santorini fissures), which provide a pathway for the spread of severe bacterial infection to the parotid space, the infratemporal fossa, and the base of the skull.

The auricle and the cartilaginous meatus have very rich *lymphatic drainage* to an extensive re gional lymphatic network consisting of parotid, ret roauricular, infra-auricular, and superior deep cer vical nodes. Infections of the external meatus with regional lymphadenitis can thus cause extensive swelling in these areas.

The *sensory innervation* is supplied by the tri geminal, great auricular, and vagus nerves and the sensory fibers of the facial nerve. Irritation of the posterior meatal wall stimulates the vagus and in duces the cough reflex. Hypoesthesia of the poste rosuperior meatal wall occurs with facial nerve impingement from a vestibular schwannoma (see the discussion of Hitselberger sign, p. 13 and **Table 1.13**, p. 94).

*Relations* **Fig. 1.4**): The cartilaginous meatus abuts anteriorly onto on the parotid gland, allowing the spread of infection or malignant tumors.

The posterosuperior wall of the bony meatus forms part of the *lateral attic wall* (the partition between the external auditory meatus and the at

tic), the mastoid antrum, and the adjacent pneu matic system of the mastoid process. A middle ear infection can thus break through into the external auditory meatus, causing swelling of the postero superior wall or a fistula in acute mastoiditis. De struction of the lateral attic wall by *cholesteatoma* may also lead to an open communication between the external auditory meatus and the attic or mas toid antrum. The anterior wall of the bony meatus forms part of the temporomandibular joint. There is therefore a risk of *fracture* resulting from a blow to the chin.

**Middle Ear and Pneumatic System**

The *middle ear cavity* consists of an extensive *pneu matic system* aerated by the eustachian tube. It has the following components:

Eustachian tube.

Tympanic cavity.

Mastoid antrum.

The *eustachian tube* consists of a mobile, cartilagi nous portion (two-thirds) suspended from the skull base, and a bony portion (one-third). The bony portion, together with the tensor tympani muscle, forms the musculotubal canal in the temporal bone.

**1 Ear**

| 1  2  5  7  8  3  9  10  4  6 |
| --- |

**Fig. 1.5** Anatomy of the middle ear cavity. , Epitym panum; , mesotympanum; , hypotympanum; , mastoid antrum; , internal jugular vein. The lower part of the attic

) is markedly narrowed by the facial nerve ( ) and the horizontal semicircular canal. , External meatus; , tym panic membrane; **10,** cochlea.

This canal lies adjacent to the internal carotid artery. The funnel-shaped pharyngeal ostium of the cartilaginous part (the torus tubarius) lies in the nasopharynx. The bony end opens into the middle ear.

The junction between the two parts of the tube is very narrow. This *isthmus* is the site of predilection for inflammatory stenosis of the tube. The tube serves to equalize the pressure between the middle ear and the nasopharynx, and thus to equalize the pressure on each side of the tympanic membrane (see pp. 6, 37). An increase in pressure in the tym panic cavity is usually compensated for passively via the eustachian tube to the nasopharynx, whereas a decrease in pressure usually requires active ventilation from the nasopharynxalong the tube to the middle ear cavity. The tube opens and closes in response to movements of the neighbor ing muscles and by differences of air pressure be tween the nasopharynxand the middle ear cavity that tend to equalize spontaneously. The principal closing mechanism is elastic recoil of the cartilage

of the tube and the valvular action of the pharyn geal ostium of the tube. The tube opens by contrac tion of the tensor palati and levator palati muscles. The mechanism is partially under the control of voluntary muscle, but the reflexmovements on yawning and swallowing and the muscle tone are under autonomic control. Tension opposing the opening muscles is provided by the elastic recoil of the tubal cartilage and the pressure of the peri tubal tissues i. e., the pterygoid muscles, Ost mann s fatty bodies, the venous and lymphatic plexus of the tubal mucosa, and the pterygoid ve nous plexus.

The *middle ear cavity* is an air-containing space lying between the external ear and the inner ear. It is divided into three parts (**Fig. 1.5**):

Epitympanic recess or attic.

Mesotympanum.

Hypotympanic recess.

There are two narrow zones within the middle ear cleft. Firstly, there is an anatomic constriction be tween the epitympanum and mesotympanum that can lead to retention of secretions in inflammation and to deficient aeration of the attic. This is due to the considerable narrowing of this area caused by the head of the malleus, the body of the incus, numerous ligaments, nerves (the chorda tympani), and mucosal folds and pockets. This is one of the causes of chronic inflammation of the epitympa num (chronic epitympanitis), which is one of the causative factors for epitympanic cholesteatoma (see p. 68). A second narrow zone lies at the junc tion of the attic and the mastoid antrum (the aditus ad antrum). This may be blocked by granulation tissue in chronic inflammation, leading to deficient aeration or drainage of the mastoid cell system.

The hypotympanum is closely related to the bulb of the internal jugular vein.

The *tympanic membrane*: The lateral wall of the middle ear cavity is formed by the tympanic mem brane. The tympanic membrane consists of the pars tensa and the pars flaccida. The *pars tensa* forms the stiff vibrating surface of the membrane and is at tached to a fibrous ring (the *anulus fibrosus*), lying in the tympanic sulcus of the tympanic part of the temporal bone. The *pars flaccida* is the superior part of the membrane in the area of the tympanic notch (Rivinus notch) where the anulus fibrosus ends **Fig. 1.6a,b**

*Applied Anatomy and Physiology*

| 5  3  4  IV I  III  II  2  6 1  **a b** |
| --- |

**Fig. 1.6a, b a** The macro scopic appearance of the right tympanic membrane. , Light reflex; , pars tensa; , malleus head; , pars flaccida; , incus;

, umbo; **7,** anulus fibrosus. The visible part of the surface of the tympanic membrane is divided into four quadrants, in order of investigation: , anterosuperior; **II**, anteroinferior; **III**, posteroin ferior;and **IV**, posterosuperior.

The oto-endoscopic appear ance of a normal, transparent tympanic membrane. The tym panic ring, the handle of the malleus, and the short process of the malleus are visible. The light reflex is visible in the usual position starting from the umbo across the anterior infe rior quadrant.

The microscopic cross-sectional appearance of the tympanic membrane is shown in **Fig. 1.7**. The epithelial or cuticular layer (the stratum corneum) is similar in structure to the skin of the external auditory meatus. Close to the tympanic annulus is the marginal zone of the tympanic membrane. This section shows extremely active proliferation due to papillary ingrowths into the stratum germinati vum. This is another important factor in the genesis of cholesteatoma (see pp. 68 76).

The keratinizing squamous epithelium regener ates through migration of the epidermis from the center of the tympanic membrane to the periphe ry in contrast to superficial desquamation, as oc curs in normal skin. Migration of the outer epider mal layer forms an important part of the self cleansing mechanism of the external meatus; this can be observed clinically in the movement of a blood clot from the tympanic membrane to the external meatus.

The *lamina propria* has an external radial layer of fibers and an internal circular layer: this is evident during myringotomy. The anulus fibrosus forms a thickening of the edge of the tympanic membrane and is formed by both layers of fiber. A lamina propria can also be seen in the pars flaccida, but it lacks the characteristic radial and circular structure described above, which provides the normal pars tensa with the necessary functional tension.

The *middle ear*, or *tympanic cavity*, is empty ex - cept for air. Only the epitympanic recess contains

solid structures the ossicular chain and the chorda tympani. The ossicular chain consists of three sep arate bones connecting the lateral and medial wall of the middle ear. The medial wall of the middle ear also forms the lateral wall of the labyrinthine cap sule. The malleus is the most lateral of the ossicles. Its inferior portion, or handle, is incorporated into the eardrum, while the superior portion, or head, is located in the anterior portion of the attic. The incus is connected to the head of the malleus by a genuine articulation surrounded by a joint capsule. The long process of the incus ends in the lenticular process, which bends medially to articulate with the head of the stapes. The lenticular process is covered by cartilage to form the incudostapedial joint (**Figs. 1.8, 1.9**).

The *mucosa* that lines the middle ear space consists of stratified cuboidal epithelium, which changes to pseudo stratified ciliated epithelium around the mouth of the eusta chian tube. A few goblet cells and submucosal glands are normally present. The submucosa is very thin, so that the mucosa lies directly on the periosteum, forming a tightly bound unit called the *mucoperiosteum.* In pathologic condi tions such as tubal occlusion or chronic otitis media, the structure of the mucosa changes considerably to show hy perplasia of the glands, proliferation of the goblet cells, edema of the submucosa, vascular buds, and transformation of the flattened cuboidal epithelium to columnar epithe lium.

The middle ear mucosa forms several pouches and folds *(Prussak space, Tröltsch pouch),* which narrow the junction

**1 Ear**

| 4  3  2  1 |
| --- |

**Fig. 1.7** The microscopic appearance of a sagittal section through the posterosuperior quadrant of the tympanic membrane. , Epidermis layer, similar to the meatal skin bordering the tympanic membrane; , middle ear mucosa;

, anulus fibrosus; , bony sulcus of the fibrocartilaginous ring.

between the attic and the rest of the middle ear and be tween the attic and the antrum. The epitympanic recess may remain as a narrow cleft with development, and if chronic hyperplastic inflammation follows an infection, the mesen chyme can completely obliterate the epitympanum. Ven tilation and drainage of the attic is then impeded by thick ened masses of inflammatory tissue, despite normal tubal function. Deficient aeration and drainage of this small space

| 876  1  9  2345 |
| --- |

**Fig. 1.8** Medial view of the middle ear, with the ossicular chain and facial nerve. , Tensor tympani muscle. The pars tensa is anchored by the anulus fibrosus ( ) in the bony niche of the tympanic ring. , Stapes footplate. The handle and short process of the malleus lie lateral to the chorda tympani

), as part of the facial nerve ( ). The long process of the incus forms a joint ( ) at its lenticular process with the head of the stapes. The body of the incus ( ) forms the joint surface for the head of the malleus ( ). The malleus and incus vibrate as one body in the middle part of the frequency range. The middle ear cavity is aerated via the eustachian tube ( ).

favors the development of chronic *epitympanitis* and plays a considerable role in the pathogenesis of *chronic otitis media* (see p. 58 61), especially attic cholesteatoma.

The *arterial blood supply* originates from the basilar artery (the labyrinthine artery), the maxillary artery (the middle meningeal and tympanic arteries), and the stylomastoid artery. Venous drainage is via the middle meningeal veins, the venous plexus of the internal carotid artery and pharynx, and venous connections into the bulb of the internal jugular vein.

The *nerve supply* of the mucosa is provided from two sources: the tympanic branch of the glossopharyngeal nerve (cranial nerve IX) and the auriculotemporal branch of the trigeminal nerve (cranial nerve V).

Note: The shared sensory supply of the ear and upper respiratory tract explains why pain is referred to the ear in diseases of the teeth and the jaws, as well as of the larynx and pharynx.

**Pneumatic System of the Temporal Bones** The air-containing cells of the mastoid process are continuous with the air in the middle ear. These multiple interconnecting spaces arise from the mastoid antrum, and the extent to which they are pneumatized is extremely variable. On the one hand, *pneumatization* may be well developed, ex tending to the temporal and occipital bones and the origin of the zygomatic arch. Acute infections of the mastoid may cause inflammatory swellings in these regions. At the other extreme, in a poorly pneuma tized mastoid, the mastoid process may consist ex - clusively of compact bone, with the pneumatized cells lying in the immediate vicinity of the antrum.

The mastoid process begins to develop after birth as a small tuberosity, which is pneumatized synchronously with the growth of the mastoid an trum. In the first year of life it consists of cancellous bone, so that true mastoiditis cannot occur. Be tween the second and fifth years of life, as pneuma tization proceeds, it consists of mixed cancellous and pneumatic bone. Pneumatization is complete between the sixth and twelfth years of life (**Figs. 1.10, 1.11**).

**Principle of pneumatization (the concept of biolog ical mucosal competence).** Bone is destroyed by an enzymatic lacunar osteoclastic process. The resulting bony spaces are lined by continuous ingrowth of mucoperiosteum from the antrum. A system of hollow cavities results, con sisting of numerous spaces lined by mucosa and communi cating with each other.

*Normal tubal function* is a prerequisite for biologically active, healthy middle ear mucosa, and thus for the normal process of pneumatization. The process of pneumatization can be related to the biological competence of the middle ear mucosa. The mucosa may be described as *biologically normal* or as *inferior*, depending on the degree of pneumati zation. *Good pneumatization* indicates biologically compe tent middle ear mucosa, whereas *restricted pneumatization* indicates biological incompetence of the middle ear mu cosa. Biologically incompetent middle ear mucosa may be due to two possible mechanisms a defective enzyme sys tem that is impairing normal pneumatization, and/or a de ficient local immune system in the respiratory mucosa and middle ear mucoperiosteum that predisposes to chronic or recurrent otitis media.

Note: Characteristically, pneumatization of the tempo ral bone is absent or restricted in chronic otitis media.

*Applied Anatomy and Physiology*

| 3 1  4 2  1 |
| --- |

**Fig. 1.9** The axis of ossicular chain movement. The mal leoincudal joint can turn at a 90 angle according to the position of the footplate ( ). The footplate itself can move from anterior to posterior ( ) and in a lateral direction ( ). The incudostapedial joint ( ) moves only in a slight lateral bend.

| 7 6 3 5  1 8 2 4 |
| --- |

**Fig. 1.10** The pneumatic system of the temporal bone. , Transverse sinus; , mastoid process with tip cells; , mas toid antrum; , eustachian tube; , zygomatic cells; , cells of the squamous part of the temporal bone; , sinodural angle; , retrosinus cells.

**1 Ear**

| 7 6 5 4  8  1 2 3 |
| --- |

**Fig. 1.11** Topographic relationships in the middle ear cav ity. , Facial nerve inflammation and trauma often affect the mastoid segment; , the bulb of the internal jugular vein, which is the site of predilection for extension of a glomus tumor into the middle ear cavity; , the internal carotid artery in petrositis, the inflammation can extend into the venous plexus around the carotid artery to create a caver nous sinus thrombosis; , cavernous sinus; , apical cells

purulent infection of the cells in petrositis (see p. 80) causes Gradenigo syndrome; , the tensor tympani muscle; , the tegmen tympani, which is the site of predilection for mas toiditis to penetrate into the middle cranial fossa; , the pneumatic system of the mastoid process purulent infec tion of the cells causes subperiosteal abscess and sigmoid sinus thrombosis.

The better pneumatized the temporal bone is, the easier it is for infection to break through the thin cortical bone. When there is poor pneumatization (known as a *dangerous mastoid process*), the inflam matory process may be concealed in the depths and lead to unexpected complications.

**Inner Ear, Peripheral Hearing, and Balance Organs**

The inner ear, or labyrinth, is embedded in the temporal bone and is divided into two functionally separate receptor mechanisms:

The vestibule and semicircular canals (the ves tibular end organ).

The cochlea (the acoustic end organ).

The labyrinth can also be divided morphologically into *bony* and *membranous* parts.

**The bony labyrinth.** This is formed by the *labyrin thine capsule,* which develops by periosteal and enchondral ossification. In systemic bone diseases

(e. g., Paget disease and osteodystrophy) and in lo calized bone disease (e. g., otosclerosis), the bony labyrinth shows characteristic histopathological and chemical abnormalities. These conditions dem onstrate continuous bone remodelling.

The oval and round windows form the bony and membranous openings to the labyrinth from the middle ear cavity, and are closed by the stapes foot plate and round window membrane, respectively (see p. 5).

**Membranous labyrinth and inner ear fluids (Fig. 1.12a, b).** The *membranous labyrinth* develops from the ectodermal otic placode. It encloses a hol low system filled with *endolymph.* This passes via the endolymphatic duct to end in a blind sac, the *endolymphatic sac,* in the posterior cranial fossa. The sac lies in the epidural space on the posterior surface of the petrous pyramid, close to the sigmoid sinus.

The *perilymphatic system* forms a hollow space consisting of the scala tympani and the scala vesti buli. The system communicates directly with the subarachnoid space in the jugular foramen via the cochlear aqueduct. Perilymph separates the mem branous labyrinth from the internal layer of the labyrinthine capsule. Perilymph is the immediate substrate of the cochlear and vestibular sensory cells. The origin of perilymph is a matter of contro versy; it may form from filtration of perilymphatic capillary blood and/or through diffusion of cerebro spinal fluid.

*Endolymph* is a filtrate of perilymph that has completely different concentrations of sodium and potassium, which are kept constant by the epithelium of the *stria vascularis* (see **Fig. 1.18a**). The electrolyte composition of the endolymph reg ulates the volume of the fluid circulating in the endolymphatic system. The basis of the electrolyte exchange system, which maintains a constant ion concentration, is the cellular *potassium sodium ex change pump* found in the stria vascularis, the utricle, and the saccule. There is also passive diffu sion between the endolymphatic and perilym phatic spaces, with potassium sodium ion ex change in the endolymphatic sac. Functional dis turbances of this electrolyte regulation system lead to a disorder of the middle ear known as *Ménière disease* (see p. 97).

*Applied Anatomy and Physiology*

| 2  5  11  7  4  3  6  1  4  1  9  10  6  2 3 4  5  9  8  7  8  **a b** |
| --- |

**Fig. 1.12a, b a** The inner ear. , Oval window with stapes; , saccule; , utricle; , ampulla of the semicircular canals, with cupula; , membranous semicircular canals (horizontal, superior, and posterior); , ductus reuniens; , cochlear duct; , helicotrema; , the perilymphatic duct, which passes through the cochlear aqueduct; **10**, round window; **11**, endolymphatic sac on the posterior surface of the pyr amid.

The vestibular apparatus. , Lateral semicircular canal; , vertical semicircular canal; , posterior semicircular ca nal; , utricle; , saccule; , endolymphatic duct; , endo lymphatic sac; , ductus reuniens; , cochlea. Arrows mark the direction of velocity forces.

| 5  ~~4~~  3  6 1 2  **a b** |
| --- |

**Fig. 1.13a, b a** Static macula. A change in the polarity of the hair cells occurs below the striola. , type I hair cell; , type II hair cell; , gelatinous layer;

, statolith membrane; , stato liths, striola; , afferent nerve fibers.

Scanning electron-micro scopic image of calcium carbo nate crystals in the gelatinous layer of the utricle otoliths.

**Vestibular Semicircular Canal System** The anatomical fine structure of the balance mech anism system is shown in **Figs. 1.13a, 1.14 , 1.15**. It consists of the *utricle* and *saccule* enclosing the static *maculae* with the sensory end organs for the reception of linear acceleratory stimulation. These consist of *supporting cells* and *hair cells*, which have *cilia* embedded in a gelatinous mass consisting of sulfomucopolysaccharides. On their surface lie the *otoliths* (or statoconia), which consist of rhomboid calcium carbonate crystals (**Fig. 1.13b**). Linear ac celeration changes the otolith pressure, deflecting the sensory hairs. This stimulates the sensory cell by altering the *resting potential.*

The three semicircular canals arise from the utricle and have a pear-shaped expansion at one

end called the *pars ampullaris,* enclosing the sen sory cells, which are stimulated by angular accel eration (**Fig. 1.16**). The sense organs consist of an *ampullary crest* (crista ampullaris), on which *sen sory hair cells* are arranged in such a way that their cilia extend to the *cupula,* which reaches to the roof of the ampulla. The cupula acts as a mobile partition that closes off the pars ampullaris and is relatively impervious to endolymph (**Fig. 1.15**).

Note: The hair cells of the maculae and ampullary crests have similar structural principles. They are mechanore ceptors that respond to tangential bending of their cilia.

**10 1 Ear**

| 1  2  3  4  5  6 |
| --- |

**Fig. 1.14** A receptor in the semicircular canal. , Cupula; , cilia; , sen sory cells; , supporting cells; , crista ampullaris; , afferent nerve fibers.

| 1  4  3  2 |
| --- |

| 4  1  2  3 |
| --- |

**Fig. 1.15** The ampulla of a semicircular canal. , Cupula; , crista ampullaris; , afferent nerve fibers; , membranous semicircular canal.

**Cochlea (Acoustic End Organ)**

The macroscopic and microscopic structure of the bony and membranous cochlea are shown in **Figs. 1.17a,b, 1.18a**

**Functional structure of the organ of Corti.** The bas ilar membrane supports the sensory apparatus of the organ of Corti. It stretches between the bony spiral lamina and the lateral cochlear wall and forms the border to the scala tympani. Surrounded by supporting cells, there are two types of receptor cells: one row of inner and three rows of outer hair

**Fig. 1.16** Oscillation of the cupula. When the head is rotated (arrow), the semicircular canals rotate as well. Ow ing to its viscosity, the endolymph initially remains motion less and directs the cupula in the opposite direction. This causes the cilia to bend. , Labyrinth; , membranous canal of semicircular canal; , cupula; , vestibular nerve.

cells, totaling 16 000 sensory cells. The hair cells have fine cilia on their free surfaces, with approx imately 80 cilia per cell. So-called *tip links* 10 thick, extend from the tips of the small cilia to the longer, very fine protein strings. There are ion chan nels where the tip links connect to the cilia, provid ing the basis for transduction of the sound stimulus to a receptor potential. Lying on top of the organ of Corti is the gelatinous tectorial membrane. The cilia of the outer hair cells lie below the tectorial mem brane, while the cilia of the inner hairs cells do not insert into the tectorial membrane. The hair cells are secondary sensory cells and have no nerve cell processes. They receive fibers from the spiral gan glion. Approximately 90 % of the nerve fibers ex tend to the inner hair cells, and each inner hair cell is connected to many afferent fibers, each of which undividedly connects to an individual hair cell. The remaining 10 % of the nerve fibers are widely den dritic and innervate the outer hair cells. There are

| 7  6  8  5  9  1  10  2  11 12  14  3  **a**  4  9  8  3  ~~13~~  2  5  10  **b** |
| --- |

**Fig. 1.17a, b** Axial cross-section through the cochlea ( and cochlear canal (spiral canal) ( ). The cochlea is arranged spirally (with two and a half turns) around the central mod iolus ( ) lying horizontally. Its base lies against the lateral end of the internal acoustic meatus, and its apex is directed anterolaterally toward the medial wall of the middle ear. The spiral ganglion i. e., the ganglion of the cochlear nerve

is located within the modiolus, and its nerve fibers ( join to form the stem of the cochlear nerve, the pars coch learis of the vestibulocochlear nerve ( ). The osseous spiral lamina or spiral plate ( ) is a bony plate that runs spirally from the base to the apex ( ). Nerve fibers pass through the channels of the spiral lamina to the spiral organ of Corti (12). The cochlear duct (scala media) ( ), filled with endo lymph, lies between the scala vestibuli ( ) above and the scala tympani (**10**) below, both of which contain perilymph

). The osseous spiral lamina ( ) and the basilar membrane form the separating wall between the scala tympani, on the one hand, and the scala vestibuli and cochlear duct on the other. The Reissner membrane (**11**) separates the scala vestibuli and the cochlear duct. The tectorial membrane

**12**) covers the sensory cells of the organ of Corti. The stria vascularis (**14**) forms the lateral wall of the cochlear duct and has numerous vessels. This layer of fibrous vascular tissue is the site of production of the endolymph. Laterally, it borders on the spiral ligament of the cochlea (**13**). The perilymphatic spaces of the cochlea, the scala tympani and scala vestibuli, communicate with each other at the apex of the cochlea **a, 7**), at the helicotrema, (see **Fig. 1.12a, 8**) and are also connected with the perilymphatic space of the membranous labyrinth of the vestibule, containing both the utricle and the saccule (see **Fig. 1.12a, 2** ).

*Applied Anatomy and Physiology* **11**

30 000 40 000 axons that lead from the spiral ganglion to form the vestibulocochlear nerve (**Fig. 1.19**).

Note: The entire frequency spectrum of 18 20 000 Hz is represented in the hair cells of the organ of Corti over the entire basilar membrane. The highest frequencies are localized to the most basal segment of the cochlea and the lowest frequencies near the helicotrema in the apical turn. This arrangement forms the morphologic basis of the tonotopic organization of the cochlea i. e., the point-to-point connection between the sound wave re ceptors and the signal-converting central neurons of the auditory system.

**Central Connections of the Organ of Corti** The cochlear division of the eighth cranial nerve (pars cochlearis) is formed by the bipolar neurons of the spiral cochlear ganglion. It runs through the internal auditory meatus, unites with the vestibular division, crosses the cerebellopontine angle, and enters the brain stem at the lower border of the pons, at which point the central auditory pathway begins (**Fig. 1.20**).

The central auditory radiation incorporates the strict tonotopic arrangement, as does the *auditory cortex.* The cochlea is thus represented unrolled, as it were, from the basal turn to the helicotrema. The *auditory cortex* is consid erably larger than the area of Heschl s transverse striations, since these represent only the *primary auditory field* (AI) in which the auditory radiation ends. The secondary acoustic field (AII) and the posterior ectosylvian gyrus, like the visual cortex, include secondary integration areas such as the *Wernicke speech center.* Numerous commissural systems al low fibers to be exchanged between the two halves of the brain. These are very important for directional hearing.

**Central Connections of the Balance Mechanism** The bipolar neurons of the vestibular ganglion send out their peripheral processes as two divided neural bundles a superior division to the sensory cells in the macula of the utricle, the lateral and superior semicircular canals; and an inferior division to the posterior semicircular canal and the macula of the saccule (**Fig. 1.21**).

The central processes combine to form the ves tibular division of the eighth cranial nerve, which

**12 1 Ear**

| **b**  13  5  7  1112  6  89  10  14  2  1  1  2  3  4  15  5  34  **c**  **a** |
| --- |

**Fig. 1.18a c a, b** The cochlear duct ( ) and spiral organ of Corti ( ). The spiral organ of Corti ( ) rests on the basilar membrane ( ) in the cochlear duct. Medially, at the free edge of the osseous spiral lamina, lies the limbus of the spiral lamina ( ), with two labia enclosing the internal spiral sulcus

). The highly vascularized stria vascularis ( ) with intra epithelial capillaries lies laterally. The spiral organ of Corti ( consists of inner hair cells ( ) and outer hair cells ( ) sup ported by pillar cells ( ), constituting the borders of the inner tunnel (perilymph or cortilymph, **14**). Between the outer pillars ( ) and external phalangeal cells of Deiters **10**), which act as supporting cells for the spiral organ of

**Fig. 1.19** Scanning elec 

tron-microscopic image of

the spiral organ of Corti,

with a view of the surface

of the basilar membrane.

There are three rows of

outer hair cells in the lower

part of the picture and one

row of inner hair cells in the

upper left corner of the

picture.

unites in the internal auditory meatus with the cochlear division to form the vestibulocochlear nerve which has a common nerve sheath. The ves tibular division sends ascending fibers to the ves tibular centers after it has entered the medulla oblongata. The *secondary vestibular pathway* is con nected to the spinal cord by the *vestibulospinal tract.* Its fibers end at the spinal intermediate neu rons and activate the alpha and gamma motor neu rons of the extensor muscles. They are therefore the antagonists of the pyramidal pathway and mainly produce flexor inhibition and activation of exten sors. They form part of a phylogenetically old anti gravity system that serves to maintain balance. In

Corti, lies the Nuel space, with perilymph (**11**). In the ex treme lateral position, there is the outer tunnel (**12**), which borders on the external spiral sulcus (**15**) and the stria vascularis ( ), respectively. Above the hair cells (inner and outer, ) is the tectorial membrane (**13**), a gelatinous mass extending from the limbus of the spiral lamina ( ). The intercellular spaces of the spiral organ (**11 12 14**

contain perilymph, also known as cortilymph. The ultrastructure of the inner and outer hair cells. , Inner hair cells; , outer hair cells; , afferent nerve endings; , efferent nerve endings; , cilia.

addition, there are important ascending pathways to the cerebellum, the reticular formation (a multi sensory integration center), and the centers for the eye muscles (where the oculomotor muscles are coordinated), via the *medial longitudinal bundle.*

A vestibulocortical connection is provided via the thala mus. Vestibular stimulation is projected to a small area in the *ventral postcentral somatosensory region,* near the visual area. This region represents a primary vestibular cortical area.

Note: Connections between the vestibular centers, the centers for the ocular muscles, and the cervical muscu lature, together with the cerebellum, form the morpho logic basis for the extremely precise coordination of the three functional systems. This allows objects to be visu ally fixed even when the head is moving. Synchronized coordination of the ocular and cervical muscles is con trolled through the vestibular apparatus via the gamma neurons.

| 9  8  10 10  7  6  5  3  a  2  b  1 11 4 |
| --- |

**Fig. 1.20** The afferent auditory pathways. For the sake of simplicity, the pathways for only one cochlea are shown. , Direct auditory pathway; , indirect auditory pathway; , cochlea; , ventral cochlear nucleus; , posterior cochlear nucleus; , superior olivary nucleus; , nuclei of the lateral lemniscus; , lateral lemniscus; , inferior colliculus; , me dial geniculate body; , acoustic radiation; **10**, auditory cortex; **11**, vestibulocochlear nerve.

**Facial Nerve**

The seventh cranial nerve carries *motor fibers* for the mimetic muscles of the face, *afferent sensory taste fibers* and *visceroefferent secretory neurons* in a separate nerve bundle, the intermediate nerve. The nerve also contains the sensory fibers that supply the posterior wall of the external auditory meatus. This explains the reduced sensation of this area of skin in patients who have a vestibular schwannoma *(Hitselberger sign)* **Fig. 1.22**).

The motor fibers originate from the facial motor nucleus in the floor of the fourth ventricle, run round the abducens nucleus (the internal genu ), and exit at the lower border of the pons, together with the *visceroefferent fibers* of the intermediate nerve arising from the superior salivatory nucleus. The *gustatory fibers* insert into the subcortical taste centers in the nucleus of the solitary tract. All of these branches form the *nervus intermediofacialis,* which runs first in the internal auditory meatus (the meatal segment). It enters the bony canal im mediately adjacent to the labyrinth (the labyrin

*Applied Anatomy and Physiology* **13**

| 10  1  11  2  12 Cerebellum  3  45  6  7  8  9 |
| --- |

**Fig. 1.21** The central vestibular connections in the brain stem. , Trochlear nucleus; , abducent nucleus; , inferior cerebellar peduncle; , superior vestibular nucleus (Bekh terev nucleus); , lateral vestibular nucleus (Deiters nu cleus); , inferior vestibular nucleus; , medial vestibular nucleus; **11**, medial longitudinal bundle; , lateral vesti bulospinal tract; **10,** oculomotor nucleus; **12**, vestibulo cerebellar nerve fibers.

| 1  15  9  2  3  8  6  10  4  5  7  11  Motor fibers  14  Secretory fibers  12  Sensory fibers  13 |
| --- |

**Fig. 1.22** Course of fibers in the facial nerve. , Abducent nucleus; , secretory nucleus of the nervus intermedius; , motor nuclei of the facial nerve; , nucleus of the solitary tract; , geniculate ganglion; , greater superficial petrosal nerve; , chorda tympani; , pterygopalatine ganglion with the lacrimal anastomosis; , lacrimal gland with greater superficial petrosal nerve; **10**, nasal glands; **11**, taste fibers to the anterior two thirds of the tongue; **12**, sublingual gland; **13**, submandibular gland; **14**, submandibular gan glion; **15**, trigeminal ganglion.

thine segment) and runs to the hiatus in the canal for the facial nerve. At this point, the greater super ficial petrosal nerve divides off from the main

**14 1 Ear**

trunk. This branch goes to the lacrimal gland and also supplies fibers to the glands of the nasal mu cosa. The first genu of the facial nerve lies at the level of the geniculate ganglion. The nerve then turns into the horizontal *tympanic segment* before it passes at the level of the entrance to the mastoid antrum, the second genu, into the vertical *mastoid segment.* In this area, it branches to the stapedius muscle and the chorda tympani, which contains taste fibers for the anterior two-thirds of the tongue and carries visceroefferent fibers for the sublingual and submandibular glands. After leaving the mas toid process through the stylomastoid foramen, it divides into five extratemporal branches the tem poral, zygomatic, buccal, marginal mandibular, and cervical to the platysma. These branches are highly variable (see **Fig. 1.114**).

The facial nerve is surrounded by a tough fibrous sheath in its course through the temporal bone. Its individual fascicles are embedded in a well-devel oped *epineurium* of loose connective tissue that encloses the vessels and nerves. The fiber bundles are enclosed in a *perineurium.* When injuries to the nerve are being repaired, the epineurium has to be resected from the stump, and a perineural suture has to be used so that the site of anastomosis can be adapted precisely, to prevent the formation of a scar tissue neuroma due to connective-tissue infiltra tion of the anastomosis (see p. 114).

Note: Familiarity with the details of the regional anat omy of the facial nerve is a prerequisite for understand ing the neurologic diagnosis of facial paralysis (the differ ential diagnosis of central and peripheral paralyses and the topographic diagnosis of the lesion;see p. 47).

**Physiology and Pathophysiology of Hearing and Balance**

**Physiology of Hearing: Middle and Internal Ear**

The functions of the various parts of the ear are as follows:

The external and middle ear transport the stim ulus.

The cochlea distributes the stimulus.

The function of the outer hair cells is mechano electric transduction.

The inner hair cells transform the stimulus.

**Stimulus Transport**

In the *external auditory meatus,*the resonance effect lowers the hearing threshold to between 2000 and 3000 Hz, the main range of speech frequencies.

The *tympanic membrane* is a sound pressure receptor and transformer.

The *ossicular chain* is responsible for impedance adaptation between the middle ear, in which the medium is air, and the inner ear in a fluid medium, as well as *pressure transformation.* The pressure enhancement is 1 : 17, due to the ratio between the surface of the tympanic membrane and the stapes footplate. The ratio due to the mechanical advantage of the incudomalleolar joint is 1 : 1.3. The total pressure on the stapes footplate is therefore increased 22 times (see **Fig. 1.9**).

The physical movements of molecules that we perceive as sound set the tympanic membrane in motion. The frequency of the motion is the same as that of the vibrations of the air, and its amplitude is proportional. The transmission of sound waves from the air medium to the fluid medium in the perilymphatic and endolymphatic space requires a relative increase in power, due to the increase in density i. e., impedance adaptation through sound pressure transformation (impedance = acoustic re sistance).

For normal transmission of sound to the inner ear, the tympanic membrane has to be in a normal position and have normal mobility, and the air pressure in the outer and middle ears has to be similar. Measuring the impedance at the tympanic membrane can provide information about the func tioning of the sound transmission apparatus, and this method known as *impedance audiometry* is used for clinical investigations (see p. 36). Sound energy reaches the cochlea firstly via the sound transmission apparatus of the middle ear *(air con duction)* and secondly through the bone of the skull, which is set in motion in a sound field. The sound energy is thus transmitted directly to the cochlea via the labyrinthine capsule *(bone conduction).*

*Audiometry* is used to measure the hearing threshold for both air and bone conduction (see p. 27).

**Stimulus Distribution**

The main function of the cochlea is *mechanical frequency analysis,* which depends on its *hydrody namics.* Periodic movements at the stapes are con verted into aperiodic movements to produce a trav

eling wave on the basilar membrane (**Fig. 1.23**). Since the inner ear fluids are not compressible, volume displacement at the stapes footplate leads to an equal volume displacement at the round win dow, and this produces a bulging of the round win dow membrane that is equal in extent to the de pression of the stapes footplate. This volume dis placement, produced by *periodic vibrations* of the stapes footplate, leads to displacement of the *coch lear duct* (scala media, Löwenberg scala; the space surrounded by the basilar membrane and Reissner membrane, between the scala vestibuli and scala tympani) (see **Fig. 1.17a,b**). This initial displace ment forms a wave motion that proceeds along the partition to the helicotrema. This is an *aperiodic vibration,* or traveling wave. The wavelength be comes shorter as the wave approaches the helico trema, but the amplitude becomes greater. The am plitude reaches a maximum at one specific point and then immediately begins to fall sharply, before dying away toward the helicotrema. The traveling wave causes a displacement between the tectorial membrane and the basilar membrane at its point of maximal amplitude, so that the cilia of the hair cells are displaced at this point, forming the sensory stimulus for these mechanoreceptors (see **Figs. 1.18c, 1.25b**).

The frequency-dependent development of the maximal amplitude on the traveling wave induces a corresponding *frequency-dependent localized stimulus* on the basilar membrane in the sensory cells of the organ of Corti that lie at the point of maximal amplitude. An initial analysis of the sound is thus achieved in accurately defined frequency stimulus patterns (Békésy s dispersion or traveling wave theory).

The maximum displacement of the traveling wave lies at a different point for each frequency: it is nearer the helicotrema for the lower frequencies and nearer the stapes footplate for the higher. The tonotopic arrangement of the cochlea means that every frequency is thus represented at a particular point on the basilar membrane (**Fig. 1.24**). Since the distribution of the maximal amplitude across the basilar membrane determines the point of excita tion of the organ of Corti and thus the activity of the afferent nerve fibers in the cochlear nerve, the trav eling wave hypothesis is also a one-point hypo thesis, as suggested by Helmholtz. Each point on the basilar membrane therefore corresponds to a spe cific frequency.

*Applied Anatomy and Physiology* **15**

| 1  3  2  6  4  5 |
| --- |

**Fig. 1.23** Three-dimensional representation of the vibra tion of the basilar membrane. The traveling wave runs from the stapes along the basilar membrane, the tectorial mem brane, and the Reissner membrane to the apex of the coch lea. The location of the maximum elongation of the basilar membrane is similar to the formation of a frequency dependent maximum amplitude. , Stapes in the oval win dow; , round window; , scala vestibuli; , scala tympani;

, basilar membrane with spiral organ of Corti; , maximum amplitude of the traveling wave.

| 4000 Hz  900 Hz  200  Hz  500  Width  Hz  Width  0.34 mm  0.5 mm  1500 Hz  Top  Width  0.1 mm  Base  20 000 Hz |
| --- |

**Fig. 1.24** The human basilar membrane, showing the fre quency-dependent locations of sound receptors and analyz ing receptors.

**16 1 Ear**

**Mechanoelectric Stimulus Transduction** The cilia of the outer hair cells are bent to the great est extent when the wave motion approaches the maximum range. A force pushing on the tip links

| K+  K+  K+ K+  K+  2  K+  1  Excitation Inhibition |
| --- |

causes the ion channels to open and changes the receptor potential. The outer hair cells carry out active, oscillating extension and thus locally inten sify the travelling wave (**Fig. 1.25a**).

**Stimulus Transformation**

The actively intensified vibrations of the inner hair cells also cause the cilia of the inner hair cells to bend with the resulting opening of the ion chan nels. An influxof Ca2 + causes a basal discharge of glutamate as a transmitter, and the afferent nerve fibers of the vestibulocochlear nerve are conse quently stimulated (**Fig. 1.25b**).

**Otoacoustic Emissions**

Active contractions of outer hair cells have natural modes of vibration and are subject to distortion. In this phenomenon of normal hearing, sounds emit ted by the cochlea occur at certain frequencies as *spontaneous otoacoustic emissions* (SOAEs). *Evoked otoacoustic emissions* (EOAEs) can be recorded in the external auditory canal after induction by ex ternal acoustic stimuli (see p. 39).

| K+  K+  150 mM  5  + 85 mV  K+  1  K+  + 155 mV  Ca2+  K+  -70  -70  Cl- Cl  mV  mV  0 mV  K+ K+  K+  2 3 2 4  K+  K+  3 mM |
| --- |

**Fig. 1.25a, b a** Depolarization (excitation) of the sensory hair cells by deflection of the cilia ( ) and opening of stretch sensitive potassium ion channels. Stretching of the channels is induced by tension to the tip links ( ). K ions escape the hair cell at the base through stretch-sensitive channels, lead ing to repolarization of the cells.

The spiral organ of Corti, showing the electromotility of the outer hair cells acting as a cochlear amplifier. The fre quency-dependent length changes in the outer hair cells (blue) vibrate the spiral organ of Corti and thereby stimulate the inner hair cells (red), which are normally not in contact with the tectorial membrane ( ). The influx of K into the

hair cells is necessary for depolarization. This occurs through the high K concentration of the endolymph and the endo cochlear potential (+ 85 mV), which amounts to as much as 155 mV between the hair cell (resting potential 70 mV) and endolymph. Potassium ions leave the cells basolaterally by means of excitation-dependent K channels ( ) and are led through the cortilymph by means of K Cl cotransporters

) and nexus channels ( ) in the support cells into the spiral ligament. Excitation-dependent Ca2 + channels ( ) regulate transmitter release during depolarization through the influx of Ca2 + ions.

**Physiology of Hearing: Retrocochlear Analysis of Acoustic Information**

The electrical stimulus pattern of sensory cells in the organ of Corti is converted in the peripheral cochlear neuron into the action potential pattern of the vestibulocochlear nerve. The sound stimu lus which has many parameters, such as fre quency, intensity, temporal pattern, and the perio dicity of the action potentials has to be encoded to allow the information to be analyzed in the central nervous system.

*Sound frequency* and *sound intensity coding* play a very important role in the central analysis of the acoustic signal.

*Sound intensity coding* occurs through frequency modulation. With increasing sound intensity, the number of spikes in the sensory cell dis charge increases.

In *sound frequency coding*, specific sensory cell groups in the organ of Corti are stimulated de pending on the sound frequency. Tonotopicity (see below) allows these locally circumscribed stimulus patterns, produced on the basilar membrane, to be conducted by the vestibuloco chlear nerve to the higher centers without dis tortion.

*Tonotopy* is a point-to-point connection between the sound receptors and the neurons analyzing the signal. Each cochlear neuron what is known as

*best frequency* i. e., it responds only to an acoustic stimulus that has a frequency identical to the fre quency assigned to it.

The acoustic system can process the duration, intensity, and frequency parameters of the acoustic signal in the following ways:

With increasing intensity and constant fre quency, the action potential rate in the nerve fibers increases, and the number of stimulated afferent neurons also increases, corresponding to the extent of the deflected area of the basilar membrane.

At constant intensity and variable frequency, the deflected area of the basilar membrane is dis placed into the appropriate segment of the or gan of Corti within the cochlea, so that frequency is determined by point analysis. In addition, changes occur in the periodicity of the action potential series within the individual nerve fi bers, which are analyzed by means of periodicity

*Applied Anatomy and Physiology* **17**

analysis. This provides another means of fre quency determination.

*Frequency analysis* by means of local pattern scan ning, *intensity perception* by frequency modulation, and *time-periodicity analysis* by combined evalua tion of the time and place pattern also provide information that passes to the higher auditory cen ters as a result of tonotopicity (**Fig. 1.26a, b**).

**Pathophysiologic Basis of Hearing Disorders**

*Conductive or middle ear hearing loss* is caused by lesions of the stimulus transport organ. A character istic symptom of this type of hearing loss is that bone conduction functions better than air conduc tion. The depression of the hearing threshold for air conduction is associated with an increase in acous tic impedance, as seen with stapes fixation due to otosclerosis.

*Sensory hearing loss* is caused by lesions in the stimulus transformation organ and/or in the vesti bulocochlear nerves, and is therefore better known as *sensorineural hearing loss. Noise-induced hearing loss* and *age-related hearing loss (presbyacusis)* are caused mainly by mechanical overloading of the cochlear amplifier system of the outer and inner hair cells.

*Disorders ofsound perception* are caused by le sions in the subcortical or cortical auditory centers and by pathologic processes involving the central auditory pathway. As a result, the acoustic signals are falsely coded, stimulus patterns are wrongly analyzed, and acoustic information can no longer be integrated. The patient can then hear but not understand.

*Central hearing disorders* are characterized by a loss of the integrative functions of the auditory centers. Differences in level of tone, differences in loudness, and temporal differences of acoustic stimulus pattern can no longer be analyzed. Redun dancy is also reduced i. e., the information content is reduced due to loss of secondary and tertiary cochlear neurons. These disorders affect the under standing of speech (whereas hearing of pure tones may be preserved), directional hearing, and speech intelligibility.

*Recruitment:* In certain forms of unilateral sen sorineural deafness, the loudness perception rises quickly with increasing loudness intensity, so that despite different hearing thresholds both ears hear

**18 1 Ear**

| ∍s 12 cm  ∍s  ∍t =  355 m ~~.~~ s~~-~~1  ∍t 350 Πs  ∍t ∍s  **a**  ∍t  **b** |
| --- |

**Fig. 1.26a, b** Important connections for directional hear ing. When level differences are being determined to identify the sound source ( ), the highly stimulated neurons on the lateral superior olivary nucleus are on the sound side (low inhibition, intense excitation). With the interaural time dif ference t ( ), simultaneous maximal excitation only takes place in the neurons of the medial superior olivary nucleus on the side turned away from the sound source.

the tone at the same loudness once a certain thresh old is reached. This phenomenon is called recruit ment. The pathophysiologic basis for recruitment is loss of the cochlear amplifier mechanism, with ab normal sound processing dynamics of. *Positive re*

| Thalamus  Hypothalamus  1  Cerebellum  (fine motor  control)  2  4  Labyrinth  3  Vestibular nuclei |
| --- |

**Fig. 1.27** The input and output of the vestibular nuclei. , Visual information; , vestibular information from the semicircular canals and otolith apparatus; , kinesthetic in formation from the superficial and deep receptors in the skin, muscles, tendon, and joints, which react to pressure and traction forces caused by the force of gravity and inertia; , vestibular nuclei.

*cruitment* can generally be regarded as a sign of a cochlear lesion, whereas *absent recruitment* indi cates a retrocochlear lesion localized to the first or second neuron.

**Physiology of the Balance System**

Balance is maintained by coordination of visual kinesthetic and vestibular regulatory mechanisms. These serve for *spatial orientation, upright posture,* and *gait.* Control of all the static and motor muscle groups allows the body to counteract the influence of weight and centrifugal forces (**Fig. 1.27**).

The main functions of the vestibular system are: To send information to the central nervous sys tem about the action of linear and angular accel eratory forces.

Coordination. Movement is coordinated by con tinuous control of the tone of the skeletal muscles. Information from the vestibular sen sory receptors is coordinated and integrated with information from the visual system. Spatial orientation is also ensured.

The potential difference between the sensory cells and the extracellular fluid forms the physiologic basis for normal functioning of the vestibular sense organ. A constant discharge of action potentials passes along the vestibular nerve fibers, even

*Applied Anatomy and Physiology* **19**

| 1 11  3  2  4  5  Time Time  Time  6  **abc** |
| --- |

**Fig. 1.28a** The bioelectrical activity of the vestibular sensory cells at rest and in response to stimulation. Bending of the sensory hair cells away from the kinocilium ( ) causes hyperpo larization and inhibition of the resting activity ( ). Deflection in the opposite direction, toward the kinocilium ( ), causes depo larization and an increase in the discharge frequency of the ac tion potential. , Gelatinous layer; , cilia; , kinocilium;

, sensory cell; , synapse of the afferent nerve; , afferent nerve fiber.

when the end organs are at rest *(resting activity).* As in the cochlea, a transduction channel in the ves tibular hair cells is opened by a force pushing on the tip links, allowing an influxof ions and causing the receptor potential to change. Depending on the direction of the ciliary deflection of the sensory hair cells, the resting activity is altered by an in crease in the discharge frequency *(depolarization)* or by inhibition *(hyperpolarization)* **Fig. 1.28a** ). Modulation of resting activity thus allows the body to sense movement both in one direction and also in the opposite direction using a single receptor.

**Function of the Otolith Organ: Linear Accelera tion Measurement**

*Linear acceleration* is the sensory stimulus for the horizontally orientated macula of the utricle and the vertical macula of the saccule. Shearing forces occur during linear acceleration that shift the oto liths from their base, causing shearing of the hair cells (see **Fig. 1.12b**) and providing an adequate stimulus for the sensory cells. The resulting neuro nal impulses release the *maculo-ocular reflex,* pro ducing compensatory eye movements that ensure optimal static positioning of the eyes during linear movement. The *maculospinal reflex* is also evoked, which influences the musculature of the trunk and limbs via the motor anterior horn cells in the spinal cord to ensure that the position of the body remains stable during linear movement. The otolith appara tus also has another important function: due to the continuous effect of gravity, the otoliths exert con stant pressure on the underlying sensory cells, even at rest. This pressure influences the resting activity

of these mechanoreceptors. Linear acceleration e. g., a fall, rapid lowering of the head, air travel, or fast movement in an elevator changes this resting activity, thus guaranteeing continuous spatial ori entation during vertical movement.

**Function of the Semicircular Canals: Angular Acceleration Measurement**

Positive or negative angular acceleration causes endolymphatic movement within the semicircular canals lying in the plane of the centrifugal force. The stimulus always affects the semicircular canals on both sides; the cupula is displaced toward the utricle on one side *(ampullopetal stimulation)* and in the opposite direction on the other side *(ampul lofugal stimulation).* As a result, resting activity in creases in the semicircular canal in which the cu pula is deflected in an ampullopetal direction (de polarization effect), whereas activity decreases in the contralateral canal (hyperpolarization effect). This rule applies only to the horizontal canals, since ampullofugal deflection causes depolarization in the vertical semicircular canals. This is the neuro physiologic basis for the stimulating mechanism of the vestibuloocular reflex.

The *vestibuloocular reflex* also serves for spatial orientation. It addition, it assists in stabilizing the retinal image of the visual environment and indu ces vestibular nystagmus. Every movement of the head causes slow, conjugated movement of the eyes in the opposite direction, to stabilize the field of vision on the retina for as long as possible during the movement. Two modifiable parameters deter mine the progress of the vestibuloocular reflex: the

**20 1 Ear**

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**Fig. 1.29** Pathogenesis of disorders of orientation and balance. *Disorders of proprioceptive information:* loss of con trol over the ability to stand upright and walk straight causes a balance disorder. *Disorders of visual information:* loss of optical control of the visual field occasionally leads to *dizzi ness* due to a discrepancy between visual and vestibular information, causing disorientation. *Disorders of vestibular information* are due to involvement of the spatial orientation and stabilization of the gaze axis, leading to contradictory vestibular visual and kinesthetic information and *dizziness.* If central compensation of the loss of vestibular function is also absent, there is an additional balance disorder.

position of the head and the position of the eyes. The difference between these is the angle of vision (see **Fig. 1.16**).

Note: The vestibuloocular reflex coordinates the speed of reflex eye movements (the slow component of nys tagmus) with the speed of head movement. This ensures clear visual control of the environment during move ment. Fast return of the eyes is achieved by a reflex, the fast component of nystagmus.

Conjugated eye movements due to the vestibulooc ular reflex, with typical slow and fast components, are classified as vestibular nystagmus (see p. 42).

The intervertebral joints of the cervical spine and the deep muscles of the neck contain mechanoreceptors, which are connected to the reticular formation by afferent fibers and from there to the vestibular and oculomotor centers. The function of these receptors is to provide continuous information about the position and movement of the head and to allow coordination of eye movements through the *cervicoocular pathway.*

The central *vestibular system* includes the cere bellum and the reticular formation of the brain stem i. e., it is integrated into the centers for multi sensory data analysis. This allows multisensory control and coordination of posture, movement, and oculomotor functions.

**Pathophysiologic Basis of Functional Vestibular Disorders**

Vestibular disorders become manifest through: *Vertigo:* partial or complete loss of spatial orien tation e. g., apparent movement of the environ ment as a result of spontaneous vestibular nys tagmus; and/or

*Disturbed balance,* with an inability to maintain balance, stand upright, or walk properly (ataxia) **Fig. 1.29**).

*Vestibular disorders* may be *peripheral,* caused by sudden unilateral failure of one labyrinth, or by a unilateral lesion of the vestibular nerve. They may also be *central,* caused by a lesion in the vestibular centers or their central connections to the cerebel lum and reticular formation.

Every functional disturbance in a vestibular end organ causes unequal activity in the higher vestib ular centers. This central imbalance initially produ ces a disturbance of vestibular information. The multisensory spatial orientation is therefore no lon ger capable of functioning, since vestibular informa tion on the one hand and visual somatosensory in formation on the other hand contradict each other. This causes a disturbance of orientation, which in turn causes dizziness. If the central imbalance in the two vestibular centers influences the main neigh boring coordination centers for eye movements in the reticular formation of the brain stem, sponta neous abnormal eyemovements occur that have the characteristics of nystagmus (**Fig. 1.30**).

*Peripheral functional failure* is compensated cen trally by adjustment of the difference in neuronal activity in the vestibular centers and by substitution of visual and somatosensory regulatory mecha nisms for the loss of peripheral vestibular function. This process is called *central vestibular compensa tion.* Central vestibular disorders are only incom pletely compensated by the above mechanisms (or not at all), since the multisensory connections to the vestibular centers are damaged.

|  |
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**Fig. 1.30** Oculomotor system. All three sensory systems ) send afferent signals via relay stations ( ) in the premo tor centers of the reticular formation of the brain stem ( ). The motor neurons ( ) that innervate the eye muscles begin at this point. The cerebellum is the key to coordination: visual, somatosensory, and vestibular signals are continually being compared with one another. If this structure receives contradictory information that could lead to disorientation and dizziness, the vestibular signal is modified or, if neces sary, completely suppressed.

**Methods of Investigation**

**Inspection, Palpation, Otoscopy, Microscopy**

**Inspection of the External Ear**

The physician should look for redness, swelling, ulceration, tumors, malformations, fistula, or retro auricular scars.

**Palpation**

The mastoid process should be palpated with both hands to search for swelling and for sensitivity to pressure on the surface of the mastoid process and at its apex. The auricle is examined for pain when pressure is applied to the tragus or when the auricle is pulled. Finally, the regional lymph nodes in the preauricular and postauricular areas and the upper deep cervical chain are examined.

**Otoscopy**

The external auditory meatus and the tympanic membrane are examined, and if a perforation is present, the middle ear is also examined.

*Methods of Investigation* **21**

**Fig. 1.31** Otoscopy with 

an illuminated otoscope,

which consists of a dispos

able ear speculum, a light

source, and a magnification

attachment.

*Indirect illumination with a head mirror* is a dif ficult method of investigation for the nonspecialist, as correct adjustment of the light source and head mirror require time and practice, especially when patients are being examined in bed (see **Fig. 2.16).**

The electrical *otoscope* is more widely used, as it is easier to handle. It consists of a combination of an interchangeable *ear speculum* with a small, but strong, built-in low-voltage *light source* and a *mag nification attachment* providing a magnification of 1.5 **Fig. 1.31**).

The *otomicroscope* provides a magnification of 12 and is indispensable for accurate examina tion of the meatus, tympanic membrane, and parts

of the middle ear in cases of perforation. The *oto-endoscope* provides a wide-angled and magnified view over the tympanic membrane, al lowing complete investigation of the anulus and anterior tympanomeatal angle. Rigid scopes with and 30 views are used.

**Technique of otoscopy.** The cartilaginous part of the external meatus is stretched by pulling the auricle upward and backward. The speculum is then introduced into the long axis of the bony meatus. The instrument is held with one hand, so that the other hand remains free for handling instruments such as cotton-wool probes, hooks, an aspira tor, and aural forceps (**Fig. 1.32**). The speculum has to be introduced carefully, and the end of it should not be moved abruptly, as its opening has relatively sharp edges. The wall of the bony meatus is particularly sensitive and easy to injure, and contact with it should therefore be avoided.

**22 1 Ear**

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**Fig. 1.32** Important ear in struments. , Politzer bag with olive; , tuning fork;

, suction tube; , curette; , driller for cotton pads; , hook; , microforceps; , ear speculum; , hearing

tube with olives (of various sizes).

In *infants* and *young children,* the auricle is pulled down ward and backward to allow the speculum to be introduced. The short cartilaginous part of the external meatus is re duced to a cleft, which can only be entered with a narrow speculum with a small lumen, making otoscopy difficult. The head has to be immobilized, either by an assistant or using a headrest on the patient s chair, to prevent unnecessary movements that can cause pain.

Wax and other material obstructing the view into the external auditory meatus has to be removed using the following methods:

By syringing for foreign bodies, wax, and exudate. With the hook or curette, for hard wax.

With the aural aspirator, for exudate or fluid wax. With a cotton-wool probe for exudate.

The ear is syringed with tap water at body temperature. Hard wax is softened beforehand with softening drops such as 3 % hydrogen peroxide, 5 % sodium bicarbonate, soft soap, olive oil, or a commercial preparation.

Note: Syringing the ear is contraindicated in: Dry perforations of the tympanic membrane Fresh injuries to the tympanic membrane and meatus Longitudinal and transverse fractures of the petrous pyramid, with meatal trauma

It is important to obtain a history of any previous perfo ration, as syringing may rupture a thin scar. In the United States, failure to take a history can result in a malpractice suit.

Mistakes to be avoided:

A speculum that is too narrow and that penetrates too deeply into the sensitive bony meatus.

Introducing the speculum in the wrong direction e. g., from above downward.

Not introducing the speculum far enough, causing its opening to be blocked by otic hairs.

Unsatisfactory cleaning of the external meatus, so that a proper view of the tympanic membrane is not obtained.

**Otomicroscopy.** This is performed with a speculum under the operating microscope, with a magnifica tion of 6 40 , in all cases in which routine oto scopic examination does not allow reliable assess ment of the tympanic membrane (**Fig. 1.33**).

**Oto-endoscopy.** This is performed with a tele-oto scope (0 and 30 view). This allows examination of the whole tympanic membrane, meatus, and anu lus, as well as assessment of perforations and pock ets in the tympanic membrane, and anterior angles and open cavities after surgery (**Fig. 1.34**).

**Normal Otoscopic Appearance**

Characteristics of the tympanic membrane: The pars tensa is grayish-yellow. The cutis layer is often slightly injected. The surface is smooth and without any relieving features, apart from the handle of the malleus. The membrane is moderately translucent and is only transparent in scarred areas. A tympanic membrane showing the properties described above is described as *normal.* The mobility of the tympanic membrane can be assessed using a pneumatic oto scope (**Fig. 1.35**).

The tympanic membrane is moved back and forth with positive and negative pressure while it is in the field of vision. Atrophic parts flutter, and the movement of the pars tensa may be limited by scar tissue. In the presence of a perfora tion, the remnants of the tympanic membrane are com pletely immobile.

**Appearance of a Pathologic Tympanic Membrane** Injection of the vessels and inflammation are seen in otitis externa (occasionally), myringitis, and otitis media.

Hemorrhage is red if fresh, or brownish if old. Blood vesicles are seen in influenzal otitis, and the hemotympanum is dark blue.

Serous exudate: A fluid level can be seen, and there are air bubbles in the fluid. The tympanic membrane looks like oiled silk when there is a complete middle ear effusion. A blue tympanic membrane or blue drum is seen in advanced stages.

Retraction of the tympanic membrane as a result of decreased pressure in the middle ear: The short process of the malleus protrudes exter nally, and there is displacement of the manu brium of the malleus posteriorly and superiorly, causing an apparent shortening of the malleus handle. The triangular light reflexis fragmented, or disappears entirely.

Bulging due to the formation of exudate behind the tympanic membrane, at times with an irreg ular surface, which may be papillary, with an opaque surface.

Atrophy of the tympanic membrane with retrac tion pockets results from chronic inflammation and reduced pressure. The site of predilection is the posterosuperior quadrant.

Thickening of the tympanic membrane, as a re sult of degenerative changes or as the result of inflammation, produces a surface that is dark and lacking in luster.

*Methods of Investigation* **23**

**Fig. 1.33** Ear microscopy: 

variable magnification be

tween 6 and 12 , with a

focused light supply.

| HOPKINS |
| --- |

**Fig. 1.34** A rigid telescope with a straight or 30 angled view and a diameter of 2.7 4.0 mm is used for oto-endos copy. The external meatus should be stretched by pulling the auricle upward and backward, as in other otoscopy proce dures.

**Fig. 1.35** Pneumatic otoscope with loupe (Welch Allyn, Skaneateles Falls, New York, USA).

Scars of the tympanic membrane: These may be thickened areas, with or without calcium depos its or atrophic areas.

Tympanic membrane perforations: These may be either central or peripheral, mesotympanic or epitympanic. Central or mesotympanic de fects are the result of chronic mucosal inflam mation (see p. 67), whereas peripheral or epi tympanic perforations are usually associated with a cholesteatoma (p. 68).

**24 1 Ear**

| **a b**  4  5  30°  2  1  3 |
| --- |

**Fig. 1.36a, b** Radiographs in the Sch ller view. **1,** The exter nal and internal meatus super

imposed; , the head of the mandible; , the sigmoid sinus; , the border to the middle cra nial fossa; , the sinodural an gle.

Note: A tympanic membrane that has a surface with an opaque and dull appearance as a result of inflammatory infiltration of the pars tensa, with hyperemia, edema, formation of bullae, desquamation of the epidermal layer, and distortion of the characteristic appearance of the handle of the malleus is designated as abnormal or pathologic.

**Diagnostic Imaging**

The position in regional anatomy of the petrosal bone inside the skull base generates overlapping artifacts during radiographic examinations. Special radiographic images of the temporal bone, or of both sides for comparison, are therefore essential.

Conventional imaging of the petrosal bone using the Stenvers and Sch ller techniques has now to a large extent been replaced by computed tomogra phy (CT). In selected cases, however, the Sch ller radiographic view is still valuable for clinical evi dence.

**Conventional Radiography**

*Sch ller technique:* This provides information about the degree of mastoid pneumatization and demon strates the intercellular space and septal bone, the course of the sigmoid sinus, the tympanic roof (tegmen tympani), and the maxillary joint. These images are useful for diagnosing otitis media, mas toiditis, and fractures of the petrosal bone (**Fig. 1.36a, b**).

*Stenvers technique:* Demonstration of the inner auditory canal, and the width of the canal in partic ular. The horizontal and superior arches of the equi librium/vestibular organ, as well as the petrosal apex, are also well displayed. These images are useful in cases of acoustic neuroma, destructive processes in the internal ear, and transverse frac tures of the petrosal bone (**Fig. 1.37a** ).

**Computed Tomography**

High-resolution CT images of the petrosal bone in axial and coronal views, with slice thicknesses of 1 and 2 mm, have largely replaced conventional radiographic imaging in image-guided diagnosis. In addition to clear depiction of anatomical struc tures, CT images precisely show inflammatory changes such as soft-tissue enlargement (mucosal swelling, cholesteatoma, or tumor masses) and fluid retention, as well as osseous destruction or fractures (**Fig. 1.38a, b**). A new CT technique known as *multislice spiral CT* also allows three-dimensional interpretation of the middle and inner ear struc tures.

**Angiography**

The following supplementary *neuroradiologic in vestigations* are indicated for suspected vascular neoplasms or space-occupying lesions in the mid dle or posterior cranial fossa and cerebellopontine angle (CPA):

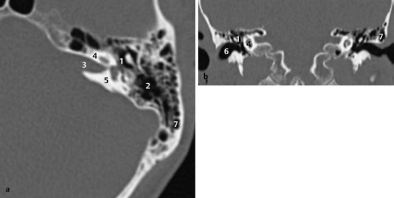
Carotid angiography.

Vertebral angiography.

*Methods of Investigation* **25**

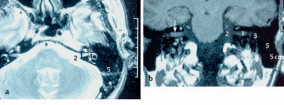
| 45°  1  2  35  10°  4  **a**  **b**  **c** |
| --- |

**Fig. 1.37a** Radiographs in the Stenver view. , Internal auditory meatus; **2,** vestibule, with the superior and horizontal semicircular canals; **3,** mandibular condyle; **4,** pneumatic system; , sigmoid sinus.



**Fig. 1.38a, b** Axial ( ) and coronal ( ) computed tomography scans of the temporal bone. , Tympanic cavity with ossicles; , antrum; , internal acoustic canal; , cochlea; , labyrinth; , external acoustic canal; , pneumatized mastoid cells.

**26 1 Ear**

**Fig. 1.39a, b** Axial ( ) and co 

ronal ( ) magnetic resonance

images of the temporal bone

area. , vestibulocochlear nerve

in the internal acoustic canal;

cerebellopontine angle (CPA);

, cochlea; , labyrinth;

, pneumatized mastoid. The

arrow in shows an intrameatal

vestibular schwannoma

(see p. 92).

**Magnetic Resonance Imaging**

Magnetic resonance imaging (MRI) is an optimal method for detecting inflammatory, traumatic, or neoplastic pathology in the temporal bone and skull base. It is routinely used with T1-weighted and T2- weighted spin-echo sequences, producing thin slice images, sometimes with contrast administra tion (gadolinium diethylenetriamine pentaacetic acid, Gd-DTPA). Very small vestibular neuromas in particular (acoustic neuromas) can be optimally visualized (**Fig. 1.39a, b**). The enhanced soft-tissue display is very helpful for assessing the ex tent of any neoplasms, complementing CT images.

Using high-resolution T2-weighted sequences such as the *constructive interference in steady state* (CISS) sequence a three-dimensional magnetic resonance sequence that displays cerebrospinal fluid spaces even details of the membranous lab yrinth and neuronal structures of the inner ear canal and cerebellopontine angle become visible. The *volume-rendering* technique can even produce three-dimensional images of the entire inner ear, as well as of the topological relationships in the entire temporal bone. In addition, it provides virtual endo scopic visualization of the inner ear region.

Views can be taken in all three planes without secondary reconstruction using a computer and without the need for special positioning of the pa tient. Pathologic formations (e. g., tumors, ischemic lesions) can be differentiated from normal struc tures using contrast administration (Gd-DTPA). Other specific MRI techniques include *functional MRI* (f-MRI), which makes localized perfusion changes in the brain visible, and *magnetic resonance angiography*, which can reveal intracranial tumors (glomus tumors) and vascular structures.

**Functional Assessment of the**

**Eustachian Tube**

Tests of tubal function are always necessary in all patients with middle ear hearing loss, particularly before an operation to improve hearing.

**Qualitative Assessment of Tubal Function**

**Valsalva test.** This test is used to demonstrate nor mal tubal patency without the need for any external aids. Failure of the test does not prove pathologic occlusion of the tube, but further functional tests may be required.

After taking a deep breath, the patient pinches his nose and closes his mouth in an attempt to blow air into his ears. Otoscopy shows bulging of the tympanic membrane, and auscultation reveals crackling.

Note: In patients with infection of the nose and naso pharynx, inflation of air involves a risk of transmitting infected secretions into the middle ear, causing tubo genic otitis media. In patients with an atrophic scar of the pars tensa, rupture of the tympanic membrane is also possible, especially during air insufflation or catheteriza tion of the tube.

**Toynbee test.** This test is used to confirm normal tubal air patency with a simple and safe method. During swallowing, pressure in the middle ear falls if the nose is closed off. This can be seen on otoscopy as a drawing-in of the tympanic membrane.

**Hearing Investigations**

**Testing Hearing without an Audiometer**

**Hearing Threshold for Whispered Voice and Conversational Speech**

Two-syllable words are articulated at a decreasing distance from the patient until the test words can be correctly repeated. The distance is recorded in meters. Alternatively, the examiner can say num bers or words at a fixed distance with decreasing loudness. When severe unilateral deafness is being assessed, and also when the hearing distance for conversational speech is being measured, it is nec essary to mask the contralateral ear. Each ear is tested separately, with the better ear being tested first. The contralateral ear canal is closed with a finger.

*Requirements:*

A sufficiently large, quiet room (6 m long). Good acoustic properties (no smooth walls with distorting echoes).

**Tuning Fork Tests**

A C fork with a frequency of 512 Hz is used.

**Weber test.** This test is based on binaural compar ison of bone conduction. The tuning fork is placed in the center of the skull at the hairline. A patient with normal hearing or with symmetrical hearing loss localizes the tone either in the center of the head or equally in both ears. A patient with unilateral con ductive hearing loss (middle ear) localizes the tone in the affected ear, whereas a patient with unilat eral inner ear deafness localizes the sound in the healthy ear.

*Theoretical explanations:*

In middle ear disorders, the mobility of the os sicular chain is reduced and it thus transmits less sound energy than it does in normal physiologic conditions (Mach s sound wastage theory).

Pathologic processes in the middle ear cause an increase in the mass of the sound conduction apparatus, so that increased forces are exerted at the oval window, due to inertia. This leads to greater stimulation of the inner ear (inertia theory) (**Fig. 1.40a** ).

**Rinne test.** This test is based on monaural compar ison of air conduction with bone conduction. If air conduction is better than bone conduction, Rinne

*Methods of Investigation* **27**

test is *positive.* This is the finding in normal hearing or sensorineural hearing loss (inner ear). If bone conduction is better than air conduction, Rinne test is *negative.* This is found in conductive or mid dle ear hearing loss.

The patient is asked whether the tuning fork placed in front of the ear is heard better than when it is placed behind the ear, on the mastoid process, without striking it again. If the patient cannot decide with certainty, the decay period of the tuning fork should be determined precisely for both air and bone conduction separately (**Fig. 1.41a** ).

**Gellé test.** This can be used to test the mobility of the ossicular chain in cases of otosclerosis (see p. 81) and fixation of the incus. The test has now been replaced by impedance audiometry (see p. 36) (**Fig. 1.42a, b**).

Note: Assessment of the hearing distance for whispered and conversational speech, along with tuning fork tests, provides valuable information about the site of a hearing disorder. These tests are still the basic diagnostic meth ods in otologic examinations (**Table 1.1**).

**Audiometry: Fundamental Physical and Acoustic Concepts**

See **Tables 1.2, 1.3, 1.4, 1.5, 1.6, 1.7**

**Pure-tone Audiometry**

An *audiometer* is an electric tone generator used to determine the hearing threshold for pure tones i. e., tones free of harmonics within a frequency range from 125 to 12 000 Hz.

The *hearing threshold* is measured for both air and bone conduction in decibel steps. The *normal hearing threshold is* indicated by a straight line at 0 dB. Hearing loss is measured in decibels relative to this threshold for all frequencies and is recorded on an audiogram (**Fig. 1.43**).

The decibel (dB) is a relative value that compares one sound pressure to another. The reference point in audiome try is the human hearing threshold of 1000 Hz. The sound pressure necessary to produce the subjective impression of hearing at a threshold of 1000 Hz is 20 Pa (2 10 bar) (see **Table 1.3**). This is the average value for young individ uals with normal hearing and is the reference point for the physical or absolute measurement of the hearing threshold

**28 1 Ear**

| r lr l **a b**  r l  **c**  **d** |
| --- |

**Fig. 1.40a** The Weber test. A vibrating tuning fork is placed on the midline of the skull.

Equal loudness perceived in both ears means sym metrical hearing.

Lateralization of sound to the affected ear (right) is present in cases of conduc tive hearing loss.

In cases of sensorineural hearing loss, the sound is lateralized to the better ear (left).

Correct orientation of the tuning fork.

in decibels (sound pressure level, SPL). The *relative hearing threshold* for pure tones is a simpler method of demonstrat ing and describing the hearing threshold. The reference point is no longer the absolute sound pressure, but the just-audible threshold of hearing, measured in dB (hearing level, HL). This makes it possible to use a coordinate system with a horizontal zero line. The *absolute hearing threshold* is curved in comparison with the relative hearing threshold. The reason for this is that greater sound pressure is needed at high and low tones to produce a similar sensation of sound near the threshold than for the central part of the frequency range around 1000 Hz (see **Fig. 1.44**).

A disorder of sound conduction can be identified

threshold for air and bone conduction, in the same way as with tuning fork tests.

**Relationship between Air Conduction and Bone Conduction**

Normal conduction of sound to the inner ear via the sound-conducting apparatus is defined as *air con duction* (conduction via earphones). Sound is also conducted via the bones of the skull to the inner ear, either via the middle ear *(osteotympanic* or *cranio tympanic bone conduction)* or by direct transmis sion via the labyrinthine capsule *(osteal* or *cranial bone conduction)* (conduction via a vibrator).

by assessing the difference between the hearing

*Methods of Investigation* **29**

| **a b c** |
| --- |

**Fig. 1.41a** The Rinne test. Air and bone conduction are compared in the same ear.

Rinne positive, normally hearing ear. Air conduction is perceived louder or longer than bone conduction in the test ear.

Rinne negative, conductive hearing loss. Bone conduction is perceived louder or longer than air conduction. Rinne positive, sensorineural hearing loss. Air conduction is perceived louder or longer than bone conduction, but the duration is shorter than in normal hearing.

| **a b** |
| --- |

**Fig. 1.42a, b** The Gellé test. A fixed ossicular chain causes conductive hearing loss.

Compression of the Politzer bag induces fluctuations of loudness in the normal ear.

The fluctuations are absent when the ossicular chain is im mobile.

| Key to audiogram symbols:  Frequency in kHz  Frequency in kHz  .125 .250 .5 1 2 3 4 6 8 10  .125 .250 .5 1 2 3 4 6 8 10  USA/International:  Right: Left:  -10  -10  Air conduction masked  0  0  Air conduction unmasked  10  10  Bone conduction unmasked  Hearing threshold (dB)  Hearing threshold (dB)  20  20  Bone conduction masked  30  30  UK:  40  40  Air conduction, masked if  50  50  necessary  60  60  Bone conduction unmasked  70  70  Bone conduction masked  80  80  Uncomfortable loudness level  90  90  100  100  110  110  120  120  130  130  Air conduction  Air conduction  Bone conduction Right ear  Bone conduction Left ear |
| --- |

**Fig. 1.43** A normal pure-tone audiogram.

**30 1 Ear**

**Table 1.1** Evaluating the results of clinical hearing tests

**Discrepancy between hearing**

**distance for whispered and**

**conversational speech**

**Weber test Rinne test**

Normal individual None Midline Positive

Conductive hearing loss Usually small Lateralized to the poorer-hearing ear in unilateral hearing loss

Negative or equivocal

Sensorineural hearing loss

Usually large Lateralized to the better-hearing ear in unilateral hearing loss

Positive

Degree of hearing loss Distance for hearing conversational speech:

Slight (> 4 m)

Medium (< 4 m, > 1 m)

Severe (< 1 25 cm)

Total deafness < 25 cm

**Table 1.2** Properties of sound

Sound A molecular vibration of an elastic me dium propagated as a waveform (in air,

water, bone, and all other media)

Speed of sound 340 m/s in air, 1400 m/s in water

**Table 1.4** Hearing range and decibel (dB) scale **Sound source Intensity ratio dB** Jet engine 1 : 10~~13~~ 130 Riveting hammer 1 : 10~~12~~ 120

Sound pressure (Pa)

This is the predominant change of pres sure in a sound field. It is a function of time at any particular point and is ex pressed in pascal units

Drilling machine 1 : 1011 110 Printing machine 1 : 1010 100 Weaving machine 1 : 10 90

Mass unit The old-fashioned unit was the microbar b), dynes/cm . The SI unit for absolute

sound pressure is the pascal (Pa), equiv

alent to newtons per m (1 Pa = 1 N/m

= 10 b)

**Table 1.3** Hearing or dynamic range and sound pressure level

Machine workshop 1 : 10 80 Street traffic 1 : 10 70 Normal speech 1 : 10 60 Soft radio music 1 : 10 50 Soft speech 1 : 10 40 Whispering 1 : 10 30

Hearing range (0 dB)

(120 dB)

Sound pressure level (SPL)

The lower limit i. e., the hearing thresh old at 1000 Hz is 20 Pa

The upper limit or pain threshold is 20 Pa

The unit is the decibel, a logarithmic unit calculated as follows:

Lp = 20log10 ) dB,

where p ist sound pressure being mea sured and p ist reference pressure, de ned as 20 Pa.

Quiet living room 1 : 10 20 Rustling of leaves 1 : 10 10 Hearing threshold 1 : 10

**Table 1.5** Sound intensity, sound volume, and loudness

*Methods of Investigation* **31**

**Table 1.6** Tone, timbre, noise

Scale of sound intensity

Volume of

loudness level

This is a physically defined decibel scale based on the square amplitude value of tones, rather than on a subjective assess ment of the loudness of the tone

Measured in phons, a logarithmic unit. The tone is compared subjectively with a refer ence sound of 1000 Hz. The sound pres sure level (SPL) of the reference tone is adjusted so that the test tone and refer ence tone sound equally loud. The result in decibels SPL is expressed in phons. A sound with a loudness level of 50 phon produces the same sensation of loudness as a refer ence tone at 1000 Hz at an SPL of 50 dB

Tone A pure sinusoidal vibration in the audible range characterized by frequency

Frequency Vibrations per second in hertz

Timbre A sound contains overtones in addition to the basic tone, which determine the sub

jective color of the sound

Noise Sound whose pressure in the sound field is not a periodic function of time

White noise Consists of equal components of all the audible frequencies from 18 to 20 000 Hz

Loud noise May be distressing or cause actual damage

Loudness The unit is the sone, which is a linear scale depending on subjective comparison with

a measured value. The loudness of a test

**Table 1.7** Impedance

Isophon curves

tone is compared with that of a reference tone of 1000 Hz and 40 dB SPL

See **Fig. 1.40**. Consist of curves of the same loudness level measured in phons, but at di erent frequencies (in Hz) and SPL (dB)

Acoustic impedance

Resistance to the flow of sound pressure waves through a medium, proportional to:

The mass of the vibrating system Its resistance

Its elasticity

Hearing range Between the hearing threshold at 4 phons and the threshold of pain at 130 phons (see

**Fig. 1.40**

Note: The audiometric characteristic of conductive or middle ear hearing loss is that the threshold for air con duction is poorer than that for bone conduction, produc ing an air bone gap.

*Conductive hearing loss*results from an increase in impedance **Fig. 1.45a** ). If the elastic recoil due to air in the middle ear and mastoid process increases, mobility in the middle and low tones decreases at constant mass and tension. The resonance point of the middle ear is displaced to the upper frequencies. Conductive hearing loss is characterized by greater loss of hearing for air conduction in the lower fre quencies as seen, for example, in ossification of the stapes annulus in *otosclerosis* **Fig. 1.45c**;see also **Fig. 1.101**). The conduction system is increasingly damped by the increase in

Resistance The frictional resistance in the joints, liga ments, and muscles of the sound-conduct

ing apparatus

Reactance An imaginary component determined by the stiffness and mass of the system

Compliance The flexibility of the tympanic membrane

mass and tension, and the resonance point of the middle ear is thus displaced into the lower tones. The hearing loss that results that is greater for air conduction in the middle and higher tones e. g., as occurs with glue ear exudate in the middle ear (**Fig. 1.45b**) and impacted wax.

Conductive hearing loss independent of frequency is caused by simultaneous elastic stiffening and dampening of the sound conduction apparatus. This may occur in ad vanced *otosclerosis,* in *middle ear cholesteatoma* with destruc tion of the ossicular chain, in *tympanosclerosis,* and in *con genital anomalies.* A flat air conduction curve is found in such cases.

**32 1 Ear**

| 2.102  (Phon)  140  Subjective sound level (Phon)  ~~130~~  Sound pressure level (dB SPL)  2.101  120  Sound pressure (Pa)  2  100  ~~100~~  Loudness  2.10-1  ~~80~~  80  2.10-2  60  ~~60~~  2.10-3  40  ~~40~~  2.10-4  20  ~~20~~  2.10-5  4  0  20 31.5 63 250 1000 4000 16000  Phon = dB  Pain threshold  1000 Hz  Discomfort threshold  Normal hearing threshold |
| --- |

**Fig. 1.44** Human auditory field. The sound pressure (in Pa), the sound pressure level (in dB), and the loudness (in phon) are shown together in a coordinate system with the spectrum of human hearing in hertz. The abscissa shows frequencies, the ordinate decibels and phon. Isophons are curves of equal loudness. The curves for decibels and phon coincide only at 1000 Hz and deviate from each other above and below this frequency.

Note: The bone conduction threshold curve is an ex pression of the function of the inner ear and, to a limited extent, of its central connections.

This rule applies with a few unimportant excep tions e. g., bony closure of one or both windows. The audiometric characteristic of all forms of sensorineural hearing loss (inner ear and retro cochlear hearing loss) is that the thresholds for air and bone conduction coincide (**Fig. 1.46a** ). Sup plementary *suprathreshold tests* have to be per formed to differentiate inner ear hearing loss from retrocochlear hearing loss.

**Demonstration of Recruitment**

Patients with *inner ear hearing loss* showing *recruit ment* often have difficulty in hearing relatively soft tones. In contrast, they hear loud conversational speech as well as individuals with normal hearing. They find excessive loudness upsetting due to dis tortion and painful sensations, as the *threshold of discomfort* is exceeded. In inner ear deafness, re cruitment occurs in the frequency range of the damaged hair cells, which require a considerably higher sound pressure in comparison with the nor

mal hair cells to produce a response. The resulting reduction of the dynamic hearing range has ex tremely deleterious effects as far as hearing of speech is concerned (see **Fig. 1.44** and pp. 108 110). The following tests are used:

**Fowler test.** *Principle:* This test is based on a subjective comparison of loudness between the right and left ears. A tone of the same frequency and loudness is presented alter nately. A recruitment phenomenon is present if a difference in the hearing threshold between the two sides disappears as the loudness of the test tone increases (**Fig. 1.47**).

Note: Demonstration of the recruitment phenomenon is currently accepted as indicating an inner ear or hair cell lesion, whereas this phenomenon is usually absent in retrocochlear neural hearing loss due to vestibular schwannoma, for example.

**Tone intensity difference threshold (L scher test).** *Principle:* The threshold of intensity difference in decibels at the same distance above the hearing threshold is smaller in an ear affected with recruitment than in the normal ear.

**Short increment sensitivity index (SISI) test.** *Princi ple:* A test tone is produced 20 dB above the patient s thresh old and is increased by 1 dB every 5 seconds with a duration of 0.2 s. Negative results are obtained in retrocochlear le sions with pathologic fatigue. The score is greater than 80 % in patients with cochlear hearing loss showing recruitment.

**Demonstration of Pathologic Fatigue** *Pathologic auditory fatigue* is a sign of a retroco chlear hearing loss. It can be demonstrated using the technically simple *tone decay test* and the *Bé késy test.* These two methods have now been largely replaced by measurement of auditory evoked po tentials (AEPs; see p. 37), which allows objective testing of auditory functions and considerably more accurate diagnosis of retrocochlear hearing disor ders.

**Speech Audiometry**

*Speech audiometry* is an integral part of audiometric methods of investigation. The ability to hear and understand speech is more important in human communication than the ability to hear pure tones. Speech audiometry therefore has both diagnostic and therapeutic significance. To understand the re

| Frequency in kHz  Frequency in kHz  .125 .250 .5 1 2 3 4 6 8 10  .125 .250 .5 1 2 3 4 6 8 10  -10  -10  0  0  10  10  Hearing threshold (dB)  Hearing threshold (dB)  20  20  30  30  40  40  50  50  60  60  70  70  80  80  90  90  100  100  110  110  120  120  130  130  **a** Perforation of tympanic membrane **b** Middle ear effusion  Frequency in kHz  Frequency in kHz  .125 .250 .5 1 2 3 4 6 8 10  .125 .250 .5 1 2 3 4 6 8 10  -10  -10  0  0  10  10  Hearing threshold (dB)  Hearing threshold (dB)  20  20  30  30  40  40  50  50  60  60  70  70  80  80  90  90  100  100  110  110  120  120  130  130  **c** Fixation of stapes in otosclerosis **d** Mixed hearing loss  Air conduction  Bone conduction |
| --- |

*Methods of Investigation* **33**

**Fig. 1.45a** Audiograms showing conductive hearing loss (right ear).

sults of speech audiometry, it is necessary to know the frequencies contained in speech. The funda mental vocal frequencies for men (125 Hz) and women (250 Hz) are shown in a tone threshold audiogram in **Fig. 1.48**

The loudness of speech is perceived as an acous tic image, the frequencies of which extend from 100 to 8000 Hz. *Hearing loss for speech* is assessed using two-syllable test words, and maximum discrimina tion is also measured using one-syllable test words **Fig. 1.49a**).

Speech audiometry is not performed in the same way as testing of the vocal speech (see p. 127) i. e., with an increasing distance between the patient and the sound source but rather by varying the loudness as measured in decibels, i. e., with a

speech sound level above 20 Pa (see p. 30, **Table 1.2**).

The speech or test material is recorded on a disk and is presented to the patient either using earphones or in a free field using a loudspeaker with varying loudness levels. The percentage of numbers, words, or sentences understood correctly at each loudness level is then assessed.

The dependence of speech comprehension on the loud ness level is tested using speech audiometry. In the stand ardized test (e. g., the *Freiburg speech test*), multisyllable numbers are first used. This can provide a rapid rough estimate of the extent of hearing loss.

An individual with normal hearing understands 50 % of numbers presented at 18.5 dB. This normal value forms the basis for assessing hearing loss for numbers. The patient ability to comprehend monosyllabic words is also tested.

**34 1 Ear**

| Frequency in kHz  Frequency in kHz  .125 .250 .5 1 2 3 4 6 8 10  .125 .250 .5 1 2 3 4 6 8 10  -10  -10  0  0  10  10  Hearing threshold (dB)  Hearing threshold (dB)  20  20  30  30  40  40  50  50  60  60  70  70  80  80  90  90  100  100  110  110  120  120  130  130  **a** Low-frequency hearing loss **b** High-tone hearing loss Frequency in kHz  Frequency in kHz  .125 .250 .5 1 2 3 4 6 8 10  .125 .250 .5 1 2 3 4 6 8 10  -10  -10  0  0  10  10  Hearing threshold (dB)  Hearing threshold (dB)  20  20  30  30  40  40  50  50  60  60  70  70  80  80  90  90  100  100  110  110  120  120  130  130  **c** High-frequency notch **d** Pancochlear hearing lossAir conduction Bone conduction |
| --- |

**Fig. 1.46a** Audiograms showing sensorineural hearing loss.

= no response

These words are considerably more difficult to understand than multisyllabic numbers. The purpose of the monosyl lable test is to assess percentage comprehension and ulti mately to achieve 100 % comprehension values, if possible, by increasing the loudness level. Normal individuals hear 100 % of monosyllables at 65 dB, and in favorable conditions at 50 dB, whereas 100 % speech comprehension cannot be achieved even in normal individuals at a sound pressure level of less than 50 dB.

Speech audiometry allows quantitative meas urement of hearing. The speech audiogram indi cates the percentage of syllables, words, or senten ces that the individual has heard correctly in each test series. The result of a speech audiogram de pends not only on hearing, but also on higher cog nitive functions such as memory, language compre hension, and motor speech. Other factors that in fluence the results include whether the patient

mother tongue is being used and the patient s vo cabulary range.

**Comparison of pure-tone and speech audiograms.** Discrepancies between the results of pure-tone and speech audiometry are mainly found in retroco chlear hearing disorders. In such cases, hearing for speech is considerably worse than hearing for pure tones. The pathophysiologic basis for this is de scribed on p. 17.

Diagnosis of *central hearing disorders* is based on tests of the central understanding of speech. The classic methods of testing hearing fail in such cases due to the phenomenon of *redundancy.* This is the safety margin within the auditory pathways, which can transmit and analyze billions of information units, whereas only 100 are necessary for recogniz-

ing and decoding acoustic information. A disorder of the central summation and integration capacity can only be demonstrated with difficulty e. g., by dis torting speech by filtering out high frequencies and inserting periodic interruptions of the speech signal, or with binaural application of garbled test words, reducing the information content of normal speech to a minimum (Feldmann dichotic speech test).

Note: Speech audiometry is indispensable for: Assessing residual hearing for speech. This makes it possible to predict the probable benefit to be ex pected from a hearing aid. The loss of discrimination and the threshold of discomfort can be measured. Assessing the need for hearing aids and surgery to improve hearing.

Investigating central hearing loss. This allows assess ment of the integrative performance of the auditory centers.

Assessment, for insurance purposes, of a loss of hear ing for speech leading to a loss of earning capacity.

**Objective Hearing Tests**

Behavioral, pure-tone audiometry is based on a subjective response from the patient. In contrast, objective audiometry makes it possible to carry out testing without eliciting a patient response. This method uses tests based on involuntary physiologic reactions and objective parameters. These objec tive responses support the interpretation of pure tone audiometry and are very important for audio metric diagnosis in infants, small children, and pa tients with mental and cognitive impairment.

Three main methods are used in objective audio metry:

Measurement of changes in the acoustic impe dance of the tympanic membrane*: impedance audiometry.*

Measurement of acoustically evoked bioelectric responses of the cochlea, vestibulocochlear nerve and tract, or cerebral cortex: *auditory evoked potentials (AEPs).*

Measurement of spontaneous or acoustically evoked vibrations of the cochlea: *otoacoustic emissions (OAEs).*

*Methods of Investigation* **35**

| Loudness dB  100  80  60  40  20  Right  0  20 40 60 80 100  Left Loudness dB |
| --- |

**Fig. 1.47** Fowler s loudness balance test in a patient with unilateral left-sided sensorineural hearing loss, with a 40-dB loss at 1 kHz.

| Frequency (kHz)  0.125 0.25 0.5 1 2 3 4 6 8 10  0  10  20  th  f  30  ~~m~~ sh s  n  o u  40  u o ~~a e i~~ i ~~e~~ ~~a~~  SF  50  Hearing loss (dB HL)  60  70  80  90  100  110 |
| --- |

**Fig. 1.48** The speech field. The fundamental voice fre quency is 125 Hz in men and 250 Hz in women. Vowels are formed between 500 Hz and 4000 Hz and are spoken 10 20 dB louder than consonants in normal conversational speech. Several consonants lie in a higher frequency range (s, t) and therefore cannot be perceived by patients with high-frequency deafness; as in bed,” “ as in bar. Dark green area = region of the first formant;medium green area = region of the second formant;light green area = region of the speaker s formant (SF);red area = resonance of the nasal tract.

**36 1 Ear**

| Comprehension in %  Comprehension in %  10  10  30 50 70 90  30 50 70 90  0  0  10  10  0  0  20  20  ~~1~~  ~~1~~  Speech sound level (dB SPL)  Speech sound level (dB SPL)  10  10  30  30  ~~2~~  ~~2~~  20  20  40  40  30  30  50  50  40  40  60  60  50  50  70  70  ~~3~~  60  60  80  80  ~~4~~  90  90  70  70  ~~H~~e~~a~~r~~in~~~~g~~ ~~thresho~~l~~d~~ ~~(dB~~)  ~~H~~e~~a~~r~~in~~~~g~~ ~~thresho~~l~~d~~ ~~(dB~~)  100  100  110  110  120  120  80 60 40 20 0  80 60 40 20 0  100  100  **a**  Discrimination loss in %  **b**  Discrimination loss in %  Comprehension in %  10  30 50 70 90  0  10  0  20  ~~1~~  Speech sound level (dB SPL)  10  30  ~~2~~  20  40  30  50  40  60  ~~5~~  50  70  ~~60~~  80  90  70  6  ~~H~~e~~a~~r~~in~~~~g~~ ~~thresho~~l~~d~~ ~~(dB~~)  100  110  120  80 60 40 20 0  100  **c**  Discrimination loss in % |
| --- |

**Fig. 1.49a** Speech audiome try is carried out using uniform test content consisting of multi syllabic numbers and monosyl labic words.

An individual with normal hearing understands 50 % of numbers heard at 18.5 dB and 100 % of those heard at 30 dB ( ). For monosyllabic test words ( ), intelligibility is 50 % at 30 dB and 100 % at 50 dB.

In patients with conductive hearing loss, a parallel shift to ward higher sound levels occurs in the performance intensity function ( ), but nearly 100 % comprehension can still be achieved at sufficiently high lev els ( ).

Sensorineural hearing loss leads to a flattening of the perfor mance intensity function for monosyllabic words ( ). Loss of intelligibility and a decline in speech recognition at higher sound levels are signs of abnor mal speech processing, such as that caused by cochlear damage or neural disturbances ( ).

**Impedance Audiometry**

This technique is part of the functional diagnosis of the sound conduction apparatus. It includes the following two investigation methods:

*Tympanometry:* This involves recording the im pedance (see p. 14) or indirect measurement of pressure in the middle ear, when the tympanic membrane is intact, by means of pressure in the external meatus. This is an indirect test of tubal function.

*Measurement ofthe acoustic reflex:* The change in

***Technique***

The external auditory meatus is closed by an air tight plug, through which three tubes pass. One tube carries the test tone; the second is connected to the pressure regulator, which allows positive or negative pressure ( 400 mmH O) to be produced in the external auditory meatus. A microphone is connected to the third tube, allowing measurement of the sound pressure of the test tone reflected from the tympanic membrane as the impedance changes **Fig. 1.50**).

impedance caused by the acoustic stapedial re flexis measured.

| Probe  Reflex  tone  tone  “ipsi”  220 Hz  Loudspeaker  Microphone  Air pump |
| --- |

*Methods of Investigation* **37**

**Fig. 1.50** Tympanometry. Tubes passing through an air tight plug transmit the test tone and the reflected tone. The flexibility of the tympanic membrane, compliance, is cal culated from the measured sound level. Pressure-depen dent displacement of the tym panic membrane is regulated by an air pump.

**Tympanometry.** Normally, there is no pressure dif ferential between the two sides of the tympanic membrane, so that the acoustic resistance of the tympanic membrane is minimal. Recording the im pedance of the tympanic membrane during a change in pressure in the external auditory meatus allows the pressure difference on the two sides of the tympanic membrane to be determined by measuring its compliance. The greater the pressure differential, the greater is the impedance of the tympanic membrane. Recording the impedance at pressures from 300 mmH O to + 300 mmH O pro duces a curve with a peak at zero for a normally mobile tympanic membrane. This represents the maximum flexibility i. e., compliance of the tym panic membrane, and thus minimal impedance. The apexof this curve is lower if the tympanic membrane is stiffened by scar tissue or damped by exudate in the middle ear. It becomes higher with increasing compliance due to atrophic scars of the pars tensa (**Fig. 1.51a** ).

**Stapedial reflex.** The *principle* of this test is that a sound stimulus greater than 70 dB above the threshold induces a reflexcontraction of the stape dius muscle. This causes a change of impedance at the tympanic membrane, which can be recorded graphically. The effect is absent when the tympanic membrane is immobile, when the ossicular chain is disrupted, and when the stapes is fixed in the oval window by otosclerosis. In simulated deafness, this reflexis activated by loudness approaching the norm. In this case, simulation can be assumed.

The stapedial reflexis an acousticofacial reflex. The afferent limb is the vestibulocochlear nerve and parts of the central auditory pathway up to the auditory centers. The efferent limb is formed by the connections between the auditory centers and the facial nucleus, and finally by the facial nerve. Measurement of the stapedial reflexis therefore very useful in topical diagnosis of facial paralysis.

Testing the threshold for the stapedial reflexis of considerable diagnostic importance for assessing the following hearing disorders: otosclerosis, re cruitment (Metz recruitment is reduction of the difference between an elevated hearing threshold and the threshold for the stapedial reflex, with in creasing hearing loss for high tones), retrocochlear deafness, and brain stem lesions.

The stapedial reflexis absent in:

Retrocochlear sensorineural deafness as a result of auditory fatigue i. e., in vestibular schwan noma.

Otosclerosis and other middle ear diseases. Facial nerve damage proximal to the point at which the stapedius muscle is innervated. Brain stem lesions with damage to the central reflexarc.

**Auditory Evoked Potentials (AEPs)**

The patient is repeatedly exposed to an acoustic stimulus, either regularly or irregularly, and an electroencephalogram (EEG) is used to assess whether there is any change in brain activity. The AEPs are recorded from the scalp using needle or surface electrodes. As the amplitudes of the AEPs

**38 1 Ear**

| daPa  -300 -200 -100 0 100 200 300 10  Normal  Compliance  5  **a**  0  Compliance  Compliance  **increased**  **b**  **Negative**  Compliance  **pressure peak**  **c**  **Flat**  Compliance  Compliance  **decreased**  **d** |
| --- |

**Fig. 1.51a** Summary of the four most important results of a tympanogram. The curve shows the compliance of the tympanic membrane to changes in pressure in the external canal.

Normal: the apex of the curve (daPa) lies close to 0 on the pressure scale when the pressures in the meatus and in the middle ear are equal.

Increased compliance: the apex of the curve will be abnor mally high if the tympanic membrane is extremely mobile. This situation may occur with atrophic scars of the pars tensa or interruption of the ossicular chain.

Negative peak pressure: the apex of the curve is displaced below 100 daPa due to reduced pressure in the middle ear. Flat tympanogram with no compliance peak. This is seen when the tympanic membrane is dampened due to the middle ear effusion. This type of curve also occurs if the tympanic membrane is perforated, but the equivalent (or ear) canal volume (ECV) will then be high.

are very small relative to the total activity of the brain, averaging the potentials is necessary (see **Fig. 1.49**). Averaging means that the individual re sponse, which is concealed on the EEG by the

noise of brain activity, can be distinguished by mathematical analysis of numerous evoked indi vidual potentials. The intermittent acoustic stimu lus produces a uniform potential during a time interval that always occurs at the same time and can be amplified by repetitive summation of the EEG segment.

The properties and shape of AEPs depend partly on the time at which they occur after presentation of the acoustic stimulus, or their latency (in milli seconds). Several types of AEP can be distinguished on the basis of different sites of origin and latency.

***Classification***

**Electrocochleography (ECochG).** This measures the potentials arising in the cochlea and vestibuloco chlear nerve. These potentials occur 3 ms after the stimulus is presented. The two most useful diagnostic parameters are *cochlear microphonics* (CM) and the *action potential* of the vestibuloco chlear nerve (PI).

**Auditory brain stem response (ABR) audiometry (brain stem evoked response audiometry, BERA).** This measures the potentials arising in the vestibu locochlear nerve and brain stem structures, with a latency of up to 10 ms. The latency of individual potentials, particularly between potential peaks I and V, is very important for recognizing retroco chlear hearing disorders (**Fig. 1.52**).

**Auditory middle latency potential (AMLP) audio metry.** This measures potentials with a latency of 10 100 ms that originate in the thalamus and pri mary auditory cortex.

**Cortical evoked potentials (CEPs).** This measures potentials with a latency of 100 1000 ms, which express generalized higher-order cortical function.

Measurement of the auditory brain stem re sponse (ABR) and electrocochleography (ECochG) are two of the most important diagnostic methods for accurate differentiation between cochlear and retrocochlear deafness. The latter is due to space occupying formations in the CPA (e. g., vestibular schwannoma), a tumor of the posterior cranial fossa, or multiple sclerosis. AEP is also very useful for investigating deafness in infants and young chil-

dren. It can also be used to assess residual function of the central nervous system in patients with se vere head injuries, coma, or other conditions marked by a complete loss of consciousness. It does not, however, replace pure-tone audiometry or tympanometry (including the stapedial reflex), which still form the basis for audiometric evalua tions. ABR is also tested intraoperatively in order to monitor hearing.

**Otoacoustic Emissions (OAEs)**

Otoacoustic emissions are sound signals emitted from the inner ear in response to acoustic stimula tion. The signals are vibrations produced by the biomechanical cochlear amplifier (see p. 16). They occur spontaneously or in response to an acoustic stimulus and are transmitted in retrograde fashion across the ossicles to the tympanic membrane. The membrane acts like a loudspeaker membrane, so that emitted vibrations can be measured as sound waves in the external ear canal. These active co chlear vibrations can be detected by a sensitive microphone.

Otoacoustic emissions are clinically important, as they reflect the functional integrity of the co chlea. OAE detection depends on normal middle ear function for good transmission to the tympanic membrane.

***Classification***

**Spontaneous OAEs (SOAEs).** Vibrations can arise spontaneously in the cochlea without any external stimulus. They are detectable as low-level, contin uous tones in 50 % of individuals with normal hearing.

**Transient evoked otoacoustic emissions (TEOAEs).** Emissions are detected in response to an acoustic stimulus (click) in individuals with normal cochlear function. An averaging technique is used (as in ABR; see p. 38). This measurement is used as an objective audiometric testing method (**Fig. 1.53a**). TEOAEs occur in normal hearing and confirm cochlear in tegrity; they are absent in patients with middle ear disease or cochlear hearing loss with a threshold increase of 30 dB. The amplitude in infants with normal hearing is usually higher than in adults.

**Distortion product otoacoustic emissions (DPOAEs).** Acoustic distortions in the cochlear am plifier can be detected by stimulation with two

*Methods of Investigation* **39**

| Amplitude (ΠV)  IV  V  III  VI VII  0.5  I  0  II  -0.5  0 1 2 3 4 5 6 7 8 9 10 11  Time after acoustic stimulus (ms) |
| --- |

**Fig. 1.52** Auditory brain stem response (ABR). The typical waveform consists of five or seven waves (I VII), which reflect the acoustically induced activity of the anatomical structures of the auditory system (see **Fig. 1.20**).

continuous tones that have different, but adjacent, frequencies. DPOAEs are intimately linked to outer hair-cell function. This is another frequently used objective audiometric testing method (**Fig. 1.53b**).

The most important application of otoacoustic emissions is for screening cochlear function in new borns, infants and small children. DPOAEs can be used to detect early discrete lesions of the outer hair cells, and they provide an important noninva sive screening method for cochlear impairment that can even be used without sedation or general anesthesia. OAEs can also be used to investigate nonorganic hearing loss, to objectify audiometric findings in adults, and to assess cochlear function in risk groups (ototoxic medication).

Note: In the absence of OAEs, additional audiologic tests such as auditory evoked potentials and pure-tone audiometry should be used.

**40 1 Ear**

| Loudspeaker  Loudspeaker 1  Loudspeaker 2  Microphone  Microphone  dB  Stimulus Spectrum  15  Sound pressure level  (dB SPL)  f1f2  0  60  40  -15  20  mPa TEOAE  (2f1-f2)  1 2 3 4 5  0  0.5  Pressure  (kHz)  -20  -40  0  2 2.5 3 3.5 4 4.5 5  -0.5  Frequency (kHz)  0 5 10 15 20  **a b**  Time (ms) |
| --- |

**Fig. 1.53a, b a** The system for measuring transient evoked otoacoustic emissions (TEOAE). A measuring probe with a microphone and loudspeaker is placed in the external ear canal. A click impulse is induced by the loudspeaker, and evoked emissions from the cochlea are recorded by the miniature microphone.

**Hearing Tests in Infants and Young Children**

Note: Every child who does not respond normally to sound stimuli soon after birth and at the latest after the first 6 months must undergo otologic examination.

Since even a completely deaf child passes through a period of crying and babbling, serious hearing loss only begins to be suspected when speech does not develop. Most children with hearing disorders are therefore presented to the general practitioner or otologist between the first and third years of life. As hearing is not an obvious condition in the newborn, it needs to be detected using screening, which is indicated in particular in the following cases:

The system for measuring distortion products of otoacous tic emissions (DPOAE). The cochlea is stimulated with two tones (f and f ). The sound pressure changes as a response in the external ear canal are recorded by the microphone along with the primary tones. The curve represents the frequency spectrum of the microphone signal.

Necessary treatment for more than 48 hours in an intensive-care unit.

Positive family history of hearing impairment. Manifest craniofacial anomalies.

*Universal screening*: Every newborn should be screened on the second or third day after birth during the second routine examination. Eighty per cent of all hearing problems can be detected using this method. The organization required for this method of screening depends on the local health care system.

*Additional screening* should be performed at rou tine pediatric visits or in a preschool medical ex amination.

**Tests**

**Otoacoustic emissions (OAEs).** If OAEs are present, the peripheral hearing is satisfactory, but this does not exclude a hearing disorder. The degree of any hearing loss cannot be determined.

*Methods of Investigation* **41**

**Table 1.8** Checklist for suspected congenital or early ac quired hearing loss

Family history Hearing and speech disorders, psychiatric and neurologic diseases, congenital

anomalies

**Auditory brain stem response (ABR).** If an ABR is not elicited, severe hearing loss is present. The hearing threshold can be determined when audi tory evoked potentials are measurable.

**Pediatric audiology with behavioral tests.** Subjec tive responses in pediatric audiometric testing are important methods and can be performed at virtu ally any age, but should be age-appropriate. The reliability of the test results is variable.

History of pregnancy

Perinatal history

Postnatal history

Virus infection with rubella, measles, in fluenza, herpes zoster, coxsackievirus, or *Toxoplasma;* drugs such as thalidomide or aminoglycosides;diseases such as diabe tes or neuropathy;or vaccination

Forceps or other mechanical damage, as phyxia, prematurity, kernicterus

Infectious disease, vaccination reaction, diseases of the central nervous system, trauma to the skull, intoxication, and drugs

**Test Methods**

**Reflex audiometry.** Nonspecific responses to audi tory stimuli, such as sucking responses, motor re sponses (Moro reflex, acousticopalpebral reflex) or breathing responses, can be elicited in normal in fants from birth on. The reflexes can be stimulated only by a loud noise (nearly 80 dB).

**Response audiometry.** By the second half of the first year of life, acoustic stimuli evoke typical re sponse patterns. A normally hearing infant turns his or her head toward the sound source that is out of the range of vision.

**Distraction test.** A tester attracts the child s atten tion with a toy, and the examiner presents an acous tic stimulus invisible to the child and observes the reaction.

*Visual reinforcement audiometry (VRA):* An acoustic stim ulus is combined with the activation of a moving toy. After conditioning, the child moves toward the toy when it hears the acoustic stimulus.

**Play audiometry.** As a variation of pure-tone audio metry, tasks and responses to tone testing are in corporated into a play setting (e. g., while playing, the child has to react when an acoustic stimulus is presented).

**Pediatric speech audiometry.** Children aged 3 years can be examined using audiometric speech tests specially designed for children (e. g., the Pe diatric Speech Intelligibility Test).

Hearing Reaction to noise and speech, directional hearing, the time when the hearing disor

der began, and the progress of the symp

toms

Speech Age at which the first sounds, words, and sentences were uttered

Note: The sense of hearing is a vitally important factor for acquiring speech. It is therefore essential for hearing loss in a child to be recognized and treated. The earlier the treatment is instituted, the more successful it is. Treatment should be started in the second half of the first year (**Table 1.8**;see also **Table 1.25**).

**Vestibular Function Tests**

Investigations of the vestibular system comprise: 1. Case history and analysis of symptoms. 2. Testing of the vestibulospinal reflexes. 3. Testing for spontaneous and provoked nystag mus.

4. Experimental testing of the vestibular and opto kinetic systems.

**Case History**

The subjective feeling of dizziness is generally re garded as being an expression of a disturbed neuro nal discharge pattern in the cortical projection

**42 1 Ear**

**Fig. 1.54** The Unter 

berger stepping test. The

patient is asked to walk on

the spot with the eyes

closed.

**Fig. 1.55** The patient 

position for static posi

tional tests. A spontaneous

deviation reaction and

spontaneous tone reaction

in the arm are observed

while the patient is sitting

on a chair.

areas. A thorough case history is required in order to achieve a structured analysis, allowing differen tial-diagnostic classification of:

Peripheral vestibular dizziness.

Central vestibular dizziness.

Nonvestibular dizziness.

The case history should include questions about previous illnesses, medications, and noxae. Questions regarding the type of subjectively perceived dizziness, as well as its dura tion and intensity, are important. Dizziness-causing factors and secondary symptoms are also important details to clarify.

**Vestibulospinal Reflexes**

In peripheral vestibular lesions, the body s center of

labyrinthine lesion is located. In central disturban ces of balance, the pattern of unsteadiness of gait and the direction of falling are irregular. Body sways can also be registered on an electronic scale (pos turography).

**Romberg test.** The patient is asked to stand with the feet together (touching each other) and to close the eyes. A check is made to see whether there is then any unsteadiness or a tendency to fall.

**Blindfold gait and walking a straight line.** Only gross abnormalities of gait are diagnostically im portant. The patient deviates to the same side as in the Romberg test.

**Unterberger stepping test (Fig. 1.54).** *Stepping on the spot with the eyes closed:* patients with periph eral disorders show rotation of the body axis to the side of the labyrinthine lesion; in central disorders, the deviation is irregular. Only deviations of more than 40 are of diagnostic significance.

***Static positional tests***

(see also p. 45)

**Spontaneous deviation reaction, past pointing.** Parallel displacement of *both* arms (with arms in the supine position) occurs in accordance with the vestibulospinal reflexes.

**Spontaneous tone reaction in the arms.** The arm on the side of the cerebellar lesion sinks as a result of loss of tone of the muscles (**Fig. 1.55**).

**Finger nose pointing test.** The indexfinger of the outstretched arm is brought to the tip of the nose with the eyes closed. Ataxia and disorders of coor dination (overshooting) indicate an ipsilateral cer ebellar lesion or a disorder of positional sense and deep sensation.

**Spontaneous and Provoked Nystagmus**

**Nystagmus.** This is a conjugated, coordinated eye movement around a specific axis; the movement consists of rhythmically alternating slow- and fast beating phases. The direction of the fast component of the nystagmus determines the laterality of the nystagmus.

gravity is usually displaced to the side on which the

**Tests**

**Observation with and without Frenzel s glasses.** This is used for the diagnosis of a *spontaneous nys tagmus.* The patient is examined in a darkened room with + 15-diopter lenses that almost com pletely suppress optical fixation, so that the visual fixation suppression of the vestibular nystagmus is eliminated (**Fig. 1.56**).

Direct gaze, with and without fixation, is used to recognize *fixation nystagmus.* Lateral gaze and gaze upward and downward are used to confirm *gaze directional* or *gaze-paretic* nystagmus.

The direction (←), frequency (>> ), and ampli tude (=) of the eye movements observed are re corded on a Frenzel s chart (**Figs. 1.57**).

**Electronystagmography (ENG).** The eye is a dipole in which the cornea is electropositive and the retina electronegative. The periocular electrical field therefore changes when the eyes move. This change in the corneoretinal potential is proportional to the amplitude, frequency, and speed of the nystagmus. It can be picked up and recorded by electrodes and analyzed. The direction of the eye movements is demonstrated by a positive or negative corneoreti nal potential (**Fig. 1.58a** ).

**Video nystagmography (VNG).** The eye move ments are recorded by a touchless video camera. The position of the dark pupil of the eye can be recorded by a processor that analyzes the eye

horizontal and vertical rotation.

**Spontaneous Nystagmus**

This term includes all eye movements that have the character of nystagmus and are not induced by external stimulation of the vestibular and visual systems (**Fig. 1.59**). The fast component usually beats toward the side of the functionally dominant vestibular center.

Three main forms of spontaneous nystagmus can be distinguished:

**Spontaneous vestibular nystagmus.** This disorder may be due either to a peripheral vestibular disor der, in which case the fast component of the nys tagmus always beats toward the dominant laby rinth; or it may be caused by a central vestibular disorder. The inhibitory impulses on the vestibular center are suppressed (see p. 20). The nystagmus beats on the side of the lesion.

*Methods of Investigation* **43**

**Fig. 1.56** Frenzel 

glasses with magnifying

lenses allow assessment

of nystagmus.

*Recovery nystagmus* may be due either to a central com pensatory process after a peripheral lesion, or to the recov ery of peripheral function. In both cases, it is directed toward the side of the dominant vestibular center i. e., in this case toward the affected ear.

**Gaze-evoked and gaze-paretic nystagmus.** This form of nystagmus is always induced by a central lesion. Often it beats to both sides and in both the horizontal and vertical planes. It only appears after deviation of the globe by more than 30 for at least 30 s.

*An exceptional form* of toxic gaze-evoked nystagmus may occur after barbiturate or alcohol poisoning, due to release of the central inhibitory effect.

This form of nystagmus is due to a lesion affecting voluntary motor control of gaze, which in serious cases is accompanied by paralysis of gaze. Transitions from gaze evoked to gaze-paretic nystagmus are fluid. The latter is characterized by a nystagmus to the side of the gaze paresis.

This is due to a congenital or acquired disorder (such as multiple sclerosis) of the gaze centers of the reticular for mation of the pons (the center for horizontal gaze move ment) and of the tegmentum of the midbrain (the center for vertical gaze movements). These centers are involved in central voluntary motor control of gaze (integration of vol untary gaze impulses and visual and vestibular afferents), binocular coordination via the medial longitudinal bundle (see **Fig. 1.21**), and the rhythm of nystagmus. Lesions in this area of the brain stem therefore lead to serious abnormal ities of gaze movements and nystagmus such as changes in the rhythm and form of beat, dissociation of movements of the right and left eyes, extinction of the fast phase of nystagmus, unilateral or bilateral enhancement of optoki netic nystagmus, gaze-evoked and gaze-paretic nystagmus, and internuclear ophthalmoplegia.

**44 1 Ear**

| Nystagmus  Upward  Fine Average Coarse  Right Left  Gaze straight  Little Average Very frequent  Downward  Without Vertigo With  Uncertain,  only a suspicion  Horizontal  rotatory  of nystagmus |
| --- |

| + +  +  +  +  +  +  +  +  +  +  -  +  -  -  -  -  -  -  -  -  -  -  -  Nystagmograph  Nystagmograph  Nystagmograph  V  V  V  **a bc**  t  t  t |
| --- |

**Classification of spontaneous nystagmus**

Eyes open in the

**Fig. 1.57** Symbols for record ing nystagmus and vertigo (left). The direction is recorded on a Frenzel s chart (right). > = direction,

= amplitude,

>> = frequency.

**Fig. 1.58a** The principle of nystagmography.

Gaze straight ahead. The nasal and temporal electrodes are positive, and the isoelectric baseline is horizontal.

The eyeball is turned slowly to the right (slow phase). The nasal electrode is positive, the tem poral electrode is negative, and the baseline is displaced superi orly.

The eyeball returns quickly (fast phase), the baseline returns to the neutral position, and both electrodes are positive.

**Fig. 1.59** Classification of spontaneous nystagmus (u = upward, s = straight,

Spontaneous vestibular nystagmus

Gaze-evoked or

gaze-paretic

nystagmus

Fixation nystagmus

dark with Frenzel’s glasses

Even open

in the light,

30° lateral gaze

d = downward).

a) Pendular nystagmus Binocular fixation r l u

b) Latent nystagmus

s d

Monocular fixation

**Fixation nystagmus.** This form of nystagmus does not have any typical fast or slow components, but rather a pendular movement. It almost always oc curs with binocular fixation, but may rarely be seen with monocular fixation. It is often congenital and may even be hereditary. Synonyms for it include *congenital* or *hereditary pendular nystagmus.*

The three main forms of spontaneous nystagmus should not be confused with the following:

*Endpoint nystagmus,* a short-lived, nonpathologic, rapidly decaying beat at the extremes of gaze i. e., more than 50 deviation.

*Fatigue nystagmus,* which occurs during prolonged lateral gaze due to fatigue of the lateral rectus muscle, similar to tremor in skeletal muscles. This is also nonpathologic. *Adjustment nystagmus,* which is due to adjustment of movements of a nystagmoid character when fixing on an object in the visual field. There is a rapid beat that fatigues quickly. This, too, is nonpathologic.

**Provoked Nystagmus**

Unlike spontaneous nystagmus, this is ex clusively a vestibular-induced nystagmus that only appears after specific stimuli, such as changes in the posi tion of the body or of the head.

Frenzel s glasses are used to investigate this con dition. The same criteria are used for assessing pro voked nystagmus as for spontaneous nystagmus; however, the duration of eye movements is also taken into account. One of the following patterns of nystagmus may be seen:

*Transitory* nystagmus, which lasts less than 60 s. Continually beating *persistent* nystagmus. *Head-shaking nystagmus* i. e., release sponta neous nystagmus of peripheral or central origin. This may be transitory or persistent.

***Provocation Measures***

**Head-shaking.** Spontaneous nystagmus can be pro voked by gentle, passive, horizontal shaking of the patient s head.

**Positional testing (static).** The nystagmus is in duced by adopting various body positions in slow motion (supine, lateral decubitus, head-hanging). The vestibular apparatus and the otolithic organs in particular are exposed to various gravitational stimuli in the different positions (**Fig. 1.60a**).

*Methods of Investigation* **45**

**Positional testing (dynamic).** This involves a uni lateral quick movement of the patient back to a head-hanging position for 60 s. The test is repeated on the opposite side (*Dix Hallpike maneuver*) (**Fig. 1.60b**).

It is a useful test for diagnosing *benign paroxysmal posi tional vertigo* (BPPV, see p. 100). The classic finding includes, after a short latent period of 5 10 s, a horizontal or rotatory downward-beating nystagmus toward the underlying ear, with an increase in intensity. Later, after 15 30 s, it de creases and a marked subjective feeling of vertigo arises. Occasionally, a transitory nystagmus beating in the opposite direction occurs when the patient sits up. In addition to this *paroxysmal positional nystagmus*, persistent or transitory direction-determined nystagmus, regular direction chang ing, and irregular positional nystagmus may be observed. The latter is always of central origin.

**Experimental Tests of the Vestibular System**

These tests and analysis of them should be per formed by a specialist.

**Turning test.** The rotatory or turning test uses *an gular acceleration* as the stimulus for investigating the sensitivity of the horizontal semicircular canals. Rotation around an axis passing through the head stimulates one or more semicircular canals on each side, depending on the head position. The left and right sides are stimulated in an opposing fashion. Rotation of the head in one direction (e. g., to the right) induces nystagmus toward the same side (rotatory nystagmus). When the rotation ceases, the nystagmus reverses to the opposite direction (postrotatory nystagmus). The test is carried out in a darkened room using a rotating chair.

Using the rotatory test, it can be determined whether the vestibular apparatus is functioning properly or whether there are signs of a functional or regulatory disorder (imbalance to one side, swinging).

**Caloric labyrinthine testing.** The principle involved in these tests is illustrated in **Fig. 1.61a** . The horizontal semicircular canal is brought into a ver tical position in the supine patient. Cooling or warming the labyrinthine capsule by irrigation with water at 30 C or 44 C for 30 40 s induces convection currents and a slight change in the volume of the endolymph. This produces move

**46 1 Ear**

| 1  Step 3  2  Step 2  3  4  Step 1  **a b** |
| --- |

**Fig. 1.60a, b a** Positional testing (static). Beginning with a supine position on the examining table, the patient rolls onto the right side and then rolls back to the supine position and continues rolling onto the left side. After that, the patient adopts a head-hanging position.

Positioning test (dynamic). A dynamic provocation is car ried out. Starting in the sitting position, the patient adopts a head-hanging position, and the upper body is then swiftly brought back to the sagittal plane (step 1). The head is then turned to the left (or right) and the patient adopts the head hanging position for each side (steps 2 and 3).

| 44°C  1  2 2  30°C  Nystagmus  3 **a** |
| --- |



**Fig. 1.61a** Principle of caloric labyrinthine tests. Temperature changes at the lateral labyrinthine capsule cause a change in the density of the perilymph, leading either to an influx away from the ampulla (cold stimulus) or to an influx toward the ampulla (warm stimulus). The

oscillation of the cupula produces a neural stimulus that is transmitted by the vestibular nerve ( ) to the vestibular nucleus ( ) and ocular muscle nucleus ( ).

Examination with electrode deflection.

Examination with video nystagmography.

ment of the endolymph, which deflects the cupula. This process has exactly the same electrophysio logic effect as deflection of the cupula by angular acceleration (see **Fig. 1.28a** i. e., it induces nys tagmus via the vestibuloocular reflex.

The *extent ofthe caloric response* (the nystagmus and the subjective feeling of dizziness) gives some indication of the function of the stimulated laby rinth. *Reduced excitability* indicates partial func tional loss, and *lack ofresponse indicates subtotal or complete loss offunction.* The advantage of caloric tests is that the labyrinth on each side can be inves tigated separately.

**Fistula test.** In the presence of a fistula in the hor izontal semicircular canal or elsewhere in the la byrinthine capsule (see p. 8), caused by an inflam matory osteolytic process such as cholesteatoma, a sudden increase in pressure in the external auditory meatus produces a subjective feeling of dizziness, objective nystagmus, and lateropulsion (**Fig. 1.62**). The same phenomenon can also occur when there are adhesions between the membranous labyrinth and the stapes footplate (fistula symptom without a labyrinthine fistula, see p. 81).

*Technique:* A Politzer balloon with a perforated olive is introduced into the meatus. Compression induces nystagmus toward the affected ear and aspiration to the other side.

*Pseudofistula symptom:* Inflation or aspiration of air in a patient with a large defect in the tympanic membrane produces cooling of the horizontal semicircular canal, which induces a caloric labyrin thine reaction and thus nystagmus. However, this always beats toward the sound ear for both com pression and aspiration.

Note: The fistula symptom always has to be tested in chronic otitis media with marginal perforation of the tympanic membrane, especially when there is a suspi cion of cholesteatoma of the middle ear.

**Optokinetic Function and Pursuit Tracking** The optokinetic and eye-tracking tests are among the most sensitive methods available for detecting central oculomotor lesions. They are indispensable for distinguishing peripheral from central disorders of balance, as the two systems are functionally very closely linked.

*Methods of Investigation* **47**

| 3  1  Compression  2 2 |
| --- |

**Fig. 1.62** A positive fistula test due to erosion of the right lateral semicircular canal by cholesteatoma. , Vestibular nucleus; , oculomotor nerve nuclei; , cholesteatoma.

*Principle:* Observation of an object moving within a sta tionary visual field (foveal stimulation) or observation of the displacement of the entire visual field (foveoretinal stimula tion). Only the latter induces the optokinetic reflex a con jugated reflex eye movement that shows a slow movement in the direction of the displacement of the moving object or visual field (gaze following movement) and a fast phase (the central correction movement) in the opposite direction. This is described as *optokinetic nystagmus.*

*Brain stem lesions,* especially of the pons and cerebellum, produce the changes involved in optokinetic nystagmus, such as unilateral directional preponderance, disintegration of coordinated movement of the left and right eye, and complete unilateral or bidirectional disintegration of coordi nated movement. Gaze-evoked or gaze-paretic nystagmus and simultaneous abnormalities of optokinetic nystagmus are characteristic early symptoms of multiple sclerosis.

**Investigation of the Facial Nerve**

The first and most important investigation serves to differentiate central from peripheral paralysis: In *central paralysis,* the function of the branches to the forehead is preserved.

**48 1 Ear**

In *peripheral paralysis,* all three branches are affected. The secretion of tears and sensitivity for taste are affected, and hyperacusis may occur due to disruption of the stapedial reflex.

The *topographic diagnosis* of peripheral lesions of the facial nerve is shown in **Fig. 1.22**

**Taste.** The anterior two-thirds of the tongue is in nervated by the chorda tympani. The test stimulus is 20 % sugar, 10 % saline, or 5 % citric acid solution (see **Fig. 3.2 c**).

**Gustometry.** The peripheral taste fibers are stimu lated electrically, and the threshold is measured in milliamperes (see p. 242).

**Schirmer test.** The Schirmer test uses paper strips inserted into the eye for several minutes to measure the production of tears. The eyes are closed for 5 min. The paper is then removed and the amount of moisture is measured. Sometimes a topical anes thetic is administered to the eye before the filter paper, to prevent tear production due to irritation from the paper. The use of the anesthetic ensures that only basal tear secretion is measured. The re duction in the secretion of tears due to interruption of the lacrimal anastomosis in the greater super ficial petrosal nerve is measured on the paralyzed side (see **Fig. 1.22**).

The *stapedial reflex* is measured using imped ance audiometry (see p. 37).

**Electrical and Magnetic Excitability Tests** The severity and prognosis of a paralysis can only be determined by *electrodiagnosis.*

Note: Every facial paralysis, of whatever cause, has to be investigated as early as possible using electrodiagnostic methods.

Three stages of a lesion can be distinguished: *Neurapraxia:* There is complete absence of function, but without interruption of the axon. This stage is reversible. *Axonotmesis:* There is disruption of the axon with preser vation of the connective-tissue framework of the nerve (endoneurium, perineurium, and epineurium). These le sions usually do not recover completely.

*Neurotmesis:* There is disruption of the axon and of the supporting tissues. This is irreversible without surgical intervention.

**Electromyography (EMG).** Muscle action potentials are picked up by a needle electrode on voluntary contraction of the facial musculature. The EMG is of relatively little value in the acute phase of facial paralysis, since denervation potentials only appear 12 days after the start of the paralysis.

**Electroneuronography (ENoG).** The sums of the ac tion potentials of the facial musculature induced by contraction in response to maximal percutaneous faradic stimulation are measured. The proportion of degenerated fibers can be assessed approximately by comparing the summation potential between the healthy and the paralyzed side.

**Nerve excitability test (NET).** The strength of cur rent in milliamperes that is sufficient to induce a muscle twitch at a constant duration of impulse of 0.3 ms is assessed. The threshold for the two facial nerves varies to an insignificant extent (0.4 mA) in the same individual. A difference in threshold be tween the two sides that is greater than or equal to 3.5 mA is abnormal. An increase in the threshold indicates progressive degeneration of the nerve fi bers or progressive axonotmesis.

**Transcranial magnetic stimulation (TCMS).** The stim ulation is administered by applying a quickly changing mag netic field above the motor cortex and the facial nerve. The summation potential is diverted by the mimetic muscles. This makes it possible to measure the motor transmission speed and the location of the nerve lesion.

In the acute phase of paralysis, the results of NET and ENoG (rheobase and chronaxy) provide impor tant information about the extent and progression of degenerative processes in the nerve and are de cisive in determining the choice of treatment.

*Clinical Aspects of Diseases of the External Ear* **49**

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**Fig. 1.63a, b** Bat ears are characterized by hypertrophy of the conchal cartilage or failure of the auricle to fold.

**Clinical Aspects of Diseases of the External Ear**

**Congenital Anomalies**

In addition to wide variation in the position, size, and shape of the auricle, there are also numerous disfiguring anomalies. Classifying auricular de formities provides an overview of these congenital dysplasias, at increasing degrees of severity:

*First-degree dysplasia*: minor deformities (e. g., prominent ear, macrotia, scaphoid deformity, lobular deformities, cup ear deformities types 1 and 2).

*Second-degree dysplasia*: some characteristics of a normal auricular structure are present (e. g., first- and second-degree microtia, cup ear type 3).

*Third-degree dysplasia*: absence of a normal auricular structure (e. g., third-degree microtia, anotia).

**Lop ear (bat ear** This is one of the most common disfiguring anomalies (**Fig. 1.63a, b**). It is often caused by hypertrophy or excess curvature of the conchal cartilage, or by failure of the auricle to fold due to underdevelopment or absence of the anti helix.

*Treatment:* The deep conchal cavity is corrected, and an antihelixplasty is performed. The angle be tween the auricle and the head is reduced to the ideal value of 30 and the scaphoconchal angle to 90 . The operation should be performed during pre school years.

**Fig. 1.64a, b** Dysplasia of the auricle: type II microtia ( and anotia, absence of the auricle ( ).

**Microtia, anotia.** *Microtia* (abnormal smallness of the auricle of the ear, **Fig. 1.64a**) and *anotia* (ab sence of the auricle or the ear, **Fig. 1.64b**) are often associated with auditory canal stenosis or atresia and with middle ear deformities. Deformities of the external ear and the middle ear can also occur in connection with other facial deformities (e. g., Fran ceschetti syndrome (**Fig. 1.65**), mandibulofacial dysostosis, Treacher Collins syndrome).

**Congenital aural fistulas and auricular append ages.** These are usually located in front of the auricle. They are due to incomplete closure of the first branchial cleft or incomplete fusion of the auricular hillocks. Three groups can be distin guished on the basis of the embryologic association and site:

1. Preauricular fistulas, between the angle of the mouth and the tragus.

2. Fistulas that begin in front of the ascending helix and lead toward the meatus, or open externally inferior to the angle of the jaw as a hyomandib ular fistula (**Fig. 1.66**).

3. A small fistula or pitted depression affecting any part of the auricle.

*Treatment:* Treatment is excision, with careful con sideration of the danger of damaging the parotid gland or facial nerve (see pp. 112 and 435).

**Reconstructive Operations on the Auricle**

Reconstructive procedures on the auricle vary in difficulty. Correcting an anomaly of the auricle shape (remodeling the auricular cartilage) can be relatively simple in the hands of a skilled surgeon

**50 1 Ear**

**Fig. 1.65** Franceschetti 

Syndrome: deformity of

the auricle and middle ear,

with facial deformities.

**Fig. 1.66** Preauricular 

fistula.

**Fig. 1.67** Perichondritis 

of the auricle.

and can lead to very satisfactory results. However, reconstruction of the entire auricle is one of the most difficult tasks in plastic surgery in this area.

Thorough familiarity with skin and cartilage transplanta tion techniques is a prerequisite for the success of this reconstruction procedure. Costal cartilage is an ideal source for graft, and specialist instruments are required for model ing the graft. Staged surgery is necessary. Revision surgery may be required. Providing skin to create the new auricle is not easy and requires the construction of rotation and sliding flaps from the neck and from the region of the hairline. Prostheses should be considered as an alternative in difficult cases.

Reconstruction of the external auditory meatus and the sound-conducting apparatus is discussed in the section on anomalies of the middle ear (see p. 94).

**Inflammations of the External Ear**

**Nonspecific Inflammation**

**Classification.** Bacterial or fungal inflammation of the skin of the external auditory canal, with or without inflammation of the tympanic membrane (myringitis).

**Clinical Features.** *Acute exudative inflammatory phase:*

Swelling of the skin of the auditory canal with lubricious, often fetid secretion.

The tympanic membrane is often not visible, due to shifting of the auditory canal.

There is an accumulation of detritus in the audi tory canal.

The cartilaginous part of the meatus is painful. Painful limitation of motion of the adjacent tem poromandibular joint.

The regional retroauricular lymph nodes may be so enlarged and tender to pressure in serious cases that the condition bears a strong re semblance to *pseudo-mastoiditis*

*Chronic inflammatory phase:*

The meatus is wide, the epithelial lining is atro phic, and dry scales of epidermis accumulate. There is intense itching, which causes scratching by the patient. This promotes the development of *superinfection* with acute dermatitis, with or without perichondritis (**Fig. 1.67**).

**Pathogenesis**

Maceration of the meatal skin by exogenous and endogenous factors, such as fluid, mechanical and chemical damage, allergy, and diabetes. Reduction of the elasticity of the meatal skin and atrophy of the ceruminous and sebaceous glands.

Loss of the protective film of secretion, with drying of the meatal skin, disturbance of its chemical balance, and increased susceptibility to infection by bacteria and fungi.

Disturbance of the pH balance of the meatal skin, leading to the growth of anaerobic bacteria.

Factors that encourage the elimination of patho gens in the meatal skin include:

Low pH values.

Fatty acids in the secretions of the sebaceous glands.

Normal lysozyme content of the secretions of the ceruminous glands.

A normal self-cleaning mechanism, with exter nal migration of the meatal epithelium.

Disturbance of protective factors as may occur in allergy, with displacement of the pH to alkaline values, reduction of the protective film, and changes in the composition of the secretions due to mechanical stimulation or recurrent inflamma tion can cause chronic or recurrent otitis externa.

**Diagnosis and differential diagnosis**

The inflammation is localized to the auricle, ex ternal auditory meatus, and regional lymph no des.

Inflammation is often associated with etiological skin conditions such as eczema or psoriasis. Extension to the outer and middle layers of the tympanic membrane (myringitis) is only an ex ceptional occurrence.

The middle ear and mastoid are not affected by the disease.

Conductive deafness due to obstruction of the external auditory meatus is unusual and mild in nature.

It is important to exclude acute otitis media, mastoiditis, and the acute phase of a chronic middle ear inflammation with cholesteatoma. Pain when pressure is applied to the tragus strongly suggests otitis externa.

**Investigations.** These include otoscopy, a specimen of pus for bacteriology, possibly irrigation of the ear, hearing and tuning-fork tests, audiography, and imaging. If there is any clinical suspicion of mastoi ditis or other infective complications, a CT scan should be done.

**Treatment.** The external auditory meatus is cleaned manually under vision, ideally with a mi croscope, or by irrigation with water at 37 C. The external auditory meatus is dried, and eardrops consisting of locally active broad-spectrum antibi otics and a corticosteroid are instilled several times a day during the moist phase. In severe cases, sys

*Clinical Aspects of Diseases of the External Ear* **51**

**Fig. 1.68** Erysipelas of 

the skin.

temic antibiotics are given. Once the acute inflam matory phase subsides, local applications of oint ments based on a combination of an antibiotic and a steroid are used. However, certain antibiotics, not ably neomycin, can themselves cause an allergic skin reaction. In these cases, the local use of 70 80 % pure alcohol is indicated, and in the acute inflammatory phase, a fine gauze wick should be introduced into the ear. This is moistened repeat edly with alcohol, which opens the external audi tory meatus and reduces the swelling of the meatal skin by absorbing moisture.

**Specific Forms of Inflammation of the External Ear**

**Bacterial and Viral Diffuse Otitis Externa** A characteristic form of *erysipelas* occurs in strep tococcal infection. In *swimmer s otitis,* due to mac eration of the skin by halogen-containing swim ming-pool water and deep penetration of virulent organisms, there is usually a deep-seated cellulitic inflammation (**Fig. 1.68**), with *perichondritis.* In ad dition to swimmer s otitis externa, confined to the external ear, a tubal form of swimmer s otitis media may also occur.

**Clinical Features.** These include fever, generalized illness, regional lymphadenitis, and pain when the auricle is pulled or pressure is applied to the tragus. A cellulitic form can extend to surrounding tissues and organs, such as the parotid gland, mastoid, and skull base, and in exceptional cases can cause os teomyelitis of the temporal bone and septicemia *(necrotizing otitis externa;* see p. 53). In severe cases of otitis externa, especially in infants and young children, there is complete obstruction of the ex

**52 1 Ear**

**Fig. 1.69** Edematous 

swelling of the lobule of

the auricle is a pathogno

monic sign in patients with

Lyme disease.

ternal auditory meatus, with an accompanying ret roauricular lymphadenitis. The ear is displaced lat erally, and the patient may appear to have mastoi ditis.

**Treatment.** Systemic antibiotics; local reduction of the swelling of the skin of the external auditory meatus with 70 95 % alcohol, chloramine in a 1 : 1000 irrigation, or local application of topical antibiotics with or without a steroid.

**Furuncle of the Ear (Otitis Externa Circum scripta)**

The patient is in good general condition, but has local pain in the ear. A characteristic finding is a circumscribed, exquisitely painful swelling in the cartilaginous part of the external auditory meatus (hair follicles), modest regional lymphadenitis, and pain when pressure is applied to the tragus or the auricle is pulled.

**Treatment.** Gauze wicks soaked with alcohol (70 95 %) are used until the furuncle points and bursts spontaneously. Incision and systemic anti biotics are only exceptionally indicated in patients with severe pain, a protracted course, or marked swelling.

Note: The glucose content of the urine and blood should be checked in patients with recurrent furunculo sis of the external meatus.

**Lyme Disease (Lyme Borreliosis)**

Lyme disease is a multisystemic bacterial infection caused by species of bacteria belonging to the genus *Borrelia.* It is transmitted to humans by the bite of an infected hard tick.

**Fig. 1.70** Herpes zoster 

oticus.

The early infection often starts with flu-like symptoms such as headache, stiff neck, fever, muscle aches, and fatigue. An enlarging rash, known as erythema migrans (EM), develops days to weeks later at the site of the bite (**Fig. 1.69**). Neurological problems such as severe headaches, meningitis, or cranial nerve involvement can occur at a later stage. Changes in smell or taste, vocal cord and facial nerve paralysis, vertigo with nystagmus, and hearing and swallowing disorders are typical otolaryngological symptoms. Progression of the in fection is characterized by central nervous symp toms such as cognitive changes (memory problems, difficulty in finding words, confusion, decreased concentration, problems with numbers) and be havioral changes (depression, personality changes).

**Herpes Zoster Oticus**

This disease is characterized by multiple herpetic vesicles arranged in groups on the auricle, the ex ternal auditory meatus, and occasionally the tym panic membrane (**Fig. 1.70**). In severe cases, disor ders of hearing and balance and facial paralysis may occur (see **Table 1.16**, p. 99, and p. 111).

**Bullous Myringitis**

This disease usually occurs in association with an influenzal infection. It is occasionally combined with otitis media, in which case the patient has conductive deafness. Initially there is a moist, bluish-livid bullous inflammation, which can ex tend to the tympanic membrane. After a few days, the hemorrhagic vesicles dry out and heal without complications. The patient usually reports ex tremely severe pain.

Note: If the middle ear is involved, systemic antibiotics must be administered immediately due to the danger of superinfection.

In most cases, treatment is limited to simple cleans ing of the external meatus and otoscopic control. Antibiotics are only given for protection against secondary infection.

**Necrotizing Otitis Externa**

Severe necrotizing inflammation can develop from commonplace otitis externa, especially in patients with diabetes, whether latent or manifest. The in fection is generally caused by optionally anaerobic Gram-negative organisms, usually *Pseudomonas aeruginosa.* The infection spreads through the tis sue clefts of the cartilaginous meatus and ex tends into the depths of the retromandibular fossa and along the base of the skull as far as the jugular foramen, and it leads to insidious osteomyelitis of the temporal bone.

**Diagnosis.** The hallmark of this condition is pain in and around the ear. In addition to examining the auditory canal and carrying out a hearing test, it is necessary to examine the neighboring cranial nerves (examination with Frenzel glasses for spon taneous nystagmus; examination of the facial nerve, trigeminal nerve, and abducent nerve), ultra sound examination of the parotid and neck, micro biological culture and sensitivity tests, CT/MRI of the base of the skull, blood sugar profile, C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), hemoglobin, and full blood count.

**Treatment.** Treatment of diabetes and intensive anti-inflammatory therapy with broad-spectrum antibiotics (e. g., ciprofloxacin) is fundamental. Lo cal treatment of the external acoustic meatus pro motes the drainage of infective secretions. In severe cases, generous drainage of the retromandibular space, infratemporal fossa, and pterygopalatine fossa together with debridement of necrotic tissue from the external meatus may become necessary. Ligation or resection of the internal jugular vein may also be indicated if it is involved.

*Clinical Aspects of Diseases of the External Ear* **53**

**Prognosis.** Even with massive antibiotics and ex tensive surgery, the prognosis is poor, due to septi cemia and sinus thrombosis.

**Chronic Inflammation**

*Tuberculosis* and *syphilis* (stage 2) cause local, circumscribed lesions of the external ear and auditory meatus. The therapy is focused on local dermatological treatment and specific high-dose antibiotic treatment.

**Otomycosis and Eczema**

**Otomycosis**

**Clinical Features.** The infection is caused by a fun gus and is limited to the external auditory meatus. There may be a recent history of antibiotic drops being used to treat bacterial infection. Typically, a fine, easily removable coating that is loose and fluffy, varying in color from whitish-yellow to greenish-black, is visible. The patient reports itch ing, but rarely describes pain.

**Diagnosis.** Culture and sensitivity tests demon strate fungal mycelium.

**Treatment.** The mainstay of treatment is manual cleaning, but irrigation should be avoided if possi ble to prevent the formation of a moist chamber,

which promotes the growth of the fungus. Antimycotic agents, such as 1 % clotrimazole or clioquinol, can be applied locally unless contraindi cated by perforation of the tympanic membrane. Antibiotics should not be used. Daily painting of the meatus with thiomersal or 2 % acetic acid spray helps. In severe cases, systemic antimycotics such as fluconazole can be used.

**Course.** The course is chronic and recurrent.

**Eczema of the Ear**

**Clinical Features.** The disease follows an episodic course, with intermittent acute exacerbations. The entry of fluid such as sweat or water during wash ing, or the presence of a moist ex udate, promote colonization by pathogenic bacteria or fungi in the relatively enclosed external meatus.

**54 1 Ear**

**Fig. 1.71** Contact der 

matitis caused by jewelry

containing nickel.

*In the acute stage,* there is deep-red inflammatory swel ling with moist vesicles and pustules. Later, crusts accumu late. Rhagades form around the meatal introitus, and fetid debris collects. The usual picture is one of nonspecific acute otitis externa, but occasionally the appearance may progress to chronic myringitis with superficial granulations.

*In the chronic stage,* the skin is atrophic, dry, scaly, and may be partly lichenified. The patient reports chronic irrita tion. Occasionally, stenosis may occur.

**Diagnosis and differential diagnosis.** *Contact eczema* may be due to cosmetic solutions, hair sprays, glasses frames made of metal or plastic, cement or flour dust, or medications e. g., antibiotics. Skin tests have to be per formed in order to identify the antigen (**Fig. 1.71**).

*Microbial eczema* is mainly due to infection with staph ylococci or to oral mycosis. A swab should be taken for culture and sensitivity tests.

*Seborrheic eczema* is the most common form. It is often combined with acne.

*Endogenous eczema* is a localized manifestation of a gen eralized eczema.

**Treatment.** Elimination of the allergen and local treatment, as detailed above.

**Trauma**

**Sharp or Blunt Trauma to the External Ear** Any injury to the auricle and cartilaginous part of the external auditory meatus can damage the peri chondrium, causing cartilaginous necrosis. A dis tinction is made between auricle lacerations and avulsions either of the whole auricle or parts of it.

**Treatment.** For lacerations, primary closure of the wound with monofilament suture material (6 0 or 0) is recommended, and the duration of antibi

otic treatment depends on the degree of contami nation. In comprehensive lacerations with cartilag inous involvement and extensive skin damage, it is recommended following wound debridement if necessary to close the wound, starting on the pos terior aspect of the auricle, with monofilament su ture material.

The cartilage should be repaired with 5 0 absorbable suture material, and the skin of the anterior surface with monofilament 6 0 suture material. Replantation of the avulsed part of the auricle in the acute situation is also the treatment of choice for partial avulsion injuries;ischemia periods of up to 8 hours are tolerable and still allow the graft to take. If the graft does not take, plastic reconstruction is necessary in the interval.

**Prognosis.** Bacterial infection can cause perichon dritis, with partial or complete destruction of the cartilaginous framework, leading to cauliflower ear (see **Fig. 1.73**) or atresia of the meatus.

**Hematoma of the Auricle**

This arises from closed blunt injury, with dissection of the skin and perichondrial layer from the carti lage and the formation of a subperichondrial hem atoma (**Fig. 1.72**).

**Treatment.** The aim of treatment is to remove the seroma or hematoma and to achieve long-term adaptation of the perichondrium to the cartilage in order prevent leakage into the seroma cavity.

In general, drainage from the anterior surface can be carried out, with the incision being made in the fossa of the helix (scapha). Following curettage and readaptation of the skin with monofilament suture material, light compression for 3 5 days with a dental roll, secured with through-and through sutures, is recommended. For drainage from the posterior surface of the auricle, an incision has to be made in the cartilage to reach a seroma/hematoma on the anterior surface, and a cartilage window may be necessary to achieve adherence of the perichondrium layers. Antibiotic treatment is recommended in order to prevent perichondritis.

Note: Repeated aspiration may cause a seroma or super infection, leading to perichondritis.

If a hematoma is not treated, connective-tissue or ganization, secondary calcification, and deformity of the auricle occur, leading to cauliflower ear (**Fig. 1.73**).

**Frostbite**

Grade 1: Cyanosis of the skin due to vascular spasm. Grade 2: Ischemia with formation of vesicles. Grade 3: Deep necrosis of tissue.

**Treatment.** Sterile dressings, antibiotics, intrave nous vasodilators, and possibly stellate ganglion block are used, depending on the severity of the injury. The area has to be kept dry.

*Burns* require the same treatment as burns to the skin; particular attention needs be given to the close relationship between the skin and the carti lage.

*Late complications* include necrosis of the auricle and atresia or stenosis of the external auditory meatus.

**Wax and Foreign Bodies**

**Wax**

Note: Collections of wax and cell debris are unusual if the self-cleaning mechanism of the external auditory meatus is undisturbed. The present widespread habit of cleaning the external ear daily with ready-made cotton swabs is inappropriate and can sooner or later lead to the development of chronic otitis externa.

*Wax* is a yellowish-brown mass consisting of secre tions of the sebaceous and ceruminous glands, des quamated epithelium, hair, and particles of dirt.

**Plug of wax.** The normal symptom is deafness when the external auditory meatus is completely closed, but occasional complaints include a roaring noise in the head and a feeling of dizziness.

**Differential diagnosis.** A plug of epidermis, foreign body, dried blood, purulent exudate, and cholestea toma of the meatus or of the middle ear.

*plug ofepidermis* is a compact white mass consisting of desquamated epithelial crusts, which usually adhere firmly to the skin of the meatus.

**Treatment.** The ear is irrigated with water at 37 C. Manual syringes are now rarely used, and electri cally controlled water jets with controlled pressure are used instead. It should be ensured that a perfo ration of the tympanic membrane is not hidden

*Clinical Aspects of Diseases of the External Ear* **55**

**Fig. 1.72** Auricular hem 

atoma (othematoma).

**Fig. 1.73** Untreated 

hematoma or infection

can lead to a deformity of

the auricle cauliflower

ear.

behind the plug of wax. This should be excluded by taking a careful history. If the patient does have such a perforation, the waxshould be removed manually by a specialist. Hard waxshould be first softened with soft soap drops, 5 % sodium bicarbon ate, olive oil drops, or 3 % hydrogen peroxide for up to 1 week before syringing. The moist external auditory meatus should be mopped out with cotton applicators. Local steroid or antibiotic creams or eardrops should be prescribed for patients with inflammation of the meatus. For medicolegal rea sons, the patient s hearing should be tested after this procedure.

**Foreign Bodies**

*Foreign bodies* are diagnosed using careful otoscopy. The findings vary depending on the object and the length of time it has been in the ear. In children, a careful history should be taken to establish the nature of the foreign body. Depending on their age, children may be able to indicate that they have a foreign body in the ear, or they may present with symptoms of ear pain or discharge. Insects can cause injury to the canal or tympanic membrane as a result of scratching or stinging.

Physical pain or bleeding may occur with objects that abrade the ear canal or rupture the tympanic membrane, or as a result of the patient s attempts to

**56 1 Ear**

| **a b** |
| --- |

**Fig. 1.74a, b** Attempts to re move a foreign body with simple forceps ( ) may displace the foreign body more deeply, and it may perforate the tympanic membrane, causing dislocation of the ossicles and injury to the facial canal. The foreign body can be removed easily without danger to the patient using a hook, under otoscopic or mi croscopic guidance ( ).

remove the object. Hearing loss may be observed. If the presentation is delayed, erythema and swelling of the canal and a foul-smelling discharge may be present.

Note: Foreign bodies that cannot be removed by syring ing should be removed manually, with general anesthe sia being administered in small children.

If a perforation of the tympanic membrane is known of or suspected, the ear should not be syringed.

Blind attempts to extract foreign bodies without oto scopic control, or attempts to extract them under vision with unsuitable instruments and inadequate anesthesia, should be avoided. Instrumental removal of foreign bodies from the meatus should therefore only be carried out by a specialist, except for the simplest cases.

**Figure 1.74a** shows correct and incorrect methods of extracting a foreign body from the meatus.

**Tumors**

**Benign Tumors**

These include retroauricular atheroma, cicatricial keloid, hemangioma and lymphangioma, dermoid tumors, fibroma, papilloma, keratoma, lipoma, and nevi.

**Treatment.** The treatment consists of tumor resec tion.

Meatal tumors include *hyperostosis,* caused by periosteal stimulation and resulting in appositional bone growth with progressive narrowing of the lu men of the meatus; and *exostosis,* a true bony tumor arising from the ossification centers in the anulus tympanicus. This tumor should only be removed if it is causing stenosis. *Chondrodermatitis nodularis*

*circumscripta helicis* causes painful nodes on the ear that should be distinguished from a premalignant lesion by the marked pain that occurs when pres sure is applied.

**Precancerous and Malignant Tumors**

The classification, symptoms, diagnosis, and differ ential diagnosis are shown in **Table 1.9**

**Precancerous Lesions**

Premalignant lesions often progress to true carci noma, and they should therefore always be treated with radical surgery, in the same way as true tu mors. Biopsy excision is not advisable, and any tu mor of the ex ternal ear suspected of being malig nant should be excised with a wide margin. The mainstay of treatment is surgery; radiotherapy or cryosurgery are second-best options.

*Precancerous lesions* such as senile keratosis (**Fig. 1.75a**) or Bowen disease are excised with a healthy margin. Periodic follow-up is necessary.

**Malignant Tumors**

**Basal cell carcinoma.** Surgical excision of basal cell carcinoma in an otherwise healthy patient is deci sive for the prognosis (**Fig. 1.75b**). A two-step sur gical procedure is the method of choice. The first step involves temporary wound coverage of the defect after resection of the tumor. After histolog ical confirmation that the wound margins are tu mor-free, the defect can be covered in a second operation, which may require reconstruction of the auricle. For very small tumors, a one-step pro cedure is possible, with primary wound closure after intraoperative histological control of the wound margins using frozen-section histology.

*Clinical Aspects of Diseases of the External Ear* **57**

**Table 1.9** Precancerous and malignant tumors of the external ear

**Form Color Skin surface Cartilage invasion Regional lymph node metastases**

Cutaneous horn

Senile

keratosis

A sharply limited warty growth of the epidermis

Smooth mass with indistinct borders

Inconspicuous Slightly nodular, intact

Yellowish-brown Rough, intact, occasionally cov

ered by crusts

None None None None

Bowen disease Smooth round lump

Intensive

brownish-red

Smooth but intact

None None

Basal cell carcinoma

Sharply demar cated smooth, slowly growing mass

Hyperemic, some times much more pigmented than the surrounding skin (special form:

pigmented basal cell carcinoma)

Often superfi cially ulcerated, crusted, raised edges with an atrophic center (or central ulcer)

Perichondrium some times infiltrated, tumor relatively

immobile at its base

Rarely

Squamous cell carcinoma

Exophytic tumor of relatively rapid growth with indis tinct margins

Often hyperemic Ulcerated, raised edges, superfi

cially nodular,

firm

Always

The tumor is not movable;occasionally perichondritis

20 %

Malignant melanoma

A round mass, sometimes verru cous, rapidly grow ing

Dark-brown to black, occasionally weakly pigmented, the ame lanotic melanoma

Smooth to

slightly nodular, occasionally ul cerated or bleeds easily to touch

Perichondrium often infiltrated and the tu mor is relatively im mobile at its base

Frequent

Also early distant metastases, espe cially to the lung



**Fig. 1.75a, b a** Senile keratosis of the auricle. Basal cell carcinoma.

**Fig. 1.76a, b** Squamous cell carcinoma. A small erosive carcinoma invading the helix. Carcinoma growing out of the external meatus.

**58 1 Ear**

**Keratinizing squamous cell carcinomas.** These are infiltrating, often ulcerated carcinomas (**Fig. 1.76a,** ). In 20 % of patients, the lesions have early meta stases to the regional lymph nodes. The treatment of choice is *radical surgery,* with no regard for the cosmetic result. Radiotherapy is only successful for tumors less than 1 cm in diameter that are not ulcerated, have not infiltrated the perichondrium, and have not metastasized to the regional lymph nodes. In all other cases, *partial or complete excision ofthe auricle* is the method of choice. Neck dissec tion is performed when there are regional lymph node metastases.

**Carcinoma of the external auditory meatus.** This represents 5 % of all aural carcinomas. In compar ison with tumors of the auricle, the prognosis is unfavorable due to late diagnosis and because the tumor penetrates early into the parotid space or the middle ear. The treatment of choice is ex tensive resection of the tumor with radical neck dissection and, if necessary, parotidectomy.

**Pigmented nevus and suspected malignant mela noma.** These should be treated with primary ex ci sion of the tumor, without previous biopsy. De pending on the histological results, it may be nec essary to carry out a secondary surgical excision, dictated by the extent and depth of the tumor (see p. 405), with regional neck dissection or radical neck dissection and, if necessary, parotidectomy. Postoperative radiotherapy, chemotherapy, or im munotherapy may also be necessary. Surgery should only be considered when distant metastases have been ruled out.

**Clinical Aspects of Diseases of the Middle and Internal Ear**

**Disorders of Ventilation and Drainage of the Middle Ear Spaces**

**Pathophysiology.** Most chronic middle ear diseases are based largely on two functional disturbances: *impaired middle ear ventilation* and *inflammation* Impairment of ventilation due to eustachian tube dysfunction is based on a mucosal inflammation in the nasopharynx, leading to inflammation of the middle ear mucosa.

**Pathogenesis.** The eustachian tube does not open regularly on swallowing, due to one of the following factors:

A dysfunctional tensor veli palatini muscle. Swelling of the tubal mucosa caused by chronic inflammation of neighboring structures, such as the sinuses or tonsils or by allergy.

Obstruction of the ostium of the tube by hyper trophic adenoids, in a child or adult.

Infiltration of the tube by a malignant tumor of the nasopharynx.

The middle ear is thus no longer aerated, and the remaining air is resorbed, producing a decrease in pressure that acts as an irritant to the middle ear mucosa.

In *short-term tubal occlusion* or persistent, re duced pressure in the middle ear, the following changes may occur:

Edema of the mucosa.

Middle ear fluid, due to transudation of the con stituent parts of the serum.

Stiffening of the ossicular chain, with retraction of the tympanic membrane.

Note: Ventilation and drainage disorders of the middle ear are usually caused by a dysfunctional opening mech anism of the tube, but mechanical obstruction of the tube by a lesion in the nasopharynx may also occur.

In *long-standing tubal occlusion* and reduced pres sure in the middle ear, the following changes may occur:

Metaplasia of the middle ear mucosa from the flat epithelial cells of the mucoperiosteum to form columnar, ciliated, mucus-producing gob let cells.

Increase in secretory activity of the goblet cells and mixing of the mucus with the transudate already present in the middle ear, causing a *seromucinous exudate.*

Formation of cholesterin-containing mucosal cysts (cholesterol granuloma).

The seromucinous exudate and mucosal changes substantially reduce the aeration of the middle ear, thus causing a vicious circle.

The metaplasia of the middle ear mucosa also affects the submucosa, causing firstly a proliferation of the connective tissue and secondly maturation of a local immunologically active cellular defense mechanism. The active secreting goblet cells produce a mucus blanket, which serves to trans port newly formed immunoglobulins to the mucosal sur face. The previously inactive mucoperiosteum of the middle ear is thus converted into a secreting hyperplastic respira tory mucosa, characterized by its newly acquired property of responding to every new stimulus (mechanical, chemical, bacterial, enzymatic, allergic, or autoimmune) with a com pletely mature defense mechanism. This consists of the following:

Mucociliary elements, which form the transport medium for the superficially active mucus blanket.

Enzymatic elements, which consist in particular of bac tericidal lysozymes and protease inhibitors.

Locally produced immunoglobulins.

The result of this increased activity in the local and mucosal immune system is that each bacterial stimulus causes hyper plasia or metaplasia of the superficial epithelium and indu ces mucosal edema with a cellular infiltrate. The resulting increase in the volume of the middle ear mucosa thus starts a vicious circle of deteriorating ventilation and drainage.

This hyperactivity of the middle ear mucosa continues after cessation of the external stimulus and ultimately leads to *tympanosclerosis.* Enzymes and the pathologic concentra tion of metabolic products and mediators of inflammation (lipoids, mucopolysaccharides) cause progressive metapla sia of the middle ear mucosa, resulting in fibrosis and scle rosis of the middle ear and formation of cholesterol gran ulomas. These changes lead to an irreversible tympanoscle rotic condition.

Basically, two main forms of disorders of ventilation and drainage can be distinguished: the first occurs acutely and is reversible, while the second follows a chronic course that is only partially reversible.

**Acute Tubal Occlusion (Serotympanum) Clinical Features.** A feeling of pressure in the ear occurs during a head cold (rhinopharyngitis), often accompanied by stabbing pain, deafness, and a crackling noise when swallowing.

**Diagnosis.** Otoscopy shows a retracted tympanic membrane (**Fig. 1.77a, b**) and hyperemia of the handle of the malleus and of the vessels of the tympanic membrane. If a transudate occurs, there is an amber discoloration of the pars tensa and occasionally a fluid level and air bubbles in the

*Clinical Aspects of Diseases of the Middle and Internal Ear* **59 **

**Fig. 1.77a, b** Serous otitis media. The tympanic mem brane is slightly retracted and transudate is visible in the tympanic cavity. The malleus handle is perceptibly short ened, the light reflex is widened.

middle ear. The patient also has mild to moderate conductive hearing loss.

**Differential diagnosis.** Acute otitis media has to be ruled out.

**Treatment.** This is initially directed at the under lying disease. Rhinopharyngitis is treated with de congestant nose drops, vasoconstrictors, and anti histamines to reduce hyperemia and edema in the tube. Antibiotics are given if there are dangerous signs of progression to otitis media or of acute nasal pharyngitis. Hypertrophic adenoids are removed later if necessary, and sinusitis is treated.

Note: Oral analgesics, rather than analgesic ear drops, should be used to treat pain in the ear. Ear drops obliter ate the otoscopic appearance of the tympanic mem brane by macerating the superficial epithelium and can make it difficult to recognize otitis media.

**Course and prognosis.** The symptoms usually re solve rapidly, but occasionally the disease pro gresses to chronic seromucinous otitis media.

Note: The Valsalva maneuver or air insufflation should not be performed when there is acute inflammation in the nasopharynx, due to the danger of transmitting infectious microorganisms to the middle ear and causing tubal otitis media.

**60 1 Ear**

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**Fig. 1.78a, b** Chronic seromucinous otitis media. The pars tensa of the tympanic membrane is protracted, while the pars flaccida is retracted. A bulb filled with yellow-brown liquid expands the tympanic membrane.

**Fig. 1.79** Different types 

of ear ventilation tube.

, Silver ear tubes

coated with gold, sizes I

and II; , Silastic tube for

long-term placement.

**Fig. 1.80** Middle ear 

drainage is achieved by in

serting a metal tube

grommet ).

**Chronic Seromucinous Otitis Media**

**Clinical Features.** There is a feeling of pressure and fullness in the ear, often following an infection of the upper airway, and there is a considerable de crease in hearing on one or both sides. Noises are heard in the ear when yawning, swallowing, and sneezing. Pain is absent.

**Pathogenesis.** Tubal obstruction and the resulting reduction in tympanic pressure predominate. Ob structive processes in the nasopharynxand disor ders of tubal kinetics, particularly incompetence of the muscles opening the tube in cleft palate, and viral infections are the most common underlying mechanisms.

exudate in the middle ear, and dark discoloration behind the tympanic membrane (the so-called blue drum ), with a blackish fluid level or air bub bles. There is conductive deafness for the entire frequency range up to 50 dB. A typical flat curve is found. A search for the primary cause shows en largement of the adenoids, sinusitis, allergy, or tu mor (**Fig. 1.78a, b**).

**Differential diagnosis.** This includes *hemotympa num* (the dark-brown exudate behind the tympanic membrane on occasion lends this a bluish tinge) and *chronic otitis media,* evidenced by perforation, cholesteatoma flakes, and purulent exudate. Adhe sions may occur after recurrent otitis, shown by a markedly retracted tympanic membrane with thick scars, tympanosclerotic deposits, and abnormal tu bal function. In adult patients with unilateral symp toms, biopsy from the nasopharynxshould be con sidered to exclude tumor; some tumors are submu cosal and not clearly apparent.

**Treatment.** If necessary, surgical restoration of tu bal patency by adenoidectomy or revision adenoid ectomy under direct vision, and elimination of any sinus infections.

Selective insertion of a ventilation tube after paracentesis and drainage of the middle ear (see **Fig. 1.84**), when clinically indicated by symptoms, is the treatment of choice.

An incision is made in the anteroinferior quadrant of the tympanic membrane, under general anesthesia in children and under local anesthesia in some compliant adults. The middle ear effusion is aspirated, and long-term drainage is provided for at least 6 months using a ventilation tube (or

grommet **Figs. 1.79, 1.80**). Alternatively, watchful wait ing can make it possible to avoid surgery, as the disease resolves in many cases. Adjuvant treatment consists of ste roids with antibiotic cover and mucolytic drugs. Other alter natives such as local aerosol treatment (intranasal or tubo tympanic application), -chymotrypsin (tubotympanic, transtympanic, or systemic administration), hyaluronidase, and corticosteroids (intratympanic application) are not generally used, but have been investigated in research stud ies. Antiallergic treatment includes antihistamines and intra nasal steroids if there is positive evidence of allergy. Acute otitis media is often present even if the immunological findings are normal. Mastoiditis has to be ruled out, other wise mastoidectomy should be considered.

**Diagnosis.** Otoscopy shows a markedly retracted tympanic membrane with localized protrusion, an

**Course and prognosis.** Long-term healing is only achieved in a proportion of patients. In others, there is a progressive, chronic course leading to adhesive processes as a result of connective-tissue organiza tion of the seromucinous exudate and development of cholesterol granuloma and tympanosclerosis. Hyaline degeneration of the mucoperiosteum may also occur, with the formation of sclerotic submu cosal plaques as a result of local metabolic distur bances (**Fig. 1.81a, b**).

**Syndrome of the Patulous Eustachian Tube Clinical Features.** These include *autophony,* which is a rumbling reverberation of the patient s own voice, and a noise in the ears synchronous with breathing, due to movements of the tympanic membrane and resonance in the nasopharynx.

**Pathogenesis.** The symptoms represent a masking effect of the lower and middle tones, evoked by resonance and respiratory noise. The primary cause is insufficiency of the closing mechanism of the tube (see p. 3). The secondary cause is disappear ance of the fat bolster around the opening of the tube, a gaping ostium caused by hormonal distur bances, and possibly also by use of the contracep tive pill.

**Diagnosis.** The diagnosis is made by impedance audiometry and tubal function tests.

**Treatment.** If possible, the basic cause is dealt with. The patient also needs to have the cause of the symptoms explained.

**Nonspecific Inflammation of the Middle Ear and Mastoid**

Note: Inflammatory diseases of the middle ear are im portant because of their frequency and the life-threat ening complications associated with them due to the close relationship between the middle ear and the cranial cavity.

**Acute Otitis Media**

**Clinical Features.** In the first phase of *exudative in flammation,* lasting for 1 2 days, there is an in crease in temperature to 39 40 C, and in severe

*Clinical Aspects of Diseases of the Middle and Internal Ear* **61 **

**Fig. 1.81a, b** Tympanosclerosis.

Hyaline degeneration of the mucoperiosteum occurs with the formation of sclerotic submucosal plaques. The posterior half of the tympanic membrane shows atrophic scarring, and plaque has formed on the anterior half.

cases, rigors, and occasionally meningism in chil dren. The patient has a severe pulsating pain, which is worse at night than during the day. There is a muffled noise in the ear synchronous with the pulse, deafness, and sensitivity of the mastoid pro cess to pressure. In older patients, there is often no fever.

The second phase, involving *resistance and de marcation,* lasts 3 8 days. The pus and middle ear exudate usually discharge spontaneously, after which the pain and fever subside. This phase can be considerably shortened by administering topical therapy (meatal cleansing, application of an astrin gent solution). Early antibiotic administration does not alter the clinical course of the disease signifi cantly, and it does not prevent spontaneous perfo ration of the tympanic membrane.

In the third, *healing* phase, lasting 2 4 weeks, the aural discharge dries up and hearing returns to normal.

Note: Acute middle ear inflammation may have a seri ous course, even if the tympanic membrane does not perforate.

**Pathogenesis.** *Routes ofinfection:* The tubal route is the most common. Hematogenous infection is un usual and occurs in measles, scarlet fever, typhus, and septicemia. Exogenous infection requires rup ture of the tympanic membrane or previous perfo ration, allowing bathwater or dirt to penetrate dur ing irrigation of the ear. Incorrect methods of re

**62 1 Ear**

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**Fig. 1.82a, b** Acute otitis media.

The tympanic membrane is erythematous and bulging, and the handle of the malleus shows hyperemia. The tympanic membrane also has radiating hyperemia.

moving a foreign body from the external meatus are also another cause.

Note: In healthy individuals, the middle ear is sterile if the tympanic membrane is intact.

*Type oforganism:* the infection is monomicrobial. In decreasing order of frequency, the infecting organ isms are: streptococci in adults, pneumococci in children, *Haemophilus influenzae, Moraxella catar rhalis,* and various staphylococci. A viral infection may prepare the way for secondary bacterial infec tion (herpes simplexand zoster, flu viruses). The inflammation usually affects not only the mucosa of the middle ear, but also that of the entire respira tory system.

Note: Every attack of acute otitis media is accompanied by mastoiditis.

**Diagnosis.** In the first phase, otoscopy shows hy peremia, then moist infiltration and opacity of the surface of the tympanic membrane. The contours of the handle of the malleus and its short process disappear (**Fig. 1.82a, b**). The patient has conduc tive deafness. At the height of the exudative phase, the tympanic membrane bulges, particularly its posterosuperior quadrant. Pulsation is also seen. The inflammation may extend to the external mea tus, obliterating the boundary between the meatus and the tympanic membrane (**Fig. 1.83a, b**). The accompanying mastoiditis makes the mastoid pro

**Fig. 1.83a, b** Acute otitis media (desquamative phase). The tympanic membrane is severely bulging and a livid red, partially blue-gray appearance with scales, as a result of severe desquamation.

cess tender to pressure. In influenzal otitis, hemor rhagic bullae form on the external auditory meatus and the tympanic membrane.

In the second phase of acute otitis media, imme diately before spontaneous rupture, a pinhole sized fistula forms, usually in the posterosuperior quadrant. This discharges a pulsating, thin, fluid, odorless pus. Radiography with the Sch ller view or preferably CT scanning shows clouding of the cell system without osteolysis i. e., the bony septa ap pear sharp. Imaging is indicated only if there are severe clinical symptoms (facial palsy, dizziness, vertigo, severe sensorineural hearing loss).

In the third phase of acute otitis media, the in flammation and thickening of the tympanic mem brane resolve, the pulsations disappear, and the discharge becomes mucoid and finally ceases. The perforation closes spontaneously, leaving a fine scar. Hearing returns to normal. CT scans show gradual clearing of the cell system.

**Differential diagnosis.** *Otitis externa* needs to be considered. In otitis externa, there is pain when pressure is applied to the tragus, and the exudate does not pulsate, is usually fetid, and is never mu coid. There is little or no deafness, and the cell system appears normal on radiographs.

**Treatment**

1. Systemic antibiotics at high dosages are given if condition worsens after 48 h. Hospital admis sion and intravenous application of amoxicillin and other broad-spectrum penicillins are indi cated. Culture and sensitivity tests are carried

*Clinical Aspects of Diseases of the Middle and Internal Ear* **63**

| B  **a**  A  **b** |
| --- |

**Fig. 1.84a, b** The principle of paracentesis.

The position of the myringot omy knife relative to the exter nal meatus and tympanic mem brane.

Incision into the anterior lower quadrant of the pars tensa. Correct incision; , alternative incision.

out and appropriate antibiotics are administered if the tympanic membrane perforates. 2. Nasal drops are given to decongest the nasophar yngeal mucosa around the opening of the tube. 3. Analgesics (liquid paracetamol is recommended for children) and mucolytic drugs can be given.

*Paracentesis* **Fig. 1.84a, b**), performed using a myr ingotomy with placement of a tympanostomy tube, is indicated in the following circumstances:

Marked bulging of the tympanic membrane. Persistent high fever and severe pain.

Unsatisfactory spontaneous perforation, with incomplete differentiation of the tympanic membrane.

If *symptoms ofearly mastoiditis* occur, with discrete facial palsy, acute meningitis, or labyrinthitis, and if the appearance of the tympanic membrane is in conclusive, surgery is indicated.

Note: Drops containing cortisone and antibiotic solu tion should not be used locally for aural discharge. They are ineffective, and also carry a risk of resistance to antibiotics developing. However, regular meatal cleansing should be performed for acute otitis media after the tympanic membrane perforates. The meatus can be irrigated with water at body temperature. The external meatus is then dried. Closing the meatus with cotton wool or gauze strips provides an ideal moist environment for inoculation with Gram-negative bacte ria or fungi, and the meatus therefore has to be kept open.

**Course and prognosis.** In the first, acute phase, there is a danger of early otogenic complications, depending on the virulence and resistance of the organism, until the patient s own resistance devel ops and the bacterial infection is controlled with antibiotics.

During the second phase, complications occur very rarely. However, latent otitis media and the resulting occult mastoiditis may develop during

**64 1 Ear**

this period due to insufficient antibiotic dosage, increased resistance of the organism, or inadequate resistance on the part of the patient. The general findings at otoscopy then do not correlate with the severity of the pathologic changes taking place in the middle ear and mastoid process. The paucity of symptoms leads to a false assumption that the otitis media has healed rapidly and completely. After an apparently symptom-free interval, late otogenic complications can occur in the third phase (see pp. 65 and 76). This course resembles that of the previously described, dreaded pneumococcal-type III mastoiditis.

In the third phase, most cases of acute otitis media and its concurrent mastoiditis heal com pletely. However, if latent otitis media and mastoi ditis become established in the second phase, late otogenic complications may develop in the third period i. e., 2 3 weeks after the start of the otitis. The symptoms include:

Reappearance of fever.

Recurrence of aural pain and discharge. Headaches.

Worsening of the patient s general condition. Elevated erythrocyte sedimentation rate (ESR).

**Specific Types of Inflammation of the Middle Ear and Mastoid**

**Acute Otitis M edia in Infants and Children** Severe forms are unusual nowadays, thanks to ef fective diagnosis and treatment with antibiotics. However, they can occur in patients with immune deficiency or after inappropriate therapy. They are characterized by:

Severe general symptoms, with high fever, me ningeal and cerebral irritation, vomiting, loss of appetite, and disturbance of sleep.

Immediate improvement of the child s condition after spontaneous perforation or paracentesis. A protracted course with numerous recurrences or exacerbations, with combined otitis media and bronchopneumonia, digestive and feeding upsets, and pyelonephritis.

Note: The younger the child, the more severe the gen eralized symptoms are and the more discrete the local signs are. The gastrointestinal symptoms are sometimes the most pressing.

Infants and young children have a predisposition to tubal middle ear infection because the tube is short, straight, and wide;because of the uniform character of the mucosa of the middle ear and the upper respiratory tract;and due to a higher frequency of infections of the respiratory tract, hyper plasia of the lymphoid tissue of the Waldeyer ring, poor aeration of the middle ear cavity, which is still partially filled with myxomatous tissue or hyperplastic mucosa, and a dif ference in the way in which the general and mucosal im mune system react, caused in part by the genotype and in part by the phenotype.

**Local symptoms.** These include pressure in the ear, tug ging on the affected ear, and painful reactions to pressure and traction. The tympanic membrane is grayish-red in color and bulges only slightly. Spontaneous perforation is not common;its site of predilection is the anteroinferior quad rant. Discharge is uncommon, as pus drains through the short, wide, and straight eustachian tube. If discharge oc curs, it is stringy and pulsating. Mucosal polyps may form in the middle ear;regional retroauricular lymphadenitis causes a swelling behind the ear. If the petrosquamosal fissure is open, the pus may penetrate directly from the middle ear beneath the periosteum, causing marked swelling behind the ear.

**Treatment.** Treatment consists of oral antibiotics. If the infection is severe, intravenous antibiotics are given. Decongestant nose drops and analgesics can also be administered. The ear is irrigated with phys iologic saline solution at body temperature.

*Paracentesis* (see **Fig. 1.84a, b**) should be carried out with the patient under general anesthesia early if the tympanic membrane does not perforate spon taneously.

If indicated on clinical grounds, *cortical mastoid ectomy* should be performed early, even if radio graphs are normal. *Cortical mastoidectomy* is car ried out under general anesthesia in infants and young children in whom the mastoid process is incompletely pneumatized or not pneumatized at all. The infected part of the mastoid process is cleared via a retroauricular access route, with wide opening and drainage of the mastoid antrum **Fig. 1.85**).

| 3  412  56 |
| --- |

**Fig. 1.85** The principle of cortical mastoidectomy. The posterior meatal wall (1) and attic wall (2) remain intact, while the cell system of the mastoid process (3) is cleared via a retroauricular approach. The anatomy of the external auditory meatus is not altered by this operation. 4, Sigmoid sinus;5, mastoid cavity;6, facial nerve (mastoid segment).

**Course.** The course is usually protracted, with in termittent exacerbations. There is often quick im provement and healing after surgical treatment of the affected ear. The prognosis is good with the correct treatment, but there is otherwise a danger of periantral osteomyelitis developing (infantile oc cult antritis), with vomiting and generalized toxic symptoms. In this case, immediate antrotomy is indicated.

**Specific forms.** *Influenzal otitis*: This is a hemorrhagic, bullous, acute middle ear inflammation. Primary infection with influenza A virus, combined with secondary bacterial infection *(Streptococcus pneumoniae)*, can take a fulminating course with complications (facial paralysis, labyrinthine irri tation, meningitis).

*Measles otitis*: Hematogenous viral otitis media with sub sequent tubal secondary infection, often leading to purulent mastoiditis.

*Scarlatinal otitis*: Acute necrotizing inflammation, with subtotal perforation of the tympanic membrane, necrosis of the ossicular chain, and osteomyelitis of the temporal bone.

*Clinical Aspects of Diseases of the Middle and Internal Ear* **65**

**Mastoiditis**

The most frequent complication of middle ear in flammation is *mastoiditis,* an extension of the in fection from the middle ear cavity to the pneumatic system of the temporal bone. In contrast to the mucosal inflammation, which always accompanies otitis media (see p. 61), the infection extends to, and causes dissolution of, bone. An unusually well pneumatized bone infection can extend to the pe trous pyramid (petrositis) and more rarely to the diploë of the temporal bone, causing osteomyelitis.

**Clinical Features.** Mastoiditis becomes manifest when there is a change in progress during resolu tion of acute otitis media.

*General:* Worsening of the general condition, rise in temperature, leukocytosis, and markedly in creased ESR.

*Local:* Increasing pain in the ear, synchronous with the pulse and radiating to the temporal bone and the occiput; reappearance or increase in the aural discharge, which is creamy, odorless, and purulent. The patient also has hearing loss.

**Pathogenesis.** Acute otitis media with concomitant mastoiditis usually resolves without complications. The development of complications depends on:

The anatomic relationships between the respi ratory system and middle ear space. Because of the narrow connection between the antrum and the mastoid cells, there is poor aeration from the eustachian tube.

The virulence and resistance of the organism. The local immune resistance of the mucosa. The patient s general immune resistance. The patient s general condition. Generalized dis eases such as diabetes, immunodeficiency, al lergy, and disorders of the liver and kidneys are important.

**Diagnosis**

Aural discharge.

Tenderness to pressure over the mastoid. Retroauricular swelling, with a protruding ear.

This classic triad of symptoms is now seldom seen, as otitis media is treated with antibiotics. This is especially true of the critical period that used to occur in the third week, during the preantibiotic era. The symptoms of mastoiditis are now more discrete and its course more insidious than they

**66 1 Ear**

**Fig. 1.86** Subperiosteal 

abscess, due to acute otitis

media with mastoiditis.

used to be, so that this complication is easily over looked. For this reason, the following otoscopic findings, which are also present during antibiotic treatment, need to be treated with caution:

A pale but still thickened tympanic membrane. Circumscribed inflammation and thickening of the tympanic membrane in the posterosuperior quadrant.

Thickened opaque tympanic membrane. Formation of a nipple on the tympanic mem brane, with a fine pinpoint fistulous opening. Prolapse of the posterior meatal wall, which oc curs relatively often in small children (see p. 2).

*Local findings over the mastoid process and in the surrounding area:*

*Subperiosteal abscess*: Soggy swelling of the skin is caused by edema due to spread of the infec tion. Reddening and a taut, elastic, fluctuating swelling of the skin over the mastoid process **Fig. 1.86**).

*Zygomatic bone inflammation*: There is swelling of the zygomatic process, with extension to the cheek and eyelids in an extensively pneuma tized bone. This is relatively common in chil dren.

*Bezold mastoiditis*: Visible and palpable tender swelling of the lateral triangle of the neck, with torticollis. This is due to an abscess tracking from the mastoid apexin the fascial spaces of the digastric, sternocleidomastoid, splenius, and longissimus capitis muscles.

*Radiographic signs* in the Sch ller view and/or CT of the temporal bone show a decrease in radio lucency due to a reduction in the air content in the respiratory system, haziness and opacity of the mastoid cells, haziness of the fine bony structures as a result of decalcification and lique faction of the bony septa between the cells, bone

destruction with foci of liquefaction, and erosion of neighboring structures.

**Differential diagnosis (pseudomastoiditis) (see p. 51).** Furuncle of the external ear canal, parotitis, or cervical lymphadenitis.

**Treatment**

Note: Mastoiditis in which the inflammation is no longer confined to the mucosa but has extended to the bone should be treated surgically.

It is incorrect to assume that inflammation of the pneumatic system of the temporal bone can be cured by antibiotics once it has invaded the bony structures. The poor vascular ization of the mucosa and bone makes it impossible to maintain a satisfactory concentration of antibiotic in the tissues. The poorly aerated cells, filled with hyperplastic mucosa and granulations, are an ideal culture medium for bacteria, particularly anaerobes.

*Indications for mastoidectomy:* The operation is indicated if bone infection is thought to be present at any stage of otitis media:

Symptoms of otogenic intracranial complica tions.

Signs of a subperiosteal collection.

A focus of liquefaction of mastoid cells on CT scans of the temporal bone.

Facial paralysis.

In the healing phase i. e., in the critical third week of otitis media the operation is also indicated when there is:

Recurrence of aural discharge, pain, and subfe brile temperature.

Worsening of the general condition and an in crease in CRP.

Aural discharge persisting for 4 5 weeks and resisting treatment, in the presence of good pneumatization of the temporal bone, with re duced air content on radiographs, and without serious generalized symptoms.

*Principle ofmastoidectomy:* The diseased tissue in the cell system of the mastoid process is excised via a retroauricular skin incision under general anes thesia. A wide connection is created between the mastoid antrum and the mastoid cavity, and from the latter to the middle ear cavity (see **Fig. 1.85**).

**Course and prognosis.** Two forms of true mastoidi tis with bone destruction can develop from the mastoiditis accompanying otitis media, even dur ing antibiotic treatment:

*Acute mastoiditis:* This is marked by purulent liquefaction of the bony septa of the pneumatic system, with external rupture to form a subpe riosteal or retroauricular abscess. The course is rapid and the symptoms marked. Paralysis of the facial nerve is possible.

*Chronic mastoiditis:* This is partly a productive inflammation, with obliteration of the spaces of the pneumatic system by inflammatory granu lation tissue; and partly a continuous inflamma tory breakdown of bone. The course is therefore insidious, and the symptoms are initially mild.

The prognosis is good with correct treatment, but otherwise there is a danger of late otogenic com plications (see p. 76).

**Chronic Otitis Media**

***Chronic Mucosal Inflammation (Chronic***

***Mesotympanic Otitis)***

**Clinical Features.** *There is a chronic discharge of mucoid, purulent, odorless exudate.* The otitis is as sociated with periods of complete freedom from symptoms, alternating with acute exacerbations. The exudate may be creamy and purulent in the acute phase and then becomes mucoid and stringy as the infection resolves. However, it is always odor less.

*Hearing:* The patient has conductive hearing loss. Pain is absent, and the general condition is good.

**Pathogenesis.** The disease is not one that has a single cause, but rather it is the end result of several different primary disease processes. The inflamma tion remains confined to the mucosa, but in certain patients it can lead in time to rarefying osteitis i. e., chronic inflammatory destruction of the ossicles, such as the long process of the incus. In contrast to cholesteatoma, this destructive bone process is unusual and less likely to extend and progress. Vascular obliteration can occur due to heavy scar tissue deposits in the vascular subepithelial con nective-tissue layer, leading secondarily to nutri tional disturbances of the neighboring bony tissue (aseptic bone necrosis).

*Clinical Aspects of Diseases of the Middle and Internal Ear* **67 **

**Fig. 1.87a, b** Chronic otitis media (mesotympanic). There is a large central perforation in the tympanic membrane, with only a thin edge left. The promontory is visible through the perforation. The long process of the incus, the stapes tendon, and the round window niche are visible.

*Pathogenetic factors:*

Constitutionally reduced mucosal (immunolog ical) competence (see p. 7).

Type, pathogenicity, virulence, and resistance of the bacterial organisms.

Anatomic conditions in the middle ear, such as pneumatization, and connections between the attic, antrum, middle ear cavity, and eustachian tube.

Disordered function of the eustachian tube e. g., in patients with cleft palate.

Generalized diseases such as allergy, immune defects, cachexia, and diabetes.

**Diagnosis.** The *history* shows a chronic recurrent aural discharge with reduced hearing. The *otoscopic findings* include a central defect of the tympanic membrane (**Fig. 1.87a, b**), scarring of the pars tensa, and occasionally aural polyps due to mucosal hy perplasia in acute exacerbations.

CT scanning (as well plain Sch ller radiographs) show either reduced pneumatization or opacity of the cell system, if it is well pneumatized; occasion ally, signs of bony destruction and formation of new bone (sclerosis) are seen. These are regarded as signs of chronic mastoiditis.

The *audiogram* shows conductive hearing loss.

**Differential diagnosis.** *Cholesteatoma* is associated with a marginal defect in the tympanic membrane and a fetid discharge.

*Aural tuberculosis* shows several central perfo rations of the tympanic membrane, with marked hearing loss.

**68 1 Ear**

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**Fig. 1.88a, b** Chronic otitis media (epitympanic). The mar ginal defect in the tympanic membrane in the posterosupe rior pars tensa and in the pars flaccida is causing osteitis in the bony lateral wall of the epitympanum. Granulations or polyps are typical signs.

*Middle ear carcinoma* causes a marginal defect with exuberant tissue extending into the meatus, and bone destruction in the attic and the meatal wall.

**Treatment.** *Conservative measures for drying the middle ear:* The external meatus is cleaned periodi cally. It can be irrigated with physiologic saline at body temperature. In the acute phase, pus is taken for culture and sensitivity tests, and appropriate systemic and local antibiotics are given, with care being taken not to use ototoxic drugs.

Aural polyps are removed with a wire snare or microforceps. Chronic infection of the nasopharynx and paranasal sinuses must be looked for.

*Surgery:* Mastoidectomy can be carried out to eliminate foci of infection in the temporal bone and middle ear cavity. Tympanoplasty can be per formed to reconstruct the sound-conducting appa ratus i. e., the tympanic membrane and ossicular chain.

**Course and prognosis.** The course is episodic, with exacerbations caused by exogenous infection e. g., from bathwater and tubal infection. Complica tions are very rare. Progression to cholesteatoma is exceptional, and hearing loss is usually progres sive. The prognosis is good in relation to survival, but poor with regard to function. An early tympa noplasty should therefore be carried out after in tensive preparation (see **Figs. 1.95, 1.96**).

Age in itself is not a contraindication to surgical treat ment. The key objective of the operation is to close the perforation, to avoid recurrent infection of the middle ear

mucosa via the external ear canal. Improvement of hearing can only be achieved in a small proportion of the children, and this should be taken into account and discussed during the process of obtaining consent for the operation.

Every patient presenting with active mucosal inflammation with frequent infections that do not settle with conservative medical management should be treated as early as possible using *tympa noplasty*, since the extent of the destruction of the sound-conducting apparatus increases with every inflammatory episode. If a hearing aid is necessary, tympanoplasty is also indicated, since chronic otor rhea makes it impossible to wear an earpiece.

Note: Chronic mucosal inflammation is a form of chronic otitis media in which the inflammation is mainly confined to the mucosa. It does not usually cause pro gressive bone destruction and is therefore free of com plications, but it has a protracted course.

***Chronic Bone Suppuration (Chronic Epitympanic Otitis Media)***

As a result of a marginal defect of the tympanic membrane in the posterior superior pars tensa or in the pars flaccida, inflammation spreads to parts of the bony lateral wall of the epitympanum. Gran ulations or polyps are typical signs of granulating ostitis (**Fig. 1.88a, b**).

***Acquired Cholesteatoma of the Middle Ear*** A cholesteatoma is a skin growth that occurs in the middle ear. It is usually due to repeated infection, which causes ingrowth of the skin of the eardrum. Progressive inflammation develops on the basis of a marginal tympanic membrane perforation, with osteitis on the lateral epitympanic wall. Cholestea tomas often take the form of a cyst or pouch that sheds layers of old skin, which builds up inside the middle ear.

**Clinical Features**

Fetid otorrhea that is sometimes minimal or completely absent; when present, it is always purulent, and never mucoid.

Progressive hearing loss, possibly dizziness. Otalgia and fever in acute exacerbations. Dull headaches or a feeling of pressure in the head.

**Pathogenesis**

Note: An acquired middle ear cholesteatoma is not a tumor, but rather a chronic inflammation which, unlike chronic mucosal inflammation, causes progressive de struction of the bony cells and structures.

*Promoting factors:*

Disordered ventilation and drainage of the mid dle ear (chronic reduction of pressure) with hypopneumatization.

Displaced squamous epithelium as a result of increased capacity for growth of the meatal skin in the upper part of the anulus tympanicus (the papillary ingrowths form the later *matrix,* either as a result of invagination of the pars flaccida or by formation of a retention pocket in the pars tensa).

An increased proliferative tendency in the stra tum germinativum (see **Fig. 1.7**), caused by the stimulus of inflammation.

Incompletely resolved embryonic hyperplastic mesenchymal remnants in the submucosa of the middle ear, which later form the *perimatrix.*

*Histopathogenesis:* A cholesteatoma may form a compact sac of desquamated lamellae, arranged like the layers of an onion and connected with a fairly thick pedicle to its site of origin in the tympanic membrane (the pars flaccida or tensa). Alternatively, it may consist of a widely fanned-out cholesteatoma matrix lining the antrum and mastoid cavity and sending off shoots into the furthest bony niches of the bony process. The latter type of cholesteatoma therefore has a reticular or dendritic, branched structure. The latter occurs more commonly in a tensa cholesteatoma than in a flaccida cholesteatoma. Bone destruction is caused firstly by enzymes (e. g., collagenase) formed in the perimatrix, and secondly by osteoclastic destruction of bony tissue i. e., chronic osteomyelitis.

Note: A prerequisite for the development of a choles teatoma is direct contact between the keratinizing squa mous epithelium in the external meatus and mucoper iosteum of the middle ear that has been damaged by inflammation.

This can happen as a result of:

*marginal perforation* with a destruction of the protective barrier of the anulus fibrosus.

*Clinical Aspects of Diseases of the Middle and Internal Ear* **69**

*Papillary ingrowths* e. g., in the region of the pars flaccida or of the destroyed anulus fibrosus. Formation of a *retraction pocket* in the pars tensa.

*Traumatic displacement* of keratinizing squa mous epithelium after longitudinal pyramidal fractures or ruptures of the tympanic mem brane, with the development of posttraumatic otitis media.

The same pathogenetic factors (see p. 67) are in volved in the genesis of cholesteatoma as to that of chronic mucosal inflammation.

**Diagnosis.** An inflammatory middle ear cholestea toma can be classified from several different points of view (primary, secondary, or topographic anat omy). The method using the site of origin appears to be the most sensible from the diagnostic point of view and clearer than the other classifications. Cho lesteatoma can thus be classified as follows:

*Tensa cholesteatoma* (synonym: secondary mid dle-ear cholesteatoma).

*Flaccida cholesteatoma* (synonyms: primary or genuine attic or epitympanic cholesteatoma). *Occult cholesteatoma* (synonyms: cholesteatoma behind an intact tympanic membrane, or con genital cholesteatoma).

The *tensa cholesteatoma* **Fig. 1.89a, b**) develops from a retraction pocket caused by chronic inflam mation, usually in the posterosuperior quadrant of the pars tensa. It is characterized by a posterosupe rior marginal perforation. Inflammatory granula tions, fetid exudate with flakes of cholesteatoma, and circumscribed destruction of the surrounding posterosuperior meatal wall are often found in the region of the edge of the perforation. Conductive hearing loss is also present, due to destruction of the ossicular chain. The site of predilection is the incudostapedial joint.

The *flaccida cholesteatoma* **Fig. 1.89c, d; Fig. 1.90a c; Fig. 1.91a, b**) arises from papillary in growth of the keratinizing squamous epithelium in the region of the Shrapnell membrane, in the presence of simultaneous chronic epitympanitis. The stimulus of inflammation induces increased proliferation of the squamous epithelium. Circum scribed perforation of the pars flaccida, often cov ered by a crust and usually also accompanied by destruction of the lateral attic wall, is therefore