

UNIT 14

Sensory Function

Case Study

MAKING A RAPID CHANGE TO TELEHEALTH



A 72-year-old female is due for a follow-up appointment at the outpatient ophthalmology clinic where you work. She has a history of type 2 diabetes, hypertension, and elevated cholesterol. During

her previous visit, she reported her vision was blurred when watching television and that she stopped driving after dark as she could not see properly; she was subsequently diagnosed with diabetic retinopathy. However, there is an outbreak of coronavirus disease 2019 (COVID-19) within your community; the health network system that manages the ophthalmology clinic has responded by mandating that telehealth be used through video conference service to manage all patients for clinic visits. How will you assist the clinic staff as well as the patients, particularly this patient with poor vision, to make a rapid transition to telehealth visits while also assuring continued quality outcomes?

QSEN Competency Focus: **Informatics**

The complexities inherent in today's health care system challenge nurses to demonstrate integration of specific interdisciplinary core competencies. These competencies are aimed at ensuring the delivery of safe, quality patient care (Institute of Medicine, 2003). The Quality and Safety Education for Nurses project (Cronenwett, Sherwood, Barnsteiner, et al., 2007; QSEN, 2020) provides a framework for the knowledge, skills, and attitudes (KSAs) required for nurses to demonstrate competency in these key areas, which include **patient-centered care, interdisciplinary teamwork and collaboration, evidence-based practice, quality improvement, safety, and informatics.**

Informatics Definition: Use information and technology to communicate, manage knowledge, mitigate error, and support decision-making.

SELECT PRE-LICENSURE KSAs

APPLICATION AND REFLECTION

Knowledge

- | | |
|---|---|
| Identify essential information that must be available in a common database to support patient care | Transitioning to telehealth in a short period of time can be challenging. How can you help communicate essential information and streamline the process of change? |
| Contrast benefits and limitations of different communication technologies and their impact on safety and quality | What are the benefits and limitations of using telehealth, especially with patients with low vision? |
| Describe examples of how technology and information management are related to the quality and safety of patient care | Describe how the implementation of telehealth in an outpatient clinic will increase the quality and safety for the patients who receive care there during a pandemic. |
| Recognize the time, effort, and skill required for computers, databases, and other technologies to become reliable and effective tools for patient care | How does the use of telehealth help the team in the ophthalmology clinic improve outcomes for patients? |

Skills

- | | |
|--|--|
| Seek education about how information is managed in care settings before providing care | How can you influence the safe and effective implementation of telehealth? |
| Apply technology and information management tools to support safe processes of care | How can you support safe processes of care utilizing this new technology? |
| Navigate the electronic health record | How will the telehealth system be integrated with the existing electronic health record and used to monitor outcomes of care processes? |
| Document and plan patient care in an electronic health record | Patients must now navigate the video conference system through the existing patient portal for follow-up visits. How will you communicate these changes to the ophthalmology clinic's mostly older adult patients, many of whom have poor vision and may feel challenged utilizing these types of technologic platforms? |
| Employ communication technologies to coordinate care for patients | |
| Respond appropriately to clinical decision-making supports and alerts | |
| Use information management tools to monitor outcomes of care processes | |

Attitudes

Value technologies that support clinical decision-making, error prevention, and care coordination

Reflect upon how rapid change is implemented within an organization during times of a pandemic. How can you find value in the use of telehealth for clinical decision-making, prevention of errors, and coordination of care?

Cronenwett, L., Sherwood, G., Barnsteiner, J., et al. (2007). Quality and safety education for nurses. *Nursing Outlook*, 55(3), 122–131; Institute of Medicine. (2003). *Health professions education: A bridge to quality*. Washington, DC: National Academies Press; QSEN Institute. (2020). *QSEN competencies: Definitions and pre-licensure KSAs; Informatics*. Retrieved on 8/15/2020 at: qsen.org/competencies/pre-licensure-ksas/#informatics

58 Assessment and Management of Patients with Eye and Vision Disorders

LEARNING OUTCOMES

On completion of this chapter, the learner will be able to:

- 1.** Identify the major internal and external structures and functions of the eye.
- 2.** Specify assessment and diagnostic findings used in the evaluation of ocular disorders.
- 3.** Describe assessment and management strategies for patients with low vision and blindness.
- 4.** List the pharmacologic actions and nursing management of common ophthalmic medications.
- 5.** Recognize the clinical features, assessment and diagnostic findings, as well as the medical or surgical management, and nursing management of the patient with glaucoma, cataracts, and other ocular disorders.

NURSING CONCEPTS

Assessment
Comfort
Infection
Inflammation
Patient Education
Sensory Perception

GLOSSARY

anterior chamber: aqueous-containing space in the eye between the posterior (endothelial) cornea and the anterior iris and pupil

aqueous humor: transparent, nutrient-containing fluid that fills the anterior and posterior chambers of the eye

astigmatism: refractive error due to an irregularity in the curvature of the cornea

binocular vision: normal ability of both eyes to focus on one object and fuse the two images into one

blindness: inability to see, defined as corrected visual acuity of 20/400 or less, or a visual field of no more than 20 degrees in the better eye

cataract: progressive opacity of the lens of the eye

chemosis: edema of the conjunctiva

diplopia: seeing one object as two (*synonym:* double vision)

ectropion: turning out of the lower eyelid

emmetropia: normal refractive condition resulting in clear focus on retina; no optical defects

endophthalmitis: intraocular infection

entropion: turning in of the lower eyelid

enucleation: removal of the eyeball and part of the optic nerve

evisceration: removal of the intraocular contents through a corneal or scleral incision; the optic nerve, sclera, extraocular muscles, and sometimes the cornea are left intact

exenteration: surgical removal of the entire contents of the orbit, surrounding soft tissue, and most or all of the eyelids

exophthalmos: abnormal protrusion of the eyeball (*synonym:* proptosis)

glaucoma: group of conditions characterized by increased intraocular pressure

hyperemia: red eyes resulting from dilation of the vasculature of the conjunctiva

hyperopia: farsightedness; light rays focus behind the retina

hyphema: blood in the anterior chamber

hypopyon: collection of inflammatory cells in the anterior chamber of the eye

injection: congestion of blood vessels

keratoconus: cone-shaped deformity of the cornea

myopia: nearsightedness; light rays focus in front of the retina

neovascularization: growth of abnormal new blood vessels

nystagmus: involuntary oscillation of the eyeball

papilledema: swelling of the optic disc usually due to increased intracranial pressure

photophobia: ocular pain on exposure to light

presbyopia: the loss of accommodative power in the lens due to age

ptosis: drooping eyelid

refraction: determination of the refractive errors of the eye for the purpose of vision correction

scotomas: blind or partially blind areas in the visual field

sympathetic ophthalmia: an inflammatory condition created in the fellow eye by the affected eye

trachoma: an infectious disease caused by the bacterium *Chlamydia trachomatis*—the leading cause of preventable blindness in the world

trichiasis: turning in of the eyelashes

vitreous humor: transparent, colorless gelatinous material that fills the vitreous chamber behind the lens

The eye is a sensitive, highly specialized sense organ subject to various disorders, many of which can lead to impaired vision. Impaired vision may affect individuals in many ways, including their independence in self-care, sense of self-esteem, safety, and overall quality of life. Many of the leading causes of visual impairment are associated with aging (e.g., cataracts, glaucoma, macular degeneration). Younger people are also at risk for eye disorders, particularly traumatic injuries.

Assessment and management of patients with eye and vision disorders occur in various health care settings. In addition to understanding the prevention, treatment, and consequences of eye disorders, nurses in all settings assess visual acuity in patients at risk (e.g., older adults, those with hypertension, diabetes, acquired immune deficiency syndrome [AIDS]), refer patients to eye care specialists as appropriate, implement measures to prevent further visual loss, and help patients adapt to impaired vision.

ASSESSMENT OF THE EYE

Anatomic and Physiologic Overview

Unlike most organs of the body, the eye is available for external examination, and its anatomy is more easily assessed than other body parts (see Fig. 58-1). The eyeball, or globe, is situated in the bony protective orbit (Shaw & Lee, 2017). Lined with muscle and connective and adipose tissues, the orbit is shaped like a four-sided pyramid, surrounded on three sides by the sinuses: ethmoid (medially), frontal (superiorly), and maxillary (inferiorly). The optic

nerve and the ophthalmic artery enter the orbit at its apex through the optic foramen. The eyeball is moved through all fields of gaze by the extraocular muscles. The four rectus muscles and two oblique muscles (see Fig. 58-2) are innervated by cranial nerves (CNs) III, IV, and VI. Normally, the movements of the two eyes are coordinated and the brain perceives a single image.

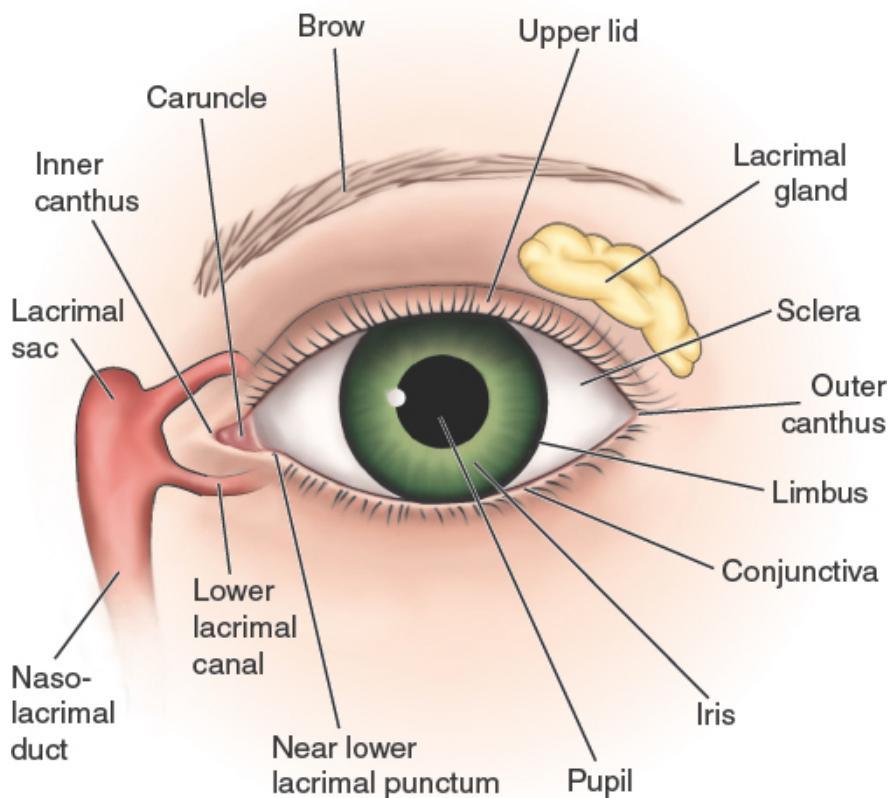


Figure 58-1 • External structures of the eye and position of the lacrimal structures.

The eyelids are composed of thin, elastic skin that covers striated and smooth muscles and protect the anterior portion of the eye. The eyelids contain multiple glands (sebaceous, sweat, and lacrimal). The upper lid normally covers the uppermost portion of the iris and is innervated by the oculomotor nerve (CN III). The lid margins contain meibomian glands, the inferior and superior puncta, and the eyelashes. The triangular spaces formed by the junction of the eyelids are known as the inner or medial canthus and the outer or lateral canthus. With every blink, the eyelids wash the cornea and conjunctiva with tears.

Tears are vital to eye health. Formed by the lacrimal gland and the accessory lacrimal glands, tears are secreted in response to reflex or emotional stimuli. A healthy tear is composed of three layers: lipoid, aqueous, and

mucoid. These layers nourish the cornea and create a smooth optical surface of the cornea and conjunctival epithelium. If there is a defect in the composition of any of these layers, the integrity of the cornea may be compromised.

The conjunctiva, a thin transparent mucous membrane, provides a barrier to the external environment extending under the eyelids (palpebral conjunctiva) and over the sclera (bulbar conjunctiva). The junction of the two portions is known as the fornix. The conjunctiva meets the cornea at the limbus on the outermost edge of the iris.

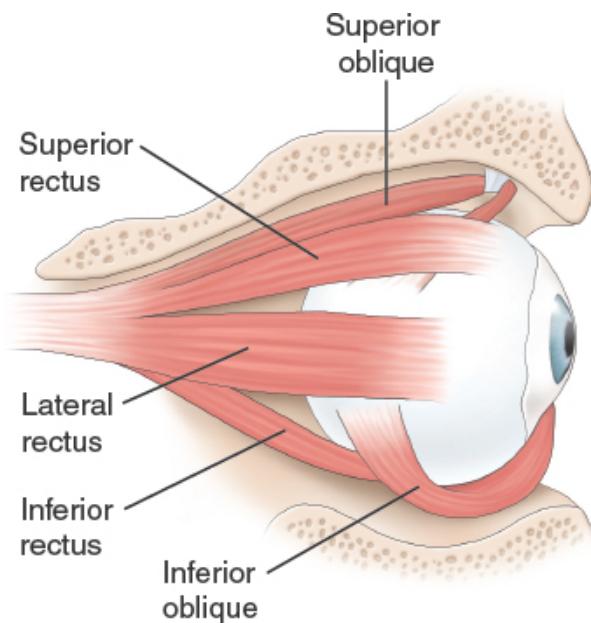


Figure 58-2 • The extraocular muscles responsible for eye movement. The medial rectus muscle (not shown) is responsible for opposing the movement of the lateral rectus muscle.

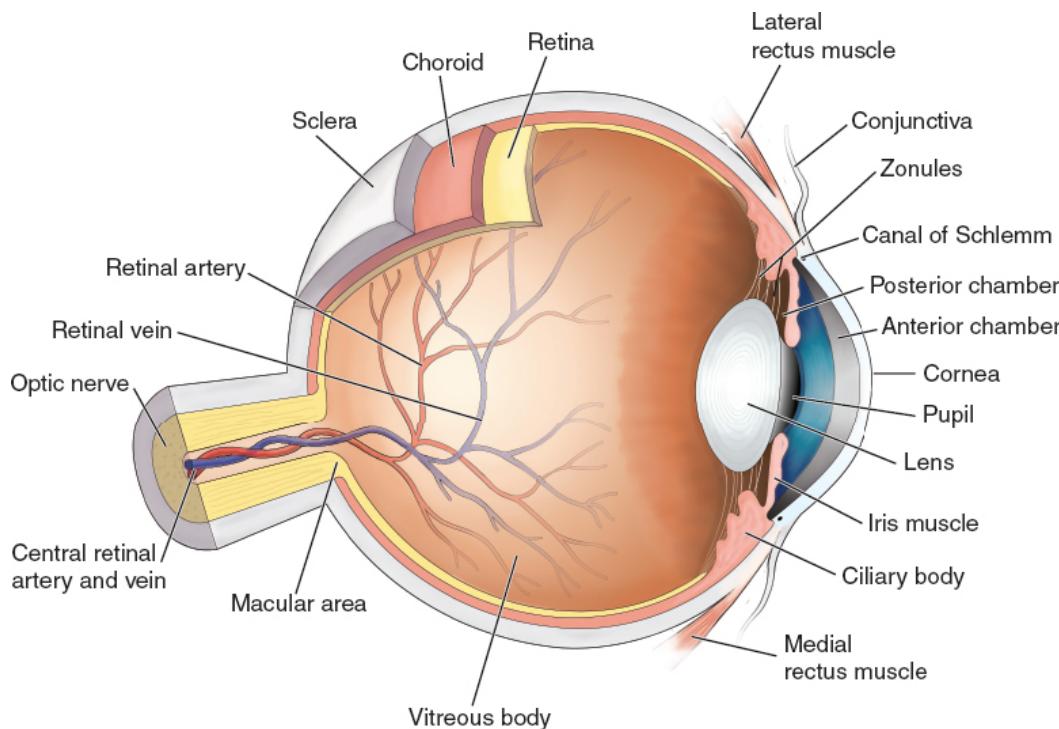


Figure 58-3 • Three-dimensional cross-section of the eye.

The eyeball is composed of the following three layers:

- The outer dense fibrous layer, including the sclera and transparent cornea
- The middle vascular layer, containing the iris, ciliary body, and choroid
- The inner neural layer, including the retina, optic nerve, and visual pathway

The eyeball is divided anatomically into two segments. The anterior segment is between the anterior cornea and posterior iris, including the anterior and posterior chambers. The posterior segment is between the posterior lens and the retina, including the vitreous chamber. The eyeball also has three fluid-containing chambers. The aqueous-filled **anterior chamber** lies between the posterior cornea and the anterior iris and pupil. The posterior chamber is a small aqueous-containing space between the posterior iris and pupil and anterior lens. The vitreous chamber, containing clear gelatinous vitreous fluid, is the largest chamber in the ocular fundus between the lens and retina (see Fig. 58-3).

The **aqueous humor** is transparent, nutrient-containing fluid that fills the anterior and posterior chambers and helps give the eye its shape (Moore, Dalley, & Agur, 2018). The aqueous is produced in the posterior chamber by the ciliary body; it flows through the pupil into the anterior chamber and drains through the trabecular meshwork into the canal of Schlemm. Production of aqueous humor is related to the intraocular pressure (IOP). Normal IOP is less than 21 mm Hg (Sihota, Angmo, Ramaswamy, et al., 2018). **Vitreous humor,**

which is composed mostly of water and encapsulated by a hyaloid membrane, helps maintain the shape of the eye. The vitreous is attached to the retina by scattered collagenous filaments. The vitreous shrinks and shifts with age. Through this degenerative process, the gel-like characteristics liquefy, causing stringy debris known as floaters.

The sclera is the white avascular dense fibrous structure that helps maintain the shape of the eyeball and protects the intraocular contents. Scleral thinning and changes of the scleral collagen fibers can cause the underlying uveal pigment to be seen, resulting in a blue or gray sclera. The episclera is a vascularized loose elastic tissue that overlies the sclera supplying nutritional support and reacting to inflammation.

The cornea, a vulnerable transparent avascular domelike structure, forms the most anterior portion of the eyeball and is the main refracting surface of the eye. It is composed of five layers: the epithelium, Bowman's membrane, stroma, Descemet's membrane, and endothelium. It contains high concentrations of nerve fibers and is extremely sensitive to pain. The epithelium, the outermost protective layer, absorbs oxygen and nutrients from the tear film nourishing the cornea. The epithelial cells readily regenerate, unlike the innermost endothelial cells, which do not regenerate and result in corneal edema when injured.

The uveal tract is the vascular middle layer of the eye consisting of the iris, ciliary body, and the choroid.

The iris surrounding the pupil is a highly vascularized pigmented collection of fibers that give the eye color. The dilator and sphincter muscles of the iris control pupil size. The dilator muscles are controlled by the sympathetic nervous system. The sphincter muscles are controlled by the parasympathetic nervous system.

The ciliary body consists of ciliary processes, ciliary muscles, and zonular fibers (ligaments) that work together to form aqueous fluid and control focusing through the zonular fibers that suspend the crystalline lens.

The choroid lies between the retina and the sclera, supplying blood and oxygen to the outer retina. Pigmented cells containing melanocytes in the choroid assist to absorb scattered light.

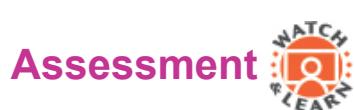
Directly behind the pupil and iris lies the lens, an avascular and almost completely transparent biconvex structure held in position by zonular fibers in the ciliary body. The lens enables focusing for near and distance vision through accommodation, the process by which the lens of the eye adjusts the focal length to focus a clear image on the retina. With aging and certain conditions (e.g., diabetes or trauma), the lens loses its transparency and ability to focus due to formation of a cataract (see later discussion).

The retina—the innermost surface of the fundus composed of neural tissue—is an extension of the optic nerve. Viewed through the pupil, the landmarks of the retina are the optic disc, the retinal vessels, and the macula. The point of

entrance of the optic nerve into the retina is the optic disc. The optic disc is pink, either oval or circular in shape, and has sharp margins. In the disc, a physiologic depression or cup is present centrally, with the retinal blood vessels emerging from it. The retinal tissues arise from the optic disc and line the inner surface of the vitreous chamber. The retinal vessels enter the eye through the optic disc, branching out through the retina and forming superior and inferior branches. The macula is the area of the retina responsible for central vision. The rest of the retina is responsible for peripheral vision. In the center of the macula is the most sensitive area—the fovea—which is avascular and surrounded by the superior and inferior vascular arcades. Two important layers of the retina are the retinal pigment epithelium and the sensory retina. A single layer of cells constitutes the retinal pigment epithelium. These cells have numerous functions, including the absorption of light. The sensory retina contains the photoreceptor cells: rods and cones. The rods are responsible for night or low light vision. The cones are retinal photoreceptor cells essential for visual acuity, color discrimination, and fine detail. Cones are distributed throughout the retina, with their greatest concentration in the fovea. Rods are absent in the fovea.

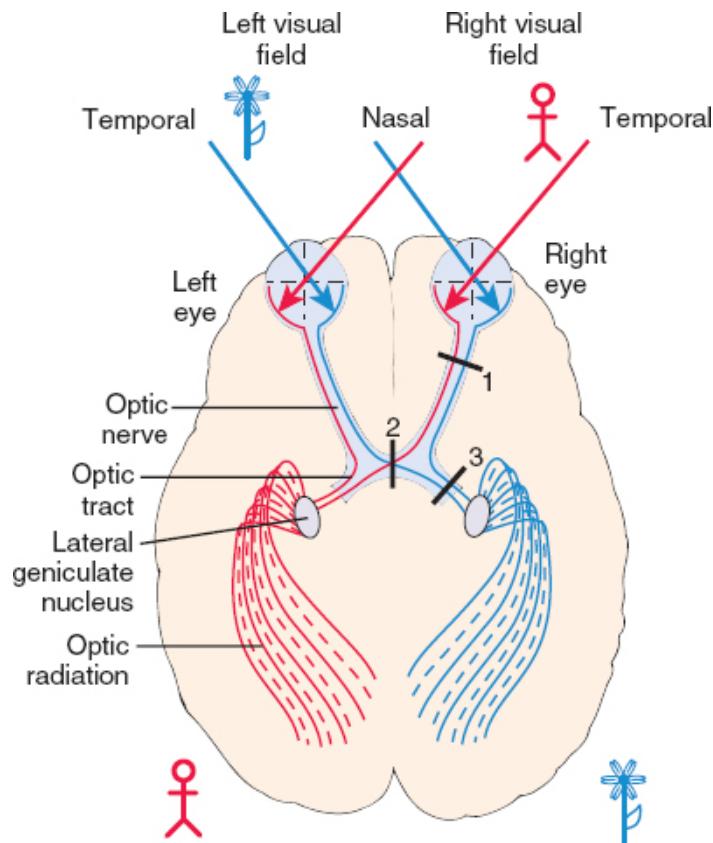
Visual acuity depends on a healthy functioning eye and an intact visual pathway. This pathway is made up of the retina, optic nerve, optic chiasm, optic tracks, lateral geniculate bodies, optic radiations, and the visual cortex area of the brain. The visual pathway is part of the central nervous system (see Fig. 58-4).

The optic nerve (CN II) transmits impulses from the retina to the occipital lobe of the brain. The optic nerve head, or optic disc, is the physiologic blind spot in each eye. The optic nerve leaves the eye and then meets the optic nerve from the other eye at the optic chiasm. The chiasm is the anatomic point at which the nasal fibers from the nasal retina of each eye cross to the opposite side of the brain. The nerve fibers from the temporal retina of each eye remain uncrossed. Fibers from the right half of each eye, which would be the left visual field, carry impulses to the right occipital lobe. Fibers from the left half of each eye, or the right visual field, carry impulses to the left occipital lobe. Beyond the chiasm, these fibers are known as the optic tract. The optic tract continues on to the lateral geniculate body. The lateral geniculate body is connected by the optic radiations to the cortex of the occipital lobe of the brain.



Ocular History

The nurse, through careful questioning, elicits the necessary information that can assist in diagnosis of an ophthalmic condition. Pertinent questions to ask when taking an ocular history are presented in [Chart 58-1](#). Genetics may play a role in the causation and progression of eye and vision disorders (Singh & Tyagi, 2018) (see [Chart 58-2](#)).



Whitened field
no vision

Left Right

Lesion 1 Right optic nerve

Lesion 2 Optic chiasm

Lesion 3 Right optic tract

Figure 58-4 • Diagram of optic pathways. The *red* lines indicate the right visual field and the *blue* lines the left visual field. Note the crossing of the fibers from the medial half of each retina at the optic chiasm. Lesion 1 (right optic nerve) produces unilateral blindness. Lesion 2 (optic chiasm) may involve only those fibers that originate in the nasal half of each retina and cross to the opposite side in each field (bitemporal hemianopia). Lesion 3 (right optic tract) interrupts fibers (and vision) originating on the same side of both eyes (homonymous) with loss of vision from half of each field (hemianopia). Reprinted with permission from Norris, T. L. (2019).

Porth's pathophysiology: Concepts of altered health states (10th ed., Fig. 19.23). Philadelphia, PA: Wolters Kluwer.

Visual Acuity

Following the health history, the patient's visual acuity is assessed. This is an essential part of the eye examination and a measure against which all therapeutic outcomes are based.

Visual acuity is tested for both near (14 inches away) and distance (20 feet away) vision and is performed on each eye separately with a standardized Snellen chart for distance and a Rosenbaum pocket screener for near vision. A tumbling "E," "illiterate E," number, or picture chart is used if the person is illiterate or unable to read the English alphabet (Weber & Kelley, 2018).

Chart 58-1



ASSESSMENT

Taking a History of Patients with Eye and Vision Disorders

- What does the patient perceive to be the problem?
- Is visual acuity diminished?
- Is the patient experiencing blurred, double, or distorted vision?
- Is there pain? Is it sharp or dull? Is it worse when blinking?
- Is the discomfort an itching sensation or more of a foreign-body sensation?
- Are both eyes affected?
- Is there a history of discharge? If so, inquire about color, consistency, and odor.
- Describe the onset of the problem (sudden, gradual). Is it worsening?
- What is the duration of the problem?
- Is this a recurrence of a previous condition?
- How has the patient self-treated?
- What makes the symptoms improve or worsen?
- Has the condition affected performance of activities of daily living?
- Are there any systemic diseases? What medications are used in their treatment?
- What other eye conditions does the patient have?
- Is there a history of eye surgery?
- Have other family members had the same symptoms or condition?

Adapted from Weber, J., & Kelley, J. (2018). *Health assessment in nursing* (6th ed.). Philadelphia, PA: Wolters Kluwer.

The Snellen chart is composed of a series of progressively smaller rows of letters; the person is asked to read the lowest line possible. The fraction 20/20 is considered the standard of normal vision. Most people can see the letters on the line designated as 20/20. The patient should be encouraged to read every letter possible.

Visual acuity is then recorded. Common abbreviations related to vision and eye health are derived from Latin terms; these include OD (*oculus dexter*, right eye), OS (*oculus sinister*, left eye), and OU (*oculus uterque*, both eyes). The following would be an example of visual acuity documentation: A patient reads all five letters from the 20/20 line on the Snellen chart with the right eye (OD) and three of the five letters on the 20/30 line with the left eye (OS); the visual acuity is documented as OD: 20/20 and OS: 20/30 (Weber & Kelley, 2018).

If the patient cannot see the big “E” at the top of the Snellen chart, the examiner should determine next if the patient can count fingers (“CF” in documentation). Initially, the examiner stands 5 feet from the person, holds up a random number of fingers, and then asks the patient to count the number of fingers seen. If the patient is unable to count fingers at 5 feet, the examiner keeps moving a foot closer until either 1 foot away or the person can correctly count fingers. If the patient correctly counts the number of fingers at 3 feet, for example, the examiner would record vision as CF/3 feet.

Chart 58-2



GENETICS IN NURSING PRACTICE

Eye and Vision Disorders

Several eye and vision disorders are associated with genetic abnormalities. Some examples include:

Autosomal dominant:

- Aniridia
- Vitelliform macular dystrophy

Autosomal recessive:

- Achromatopsia
- Homocystinuria
- Leber congenital amaurosis

X linked:

- Choroideremia
- Color blindness

Mitochondrial inheritance:

- Leber hereditary optic neuropathy

Multiple inheritance patterns identified:

- Glaucoma
- Macular degeneration
- Retinitis pigmentosa

Other genetic illnesses that will impact vision:

- Albinism
- Isolated familial congenital cataracts
- Marfan syndrome
- Stickler syndrome
- Tay-Sachs disease
- Usher syndrome

Nursing Assessments

Refer to [Chapter 4, Chart 4-2: Genetics in Nursing Practice: Genetic Aspects of Health Assessment](#)

Family History Assessment Specific to Vision

- Assess history of family members in the past three generations with glaucoma, cataracts, night blindness (retinitis pigmentosa), color blindness, or other vision impairment.
- Inquire about the age of onset of symptoms (the onset of Leber congenital amaurosis is in childhood, whereas the onset of Leber

- hereditary optic neuropathy is in young adulthood).
- Inquire about family members with other disorders that may include visual impairment, such as cutaneous, metabolic, or connective tissue disorders and hearing loss.

Patient Assessment Specific to Vision

- Assess for other systemic and/or clinical features such as cutaneous or skeletal conditions, or hearing loss.
- Look for color and clarity of iris.
- Assess for presence of strabismus (cross-eyes), amblyopia (lazy eye), nystagmus, astigmatism, and near- or farsightedness.
- Assess for changes in visual acuity.
- Assess field of vision.
- Inquire about photophobia, night blindness, or double vision.

Genetics Resources

National Ophthalmic Disease Genotyping Network, eyegene.nih.gov/

See [Chapter 6, Chart 6-7](#) for additional components of genetic counseling.

If the patient cannot count fingers, the examiner raises one hand up and down or moves it side to side and asks in which direction the hand is moving. This level of vision is known as hand motion. A patient who can perceive only light is described as having light perception. The vision of a patient who cannot perceive light is described as no light perception.

External Eye Examination

The nurse uses a systematic approach to perform an external eye examination by first assessing for symmetry and placement of eyelids, pupils, and muscles. CNs III, IV, and VI control movement and pupil size. The eyelids should rest just above and below the corneal limbus without exposure of the sclera. The nurse observes for **ptosis** (drooping of the eyelid), **ectropion** (turning out of the lower eyelid), or **entropion** (turning in of the lower eyelid). Entropion may involve **trichiasis** (turning in of the eyelashes). Eyelids and lashes should be free of drainage or scaling.

The room should be darkened so that the pupils can be examined. The pupillary response is checked with a penlight to determine if the pupils are equally reactive and regular. A normal pupil is black. An irregular pupil may result from trauma, previous surgery, or a disease process.

The patient's eyes are observed in primary or direct gaze, and any head tilt is noted. A head tilt may indicate CN palsy. The patient is asked to stare at a target; each eye is covered and uncovered quickly while the examiner looks for any shift in gaze. The examiner observes for **nystagmus** (involuntary

oscillating movement of the eyeball). The extraocular movements of the eyes are tested by having the patient follow the examiner's finger, pencil, or a hand light through the six cardinal directions of gaze (i.e., up, down, right, left, and both diagonals).

Diagnostic Evaluation

A wide range of diagnostic studies may be performed in patients with eye disorders. The nurse should educate the patient about the purpose, what to expect, and any possible side effects related to these examinations prior to testing. The nurse should be aware of contraindications, potential complications, and trends in results. Trends provide information about disease progression as well as the patient's response to therapy.

Direct Ophthalmoscopy

A direct ophthalmoscope is a handheld instrument with various plus and minus lenses (Weber & Kelley, 2018). The lenses can be rotated into place, enabling the examiner to bring the cornea, lens, and retina into focus sequentially. The examiner holds the ophthalmoscope in the right hand and uses the right eye to examine the patient's right eye. The examiner switches to the left hand and left eye when examining the patient's left eye. During this examination, the room should be darkened, and the patient's eye should be on the same level as the examiner's eye. The patient and the examiner should be comfortable, and both should breathe normally. The patient is given a target to gaze at and is encouraged to keep both eyes open and steady.

When the fundus is examined, the vasculature comes into focus first. The veins are larger in diameter than the arteries. The examiner focuses on a large vessel and then follows it toward the midline of the body, which leads to the optic nerve. The central depression in the disc is known as the cup. The normal cup is about one third the size of the diameter of the disc. The size of the physiologic optic cup should be estimated, and the disc margins described as sharp or blurred. A silvery or coppery appearance, which indicates arteriolosclerosis, should be noted. The periphery of the retina is examined by having the patient shift their gaze. The last area of the fundus to be examined is the macula, because this area is the most sensitive to light. The retina of a young person often has a glistening effect, sometimes referred to as a cellophane reflex.

The healthy fundus should be free of any lesions. The examiner looks for intraretinal hemorrhages, which may appear as red smudges, and, if the patient has hypertension, they may be somewhat flame shaped. Lipid with a yellowish appearance may be present in the retina of patients with hypercholesterolemia

or diabetes. Soft exudates that have a fuzzy, white appearance (cotton-wool spots) should be noted. The examiner looks for microaneurysms, which look like little red dots, and nevi. Drusen (small, hyaline, globular deposits), commonly found in macular degeneration, appear as yellowish areas with indistinct edges. Small drusen have a more distinct edge. The examiner should sketch the fundus and document any abnormalities.

Indirect Ophthalmoscopy

The indirect ophthalmoscope is an instrument commonly used by the ophthalmologist to see larger areas of the retina, although in an unmagnified state. It produces a bright and intense light. The light source is affixed with a pair of binocular lenses mounted on the examiner's head. The ophthalmoscope is used with a handheld, 20-diopter lens.

Slit-Lamp Examination

The slit lamp is a binocular microscope mounted on a table. This instrument enables the user to examine the eye with magnification of 10 to 40 times the real image. The illumination can be varied from a broad to a narrow beam of light for different parts of the eye. For example, by varying the width and intensity of the light, the anterior chamber can be examined for signs of inflammation. Cataracts may be evaluated by changing the angle of the light. When a handheld contact lens, such as a three-mirror lens, is used with the slit lamp, the angle of the anterior chamber may be examined, as may the ocular fundus.

Tonometry

Tonometry is a common procedure to measure IOP. The device used for measuring IOP is an accurately calibrated applanation tonometer, which measures the pressure needed to flatten the cornea. This procedure is most commonly used to screen for and monitor IOP in glaucoma.

Nursing Interventions

Providing patient education prior to tonometry helps avoid possible errors in IOP measurement. Patients are cautioned to avoid squeezing the eyelids, holding their breath, or performing a Valsalva maneuver, because these may result in abnormally increased IOP.

Color Vision Testing

The ability to differentiate colors has a dramatic effect on the activities of daily living (ADLs). For example, the inability to differentiate between red and green can compromise traffic safety. Some careers (e.g., commercial artist, [color] photographer, airline pilot, electrician) may be closed to people with significant color deficiencies. The photoreceptor cells responsible for color vision are the cones, and the greatest area of color sensitivity is in the macula—the area of densest cone concentration.

A screening test, such as the polychromatic plates discussed in the next paragraph, can be used to establish whether a person's color vision is within normal range. Color vision deficits can be inherited. For example, red-green color deficiencies are inherited in an X-linked manner, affecting approximately 8% of men and 0.5% of women (Colour Blind Awareness, 2019). Acquired color vision losses may be caused by medications (e.g., digitalis) or pathology (e.g., cataracts). A simple test, such as asking a patient if the red top on a bottle of eye drops appears redder to one eye than the other, can be an effective tool. A difference in the perception of the intensity of the color red between the two eyes can be a symptom of a neurologic problem and may provide information about the location of the lesion.

Because alteration in color vision sometimes indicates conditions of the optic nerve, color vision testing is often performed in a neuro-ophthalmologic workup. The most common color vision test is performed using Ishihara polychromatic plates. These plates are bound together in a booklet. On each plate of this booklet are dots of primary colors that are integrated into a background of secondary colors. The dots are arranged in simple patterns, such as numbers or geometric shapes. Patients with diminished color vision may be unable to identify the hidden shapes. Patients with central vision conditions (e.g., macular degeneration) have more difficulty identifying colors than those with peripheral vision conditions (e.g., glaucoma) because central vision identifies color.

Amsler Grid

The Amsler grid is a test often used for patients with macular problems, such as macular degeneration. It consists of a geometric grid of identical squares with a central fixation point. The grid should be viewed by the patient wearing normal reading glasses. Each eye is tested separately. The patient is instructed to stare at the central fixation spot on the grid and report any distortion in the squares of the grid itself. For patients with macular disorders, some of the squares may look faded, or the lines may be wavy. Patients with age-related macular degeneration (AMD) are commonly given these Amsler grids to take home. The patient is encouraged to check the grids frequently, as often as daily, to monitor macular function for early detection of changes requiring

immediate attention (Gerstenblith & Rabinowitz, 2017; Weber & Kelley, 2018).

Ultrasonography

Lesions in the globe or the orbit may not be directly visible and are evaluated by ultrasonography. Ultrasonography is a valuable diagnostic technique, especially when the view of the retina is obscured by opaque media such as cataract or hemorrhage. An ultrasonography B-scan identifies pathology such as orbital tumors, retinal detachment, and vitreous hemorrhage. Ultrasonography A-scans are used to measure the axial length for implants prior to cataract surgery (Gerstenblith & Rabinowitz, 2017).

Optical Coherence Tomography

Optical coherence tomography is a technology that involves low-coherence interferometry (Gerstenblith & Rabinowitz, 2017). Light is used to evaluate retinal and macular diseases as well as anterior segment conditions. This method is noninvasive and involves no physical contact with the eye.

Fundus Photography

Fundus photography is used to detect and document retinal lesions. The patient's pupils are usually widely dilated before the procedure. The resulting fundus photographs can be viewed stereoscopically so that elevations such as macular edema can be identified.

Laser Scanning

Various scanning techniques use laser light in the diagnostic evaluation of eye disorders. Confocal laser scanning ophthalmoscopy provides a three-dimensional image of the optic nerve topography and is used alone or in conjunction with fundus photography to provide comparative data for suspected optic nerve disease such as glaucoma and **papilledema** (swelling of the optic disc due to increased intracranial pressure) (Gerstenblith & Rabinowitz, 2017). Laser scanning polarimetry is used to measure nerve fiber layer thickness and is an important indicator of glaucoma progression.

Angiography

Angiography is done using fluorescein or indocyanine green as contrast agents. Fluorescein angiography is used to evaluate clinically significant macular edema, document macular capillary nonperfusion, and identify retinal

and choroidal **neovascularization** (growth of abnormal new blood vessels) in AMD. It is an invasive procedure in which fluorescein dye is injected, usually into an antecubital vein. Within 10 to 15 seconds, this dye can be seen coursing through the retinal vessels. Over a 10-minute period, serial black-and-white photographs are taken of the retinal vasculature (Fischbach & Fischbach, 2018).

Indocyanine green angiography is used to evaluate abnormalities in the choroidal vasculature, which often are seen in macular degeneration. Indocyanine green dye is injected intravenously (IV), and multiple images are captured using digital video angiography. The dye is quickly removed from circulation by the liver, mostly clearing within 10 to 20 minutes (Norat, Soldozy, Elsarrag, et al., 2019).

Nursing Interventions

Prior to the angiography, the patient's blood urea nitrogen and creatinine should be checked to ensure that the kidneys will excrete the contrast agent (Fischbach & Fischbach, 2018). The patient should be well hydrated, and clear liquids are usually permitted up to the time of the test. The patient is instructed to remain immobile during the angiogram process and is told to expect a brief feeling of warmth in the face, behind the eyes, or in the jaw, teeth, tongue, and lips, and a metallic taste when the contrast agent is injected.

Nursing care after angiography includes observation of the injection site (usually the antecubital vein) for bleeding or hematoma formation (a localized collection of blood). Fluorescein may impart a gold tone to the skin in some patients, and urine may turn deep yellow or orange. This discoloration usually disappears in 24 hours. Indocyanine green dye is generally well tolerated, but some patients experience nausea and vomiting. Allergic reactions are rare; however, indocyanine green angiography is contraindicated in patients with a history of iodide reactions. Fluids are encouraged following the procedure to facilitate excretion of the contrast agent (Fischbach & Fischbach, 2018).

Perimetry Testing

Perimetry testing evaluates the field of vision. Visual field testing (i.e., perimetry) helps identify which parts of the patient's central and peripheral visual fields have useful vision. It is most helpful in detecting central **scotomas** (blind or partially blind areas in the visual field) in macular degeneration and the peripheral field defects in glaucoma and retinitis pigmentosa. Visual field evaluation and optic nerve assessment are major components of monitoring and detecting glaucoma progression.

IMPAIRED VISION

Refractive Errors

In refractive errors, vision is impaired because a shortened or elongated eyeball prevents light rays from focusing sharply on the retina. Blurred vision from refractive error can be corrected with eyeglasses or contact lenses. Ophthalmic **refraction** is the determination of the refractive errors of the eye for the purpose of vision correction and consists of placing various types of lenses in front of the patient's eyes to determine which lens best improves the patient's vision.

The depth of the eyeball is important in determining refractive error (see Fig. 58-5). Patients for whom the visual image focuses precisely on the macula and who do not need eyeglasses or contact lenses are said to have **emmetropia**, a normal refractive condition resulting in clear focus on retina with no optical defects (normal vision). Some people have deeper eyeballs, in which case the distant visual image focuses in front of, or short of, the retina; those with **myopia** are said to be nearsighted and have blurred distance vision. Other people have shallower eyeballs, in which case the visual image focuses beyond the retina; those with **hyperopia** are said to be farsighted and have excellent distance vision but blurry near vision (Weber & Kelley, 2018).

Wavefront technology to measure unique refractive imperfections of the cornea or higher aberrations (i.e., myopia, hyperopia, astigmatism) can be used to customize laser-assisted *in situ* keratomileusis (LASIK) procedures. These procedures are described later in this chapter.

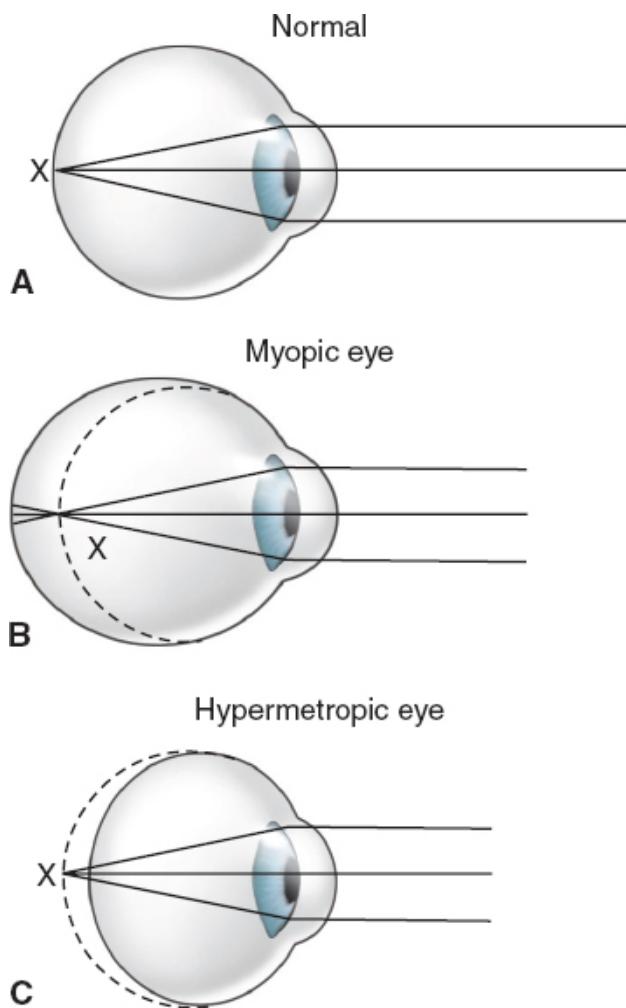


Figure 58-5 • Eyeball shape determines visual acuity in refractive errors. **A.** Normal eye. **B.** Myopic eye. **C.** Hypermetropic eye.

Vision Impairment and Blindness

Vision impairment is defined as having central visual acuity of 20/40 or worse in the better eye with the best possible correction. *Low vision* describes visual impairment that requires the use of devices and strategies to perform visual tasks.

Blindness is having best possible corrected central visual acuity that can range from 20/400 to no light perception. The clinical definition of absolute blindness is the absence of light perception. Legal blindness is a condition of impaired vision and is defined as having central visual acuity of 20/200 or worse in the better eye with the best possible correction or whose widest visual field diameter is 20 degrees or less (Bright Focus Foundation, 2020b). This definition neither equates with functional ability nor classifies the degrees of visual impairment. Legal blindness ranges from an inability to perceive light to

having some vision remaining. A person who meets the criteria for legal blindness may be eligible for government financial assistance through disability.

Assessment and Diagnostic Testing

Assessment of vision impairment includes a thorough history and examination of distance and near visual acuity, visual field, contrast sensitivity, glare, color perception, and refraction. Specially designed, low-vision visual acuity charts are used to evaluate patients.

Patient Interview

During history taking, the potential cause and duration of the patient's visual impairment are identified. Patients with retinitis pigmentosa, for example, have a genetic abnormality. Patients with diabetic macular edema typically have fluctuating visual acuity. Patients with macular degeneration have central acuity problems that cause difficulty in performing activities that require finer vision, such as reading. People with peripheral field defects have more difficulties with mobility. The patient's customary ADLs, medication regimen, habits (e.g., smoking), acceptance of the physical limitations brought about by the visual impairment, and realistic expectations of low-vision aids are identified and included in the plan of care, as well as provision of guidelines for safety and referrals to social services.

Contrast-Sensitivity Testing and Glare Testing

Contrast-sensitivity testing measures visual acuity in different degrees of light and dark contrast to determine visual function. Glare testing is also used to determine visual function. Glare can reduce a person's ability to see, especially in patients with cataracts. Those affected by loss of contrast sensitivity and glare have difficulty functioning in low light, or driving at night or in foggy conditions. People with a loss of contrast sensitivity may benefit from better illumination.



Gerontologic Considerations

Approximately half of all individuals who are identified as legally blind each year are 65 years of age or older (Eliopoulos, 2018). With aging, structural and functional changes occur in the eye (see [Chart 58-3](#)). **Presbyopia**, the loss of accommodative power in the lens, interferes with the ability to adequately focus and is the factor responsible for most older adults requiring some form of corrective lenses (Eliopoulos, 2018). Age-related changes in the eye are summarized in [Table 58-1](#).

Impaired vision is often accompanied by difficulty in performing functional activities. People with visual acuity of 20/80 to 20/100 with a visual field restriction of 60 degrees to greater than 20 degrees can read at a nearly normal level with optical aids. Their visual orientation is near normal but requires increased scanning of the environment (i.e., systematic use of head and eye movements). In a visual acuity range of 20/200 to 20/400 with a 20-degree to greater than 10-degree visual field restriction, the person can read slowly with optical aids.

The most common causes of blindness and visual impairment among adults 40 years and older are diabetic retinopathy, macular degeneration, glaucoma, and cataracts (Centers for Disease Control and Prevention [CDC], 2019). Macular degeneration is more prevalent among Caucasians, whereas glaucoma is more prevalent among African Americans (Eliopoulos, 2018).

Medical Management

Managing vision impairment involves magnification and image enhancement through the use of low-vision aids and strategies, as well as referrals to social services and community agencies. The goals are to optimize the patient's remaining vision and assist the patient to perform customary activities. [Table 58-2](#) presents low-vision aids. Medications are prescribed for glaucoma. Ongoing research suggests that gene therapy may replace or be an adjunct to pharmacologic or surgical treatment for ocular disorders in the near future (Jolly, Bridge, & MacLaren, 2019).

Chart 58-3



ETHICAL DILEMMA

Should Preservation of Patient Autonomy Threaten the Welfare of Others?

Case Scenario

You work as a staff nurse on a subacute care unit. D.P. is an 85-year-old man who has made daily visits to see his wife who has been a patient on the unit for the past week for daily physical and occupational therapy after having hip surgery. During your morning rounds today, D.P.'s wife tells you that her husband did not visit her yesterday and she is worried about him. After lunch, D.P. arrives on the subacute care unit with a sheepish grin on his face and a bandage on his forehead. When D.P.'s wife tells him that she was worried about him, he replies "Do not worry about me! I got into a fender bender yesterday but I am okay." D.P.'s wife shakes her head and says "Why are you so stubborn? You cannot see well at all anymore and should not be driving!" With tears in his eyes, D.P. says to his wife "But how else would I get here to see you, my sweetheart? Nothing could ever keep me away from you!" Based on previous conversations you have had with D.P. and his wife, you know that they live alone together in a two-bedroom ranch home that they have shared for the past 60 years. They have two adult sons who both live several hundred miles away.

Discussion

In American society, being able to drive is seen as consistent with being able to maintain an independent lifestyle. It is not uncommon for older adults to feel threatened by a possible loss of driving rights. D.P.'s apparent devotion to his wife and inability to identify an acceptable alternative means to visit her seems to be compounding his feelings of vulnerability.

There are some states that require health care providers to report drivers whom they think are impaired and unsafe. As a witness to this verbal exchange between D.P. and his wife, you may be obligated to investigate this situation further, or be liable for civil or criminal penalties.

Analysis

- Describe the ethical principles that are in conflict in this case (see [Chapter 1, Chart 1-7](#)). Is it possible to preserve D.P.'s autonomy while ensuring that neither D.P. nor others are harmed should he continue to drive?
- Regardless of the legal requirements in your state, what are your ethical and moral obligations to D.P., his wife, and to others who may be impacted by D.P.'s driving?
- What resources might you mobilize to be of assistance to you, to D.P., and to D.P.'s wife? It is not uncommon for spouses to neglect their own health needs when their partner becomes ill. You may wish to explore whether D.P. has had a recent focused visual examination, to find whether he has visual deficits amenable to correction. In addition, although D.P.'s children do not live close, D.P. and his wife may have

other social support networks that they could tap into to assist them so that D.P. can continue his daily visits with his wife. Local Area Agencies on Aging can also provide a wealth of contacts and support services for older adults that could be explored.

References

Morgan, E. (2018). Driving dilemmas: A guide to driving assessment in primary care. *Clinics in Geriatric Medicine*, 34(1), 107–115.

Resources

See [Chapter 1, Chart 1-10](#) for Steps of an Ethical Analysis and Ethics Resources.

TABLE 58-1

Age-Related Changes in the Eye

External Eye	Structural Change	Functional Change	History and Physical Findings
Eyelids and lacrimal structures	Loss of skin elasticity and orbital fat, decreased muscle tone; wrinkles develop	Lid margins turn in, causing entropion; or lid margins turn out, resulting in ectropion.	Reports of burning, foreign-body sensation, epiphora; injection, inflammation, and ulceration may occur.
Refractive changes; presbyopia	Loss of accommodative power in the lens with age	Reading materials must be held at increasing distance in order to focus.	Patient reports, "Arms are too short!"; need for increased light; reading glasses or bifocals needed.
Cataract	Opacities in the normally crystalline lens	Interference with the focus of a sharp image on the retina.	Patients report increased glare, decreased vision, changes in color values (blue and yellow especially affected).
Posterior vitreous detachment	Liquefaction and shrinkage of vitreous body	May lead to retinal tears and detachment.	Reports light flashes, cobwebs, floaters.
Age-related macular degeneration (AMD)	Drusen (yellowish aging spots in the retina) appear and coalesce in the macula. Abnormal choroidal blood vessels may lead to formation of fibrotic disciform scars in the macula	Central vision is affected; onset is more gradual in dry AMD, more rapid in wet AMD; distortion and loss of central vision may occur.	Reading vision is affected; words may be missing letters, faded areas appear on the page, straight lines may appear wavy; drusen, pigmentary changes in retina; abnormal submacular choroidal vessels.

Adapted from Eliopoulos, C. (2018). *Gerontological nursing* (9th ed.). Philadelphia, PA: Wolters Kluwer.

TABLE 58-2 Activities Affected by Visual Impairment and Suggestions for Low-Vision Aids

Activity	Optical Aids	Nonoptical Aids
Shopping	Hand magnifier	Lighting, color cues
Fixing a snack	Bifocals	Color cues; consistent food storage plan
Eating out	Hand magnifier	Flashlight, portable lamp
Identifying money	Bifocals, hand magnifier	Arrange paper money in wallet compartments
Reading print	High-power spectacle, bifocals, hand magnifier, stand magnifier, closed-circuit television	Lighting, high-contrast print, large print, reading slit
Writing	Hand magnifier	Lighting, bold-tip pen, black ink
Using a telephone	Hand magnifier	Large-print buttons, hand-printed directory Braille phones, picture/photo phones, and talking phones Accessibility settings on smartphones
Crossing streets	Lightweight handheld monoscopes/telescopes	Cane; ask directions
Finding taxis and bus signs	Lightweight handheld monoscopes/telescopes	Ask for assistance
Using ride-sharing services	Lightweight handheld monoscopes/telescopes	Ask for assistance
Reading medication labels	Hand magnifier	Color codes, large print
Reading stove dials	Hand magnifier	Color codes, raised dots
Adjusting the thermostat	Hand magnifier	Enlarged-print model, digital thermostats that can be controlled via voice or apps
Using a computer or electronic tablet	Spectacles	High-contrast color, large-print program. Screen-reader programs convert text on the computer screen to synthesized speech
Reading signs	Spectacles	Move closer
Watching sporting event	Lightweight handheld monoscopes/telescopes	Sit in front rows

Adapted from Pagliuca, L. M., Macêdo-Costa, K. N., Rebouças, C. B., et al. (2014). Validation of the general guidelines of communication between the nurse and the blind. *Revista Brasileira de Enfermagem*, 67(5), 715–721.

Referrals to community agencies may be necessary for patients with low vision who live alone and cannot self-administer their medications. Community agencies, such as the Lighthouse Guild, offer a wide variety of vision and health care services to patients with low vision and blindness.

Nursing Management

Nurses need to be sensitive to the challenges faced by patients with visual impairments. Coping with blindness involves emotional, physical, and social adaptation. The emotional adjustment to blindness or severe visual impairment determines the success of the physical and social adjustments of the patient. Successful emotional adjustment means acceptance of blindness or severe visual impairment.

Promoting Coping

Effective coping may not occur until the patient recognizes the permanence of the low vision or blindness. A patient who is newly visually impaired and their family members undergo the various steps of grieving: denial and shock, anger and protest, restitution, loss resolution, and acceptance. The ability to accept the changes that must come with visual loss and willingness to adapt to those changes influence the successful rehabilitation of the patient with vision loss. Additional aspects to consider are value changes, independence-dependence conflicts, coping with stigma, and learning to function in social settings without visual cues and landmarks.

Promoting Spatial Orientation and Mobility

A person who is blind or severely visually impaired requires strategies for adapting to the environment. ADLs, such as walking to a chair from a bed, require spatial concepts. The person needs to know where they are in relation to the rest of the room, to understand the changes that may occur, and to know how to approach the desired location safely. This requires a collaborative effort between the patient and the responsible adult who serves as the sighted guide. The nurse must assess the degree of physical assistance the person with vision loss requires and communicate this to other health care personnel.

The nurse should be aware of the importance of techniques in providing physical assistance, encouraging independence, and ensuring safety. Strategies for interacting with the patient with vision loss are presented in [Chart 58-4](#). Research supports the use of a validated protocol to educate staff to perform therapeutic communication, deliver services, and minimize communication

barriers with patients who are blind (Pagliuca, Macêdo-Costa, Rebouças, et al., 2014). The readiness of the patient and family to learn must be assessed before initiating orientation and mobility training.

Promoting Home, Community-Based, and Transitional Care

The nurse, social worker, family, and others collaborate to assess the patient's home condition and support system. A low vision specialist or occupational therapist should be consulted, particularly for patients for whom identifying and administering medications pose challenges. Referral for vision rehabilitation should be provided for appropriate patients (Shah, Schwartz, Gartner, et al., 2018).

Chart 58-4

Strategies for Interacting with People Who Are Blind or Have Low Vision

- Remember that the only difference between you and people who are blind or have low vision is that they are not able to see through their eyes what you are able to see through yours.
- Do not be uncomfortable when in the company of a person who is blind or has low vision. Talk with the person as you would talk with any other person, honestly and with respect, courtesy, and empathy; do not be concerned about using words like “see” and “look.” There is no need to raise your voice unless the person asks you to do so.
- Identify yourself as you approach the person and before you make physical contact. Tell the person your name and your role. If another person approaches, introduce them. When you leave the room, be sure to tell the person that you are leaving and if anyone else remains in the room.
- Keep in mind that it is often appropriate to touch the person’s hand or arm lightly to indicate that you are about to speak.
- When talking, face the person and speak directly to them using a normal tone of voice.
- Be specific when communicating direction. Mention a specific distance or use clock cues when possible (e.g., walk left about 2 yards; walk about 20 feet to the right; the telephone is at 2 o’clock). Avoid using phrases such as “over there.”
- When you offer to assist someone, allow the person to hold on to your arm just above the elbow and to walk a half step behind you.
- When offering the person a seat, place the person’s hand on the back or the arm of the seat.
- When you are about to go up or down a flight of stairs, tell the person and place their hand on the banister.
- Make sure that the environment is free of obstacles; close doors and cabinets so that they are not in the path.
- Offer to read written information, such as a menu.
- If you serve food to the person, use clock cues to specify where everything is on the plate.
- When the person who is blind or has low vision is a patient in a health care facility:
 - Make sure all objects the person will need are close at hand.
 - Identify the location of objects that the person may need (e.g., “The call light is near your right hand”; “The telephone is on the table on the left side of your bed.”)
 - Remove obstacles that may be in the person’s pathway and could cause a fall.
 - Place all assistive devices that the person uses close at hand; let the person feel the devices so that they know their location.

- Do not distract a service animal unless the owner has given permission.
- Ask the person, “How can I help you?” At some times, the person needs help; at other times, help may not be needed.

Adapted from Pagliuca, L. M., Macêdo-Costa, K. N., Rebouças, C. B., et al. (2014). Validation of the general guidelines of communication between the nurse and the blind. *Revista Brasileira de Enfermagem*, 67(5), 715–721.

Other interventions that are appropriate for some people with visual impairment or blindness include Braille and service animals. There has been an ever-increasing reliance on print magnification technology as well as technology-assisted speech output. However, although the use of Braille may be less important for adults who have already learned language and grammar skills, educators and low vision specialists have continued to advocate that children who are legally blind be given the opportunity to learn Braille.

Guide dogs, also known as service dogs, are dogs that are specially bred, raised, and rigorously trained to assist people who are blind. The guide dog is a constant companion to the person who is blind (also referred to as the animal's handler) and is allowed on airplanes and in restaurants, stores, hotels, and other public places. With the assistance of the guide dog, the person who is blind can be extremely mobile and accomplish normal activities both within and outside of the home and workplace. A dog in harness is a working dog, not a pet. The dog should not be distracted from his job by well-intentioned strangers who want to pet, feed, or play with the animal. The dog's handler should always be consulted before approaching the working guide dog. Most health care facilities have a service animal policy that outlines the responsibilities of the handler with regard to the care of the animal.

Unfolding Patient Stories: Vernon Watkins • Part 2



Recall from [Chapter 14](#) Vernon Watkins, who came to the emergency department with severe abdominal pain and underwent a hemicolectomy for a bowel perforation. During postoperative care the nurse determines that he has poor vision. What measures can the nurse take to maintain a safe environment for a patient with visual impairment? How will the discovery of impaired vision impact the nursing plan of care, discharge planning, and delivery of patient education?

Care for Vernon and other patients in a realistic virtual environment: [vSim\(\[thePoint.lww.com/vSimMedicalSurgical\]\(http://thePoint.lww.com/vSimMedicalSurgical\)\)](#). Practice documenting these patients' care in DocuCare (thePoint.lww.com/DocuCareEHR).

OCULAR MEDICATION ADMINISTRATION

Because medications are often prescribed to treat ocular disorders, nurses must understand the actions of the commonly used medications and effective administration. The main objective of ocular medication delivery is to maximize the amount of medication that reaches the ocular site of action in sufficient concentration to produce a beneficial therapeutic effect. This is determined by the dynamics of ocular pharmacokinetics: absorption, distribution, metabolism, and excretion.

Ocular absorption involves the entry of a medication into the aqueous humor through the different routes of ocular medication administration. The rate and extent of aqueous humor absorption are determined by the characteristics of the medication and the anatomy and physiology of the eye. Natural barriers of absorption that diminish the efficacy of ocular medications include the following:

- *Limited size of the conjunctival sac.* The conjunctival sac can hold only 50 mcL, and any excess is wasted. The volume of one eye drop from commercial topical ocular solutions typically ranges from 20 to 35 mcL.
- *Corneal membrane barriers.* The epithelial, stromal, and endothelial layers are barriers to absorption.
- *Blood–ocular barriers.* Blood–ocular barriers prevent high ocular tissue concentration of most ophthalmic medications because they separate the

bloodstream from the ocular tissues and keep foreign substances from entering the eye, thereby limiting a medication's efficacy.

- *Tearing, blinking, and drainage.* Increased tear production and drainage due to ocular irritation or an ocular condition may dilute or wash out an instilled eye drop; blinking expels an instilled eye drop from the conjunctival sac.

Distribution of an ocular medication into the ocular tissues varies by tissue type—the conjunctiva, cornea, lens, iris, ciliary body, and choroids absorb medications to varying degrees. Medications penetrate the corneal epithelium either by intracellular diffusion (passing through the cells) or by intercellular diffusion (passing between the cells). Hydrophilic (water soluble) medications diffuse through the intracellular route, and lipophilic (fat soluble) medications diffuse through the intercellular route. Topical administration usually does not reach the retina in significant concentrations. Because the space between the ciliary process and the lens is small, medication diffusion in the vitreous is slow. When high concentrations of medication in the vitreous are required, intraocular injection is often chosen to bypass the natural ocular anatomic and physiologic barriers (American Society of Ophthalmic Registered Nurses [ASORN], 2013).

Aqueous solutions are most commonly used for the eye. They are the least expensive medications and interfere the least with vision. However, corneal contact time is brief because tears dilute the medication. Ophthalmic ointments have extended retention time in the conjunctival sac and provide a higher concentration than eye drops. The major disadvantage of ointments is the blurred vision that results after application. In general, eyelids and eyelid margins are best treated with ointments. The conjunctiva, limbus, cornea, and anterior chamber are treated most effectively with instilled solutions or suspensions. Subconjunctival injection may be necessary for better absorption in the anterior chamber. If high medication concentrations are required in the posterior chamber, intravitreal injections or systemically absorbed medications are considered. Contact lenses and collagen shields soaked in antibiotic agents are alternative delivery methods for treating corneal infections.

Of all these delivery methods, the topical route of administration—instilled eye drops and applied ointments—remains the most common and widely recommended (ASORN, 2013). Topical instillation, which is the least invasive method, permits self-administration of medication and produces fewer side effects.

Preservatives are commonly used in ocular medications. Benzalkonium chloride, for example, prevents the growth of organisms and enhances the corneal permeability of most medications; however, some patients are allergic to this preservative. This may be suspected even if the patient had never before experienced an allergic reaction to systemic use of the medication in question. Eye drops without preservatives can be prepared by pharmacists.

Common Ocular Medications

Common ocular medications include topical anesthetic, mydriatic, and cycloplegic agents that reduce IOP; anti-infective medications; corticosteroids; nonsteroidal anti-inflammatory drugs (NSAIDs); antiallergy medications; eye irrigants; and lubricants.

Topical Anesthetic Agents

One or two drops of proparacaine hydrochloride and tetracaine hydrochloride are instilled before diagnostic procedures such as tonometry and minor ocular procedures such as removal of sutures or conjunctival or corneal scrapings. Topical anesthetic agents are also used for severe eye pain to allow the patient to open their eyes for examination or treatment (e.g., eye irrigation for chemical burns). Anesthesia occurs within 20 seconds to 1 minute and lasts 10 to 20 minutes.



Quality and Safety Nursing Alert

To prevent injury, the nurse educates the patient not to rub the eyes while anesthetized because this may result in damage to the cornea.

Mydriatic and Cycloplegic Agents

Mydriasis, or pupil dilation, is the main objective of the administration of mydriatics and cycloplegics (see [Table 58-3](#)). These two types of medications function differently and are used in combination to achieve the maximal dilation that is needed during surgery and fundus examinations to give the ophthalmologist a better view of the internal eye structures. Mydriatics potentiate alpha-adrenergic sympathetic effects that result in the relaxation of the ciliary muscle. This causes the pupil to dilate. However, this sympathetic action alone is not enough to sustain mydriasis because of its short duration of action. The strong light used during an eye examination also stimulates miosis (i.e., pupillary contraction). Cycloplegic medications are given to paralyze the iris sphincter.

The patient is educated about the temporary effects of mydriasis on vision, such as glare and the inability to focus properly. The patient may have difficulty reading. The effects of the various mydriatics and cycloplegics can last 3 hours to several days. The patient is advised to wear sunglasses (most eye clinics provide protective sunglasses). The ability to drive depends on the person's age, vision, and comfort level. Some patients can drive safely with the use of sunglasses, whereas others may need to be driven home.

Mydriatic and cycloplegic agents affect the central nervous system. Their effects are most prominent in younger and older adult patients; these patients must be assessed closely for symptoms, such as increased blood pressure, tachycardia, dizziness, ataxia, confusion, disorientation, incoherent speech, and hallucination. These medications are contraindicated in patients with narrow angles or shallow anterior chambers and in patients taking monoamine oxidase inhibitors or tricyclic antidepressants.

Medications Used to Treat Glaucoma

Therapeutic medications for glaucoma are used to lower IOP by decreasing aqueous production or increasing aqueous outflow. Because glaucoma calls for lifetime therapy, the patient must be educated with regard to both the ocular and systemic side effects of the medications. See section on glaucoma later in this chapter.

Anti-Infective Medications

Anti-infective medications include antibiotic, antifungal, and antiviral agents. Most are available as drops, ointments, or subconjunctival or intravitreal injections. Antibiotics include penicillin, cephalosporins, aminoglycosides, and fluoroquinolones. The main antifungal agent is amphotericin B. Side effects of amphotericin are serious and include severe pain, conjunctival necrosis, iritis, and retinal toxicity. Antiviral medications include acyclovir and ganciclovir. They are used to treat ocular infections associated with herpes virus and cytomegalovirus (CMV). Patients receiving ocular anti-infective agents are subject to the same side effects and adverse reactions as those receiving oral or parenteral medications.

TABLE 58-3



Mydriatics and Cycloplegics

Medication	Available Preparation/ Concentration	Indication/Dosage	Peak		Recovery Time	
			Mydriasis (Minutes)	Cycloplegia (Minutes)	Mydriasis	Cycloplegia
atropine	Ointment (0.5–2%) Solutions (0.5–3%)	In uveitis, or after surgery, 2–4 × daily	30–40	60–180	7–10 days	6–12 days
cyclopentolate hydrochloride	Solution (0.5–2%)	Given with mydriatics every 5–10 min × 3 or until the pupils are fully dilated for ophthalmoscopy and surgical procedures	30–60	25–75	1 day	6–24 h
phenylephrine hydrochloride	Solutions (2.5%, 10%)	Given with cycloplegics in pupillary dilation for ophthalmoscopy and surgical procedures every 5–10 min × 3	10–60	—	3–5 h	—
scopolamine hydrobromide	Solution (0.25%)	Same as atropine	20–30	30–60	3–7 days	3–7 days
homatropine	Solution (5–2.5%)	Same as atropine and scopolamine	40–60	30–60	1–3 days	1–3 days

Adapted from Comerford, K. C., & Durkin, M. T. (2020). *Nursing 2020 drug handbook*. Philadelphia, PA: Wolters Kluwer.

Corticosteroids and Nonsteroidal Anti-Inflammatory Drugs

Topical preparations of corticosteroids are commonly used in inflammatory conditions of the eyelids, conjunctiva, cornea, anterior chamber, lens, and uvea. In posterior segment diseases that involve the posterior sclera, retina, and optic nerve, topical agents are less effective and parenteral and oral routes are preferred. When a suspension is prescribed, the patient is instructed to shake the bottle several times to promote mixture of the medication and maximize its therapeutic effect. The most common ocular side effects of long-term topical corticosteroid administration are glaucoma, cataracts, susceptibility to infection, impaired wound healing, mydriasis, and ptosis. High IOP may develop, which is reversible after corticosteroid use is discontinued. To avoid the side effects of corticosteroids, NSAIDs are used as an alternative in controlling inflammatory eye conditions and postoperatively to reduce inflammation.

Antiallergy Medications

Ocular hypersensitivity reactions, such as allergic conjunctivitis, are extremely common. These conditions result primarily from responses to environmental allergens. Most allergens are airborne or carried to the eye by the hand or by other means, although allergic reactions may also be drug induced. Corticosteroids are commonly used as anti-inflammatory and immunosuppressive agents to control ocular hypersensitivity reactions.

Ocular Irrigants and Lubricants

Most irrigating solutions are used to cleanse the external lids to maintain lid hygiene, to irrigate the external corneal surface to regain normal pH (e.g., in chemical burns), to irrigate the corneal surface to eliminate debris, or to inflate the globe intraoperatively. These solutions have various compositions that include sodium, potassium, magnesium, calcium, bicarbonate, glucose, and glutathione (i.e., substance found in the aqueous humor). Sterile irrigating solutions for lid hygiene are available. Irrigating solutions are safe to use with an intact corneal surface; however, the corneal surface should not be irrigated in cases of threatened corneal perforation. For patients with severe corneal ulcer, specific instructions from the ophthalmologist must be obtained regarding whether it is safe to irrigate the corneal surface or just to cleanse the external lids. Although it is good practice to promote hygiene, prevention of complications must be the primary concern. Normal saline solutions are commonly used to irrigate the corneal surface when chemical burns occur.

Lubricants, such as artificial tears, help alleviate corneal irritation, such as dry eye syndrome. Artificial tears are topical preparations of carboxymethylcellulose or hydroxypropyl methylcellulose that are prepared as eye drop solutions, ointments, or ocular inserts (inserted at the lower conjunctival cul-de-sac once each day). The eye drops can be instilled as often as every hour, depending on the severity of symptoms.

Nursing Management

The objectives in administering ocular medications are to ensure proper administration to maximize the therapeutic effects and to ensure the safety of the patient by monitoring for systemic and local side effects (ASORN, 2013). Absorption of eye drops by the nasolacrimal duct is undesirable because of the potential systemic side effects of ocular medications. To diminish systemic absorption and minimize the side effects, it is important to occlude the puncta (see [Chart 58-5](#)). This is especially important for patients who are most vulnerable to medication overdose, including older adults; women who are pregnant or lactating; and patients with cardiac, pulmonary, hepatic, or kidney disease. A 5-minute interval between instillation of different types of ocular drops is recommended.

Before the administration of ocular medications, the nurse warns the patient that blurred vision, stinging, and a burning sensation are symptoms that ordinarily occur after instillation and are temporary. Risk for interactions of the ocular medication with other ocular and systemic medications must be emphasized; therefore, a careful patient interview regarding the medications being taken must be obtained.



Quality and Safety Nursing Alert

To prevent infection, meticulous hand hygiene before and after medication instillation is crucial. In addition, the tip of the eye drop bottle or the ointment tube must never touch any part of the eye, and the medication must be recapped immediately after each use.

If a patient who instills their own medications cannot feel the eye drops when they are instilled, the eye medication may be refrigerated, because a cold drop is easier to detect. A 5-minute interval between successive administrations allows adequate drug retention and absorption. The patient or the caregiver at home should be asked to demonstrate actual eye drop or ointment instillation and punctal occlusion.

GLAUCOMA

The term **glaucoma** is used to refer to a group of ocular conditions characterized by elevated IOP (McMonnies, 2017). If left untreated, the increased IOP damages the optic nerve and nerve fiber layer, but the degree of harm is highly variable (Eliopoulos, 2018). The optic nerve damage is related to the IOP caused by congestion of aqueous humor in the eye. A range of IOPs are considered “normal,” but these may also be associated with vision loss in some patients.

Chart 58-5

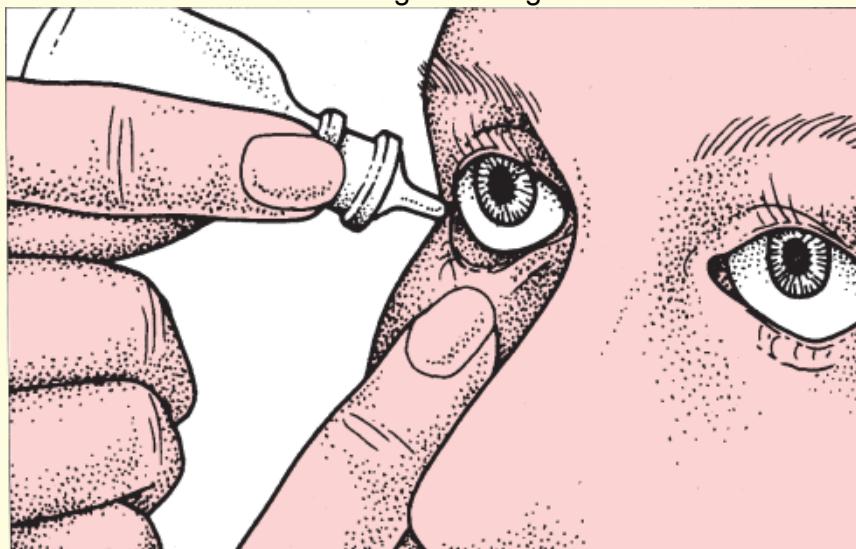


PATIENT EDUCATION

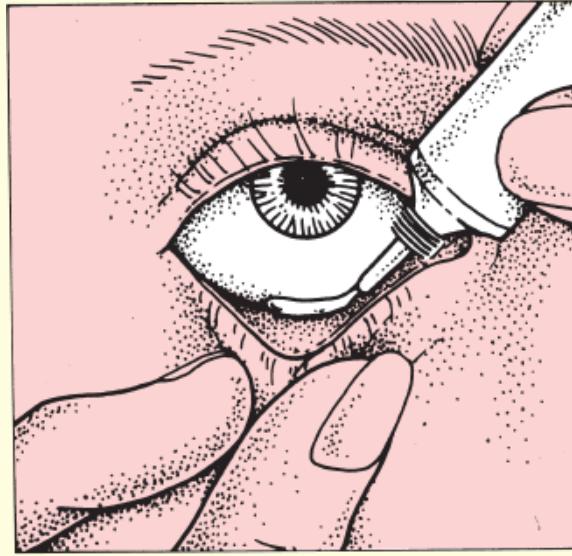
Instilling Eye Medications

The nurse instructs the patient to:

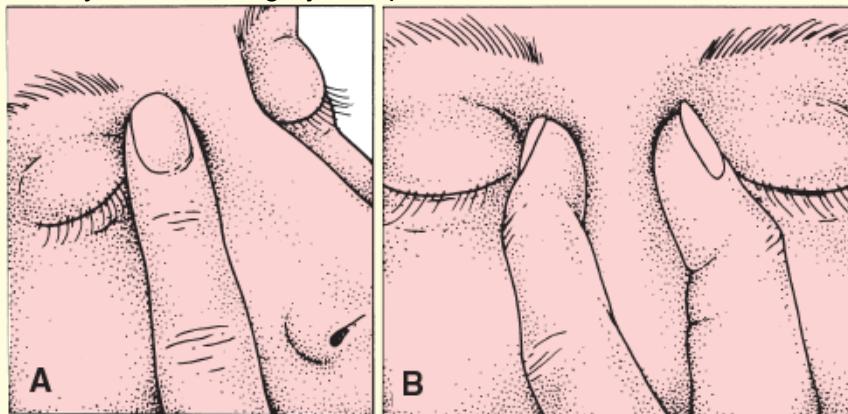
- Never use eye solutions that have changed colors.
- Perform hand hygiene before and after the procedure.
- Ensure adequate lighting.
- Read the label of the eye medication to verify that it is the correct medication.
- Remove contact lens as needed.
- Assume a comfortable position.
- Avoid touching the tip of the medication container to any part of the eye or face.
- Hold the lower lid down; do not press on the eyeball. Apply gentle pressure to the cheekbone to anchor the finger holding the lid.



- Instill eye drops before applying ointments.
- Apply a 0.25- to 0.5-inch ribbon of ointment to the lower conjunctival sac.



- Keep the eyelids closed, and apply gentle pressure on the inner canthus (punctal occlusion) near the bridge of the nose for 1 or 2 minutes immediately after instilling eye drops.



- Use a clean tissue to gently pat skin to absorb excess eye drops that run onto the cheeks.
- Wait 5 minutes before instilling another eye drop and 10 minutes before instilling another ointment.
- Reinsert contact lens if applicable.

Adapted from American Society of Ophthalmic Registered Nurses (ASORN). (2013). *ASORN recommended practice: Use of multi-dose medications*. San Francisco, CA: Author; Glaucoma Research Foundation. (2020). Eye drop tips. Retrieved on 2/11/2020 at: www.glaucoma.org/treatment/eyedrop-tips.php

Glaucoma is estimated to affect three million Americans, approximately 50% of whom are undiagnosed (Moore et al., 2018). Glaucoma is more prevalent in people older than 40 years, and it is the third most common age-related eye disease in the United States. [Chart 58-6](#) presents the risk factors for

glaucoma. There is no cure for glaucoma, but the disease can be controlled (Glaucoma Research Foundation, 2019).

Physiology

Aqueous humor flows between the iris and the lens, nourishing the cornea and lens. Most (90%) of the fluid then flows out of the anterior chamber, draining through the spongy trabecular meshwork into the canal of Schlemm and the episcleral veins (see Fig. 58-6). About 10% of the aqueous fluid exits through the ciliary body into the suprachoroidal space and then drains into the venous circulation of the ciliary body, choroid, and sclera (Norris, 2019). Unimpeded outflow of aqueous fluid depends on an intact drainage system and an open angle (about 45 degrees) between the iris and the cornea. A narrower angle places the iris closer to the trabecular meshwork, diminishing the angle. The amount of aqueous humor produced tends to decrease with age, in systemic diseases such as diabetes, and in ocular inflammatory conditions.

Chart 58-6 RISK FACTORS

Glaucoma

- Black or Asian race
- Cardiovascular disease
- Diabetes
- Family history of glaucoma
- Migraine syndromes
- Myopia (nearsightedness)
- Obstructive sleep apnea
- Older age
- Previous eye trauma
- Prolonged use of topical or systemic corticosteroids
- Thin cornea

Adapted from McMonnies, C. W. (2017). Glaucoma history and risk factors. *Journal of Optometry*, 10(2), 71–78; Norris, T. (2019). *Porth's pathophysiology: Concepts of altered health status* (10th ed.). Philadelphia, PA: Wolters Kluwer.

IOP is determined by the rate of aqueous production, the resistance encountered by the aqueous humor as it flows out of the passages, and the venous pressure of the episcleral veins that drain into the anterior ciliary vein.

When aqueous fluid production and drainage are in balance, the IOP is between 10 and 21 mm Hg. When aqueous fluid is inhibited from flowing out, pressure builds up within the eye. Fluctuations in IOP occur with time of day, exertion, diet, and medications. IOP tends to increase with blinking, tight lid squeezing, and upward gazing. Systemic conditions such as diabetes and intraocular conditions such as uveitis and retinal detachment have been associated with elevated IOP. Glaucoma may not be recognized in people with thin corneas because measurement of the IOP may be falsely low as a result of this thinness.

Pathophysiology

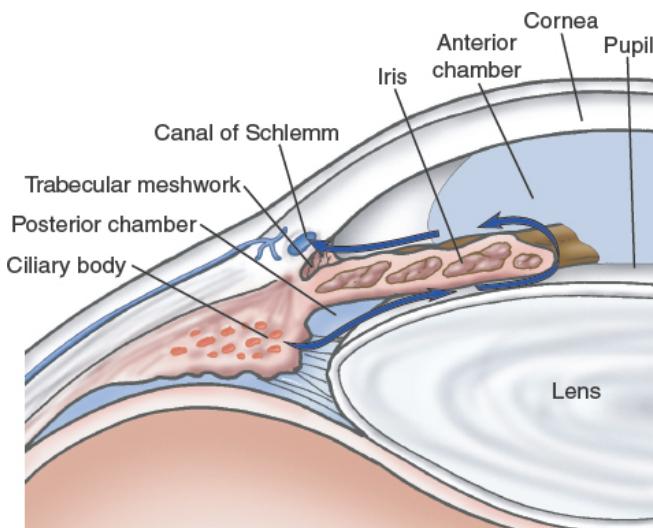
There are two theories regarding how increased IOP damages the optic nerve in glaucoma. The direct mechanical theory suggests that high IOP damages the retinal layer as it passes through the optic nerve head. The indirect ischemic theory suggests that high IOP compresses the microcirculation in the optic nerve head, resulting in cell injury and death. Some glaucomas appear as exclusively mechanical, and some are exclusively ischemic types. Typically, most cases are a combination of both.

Classification of Glaucoma

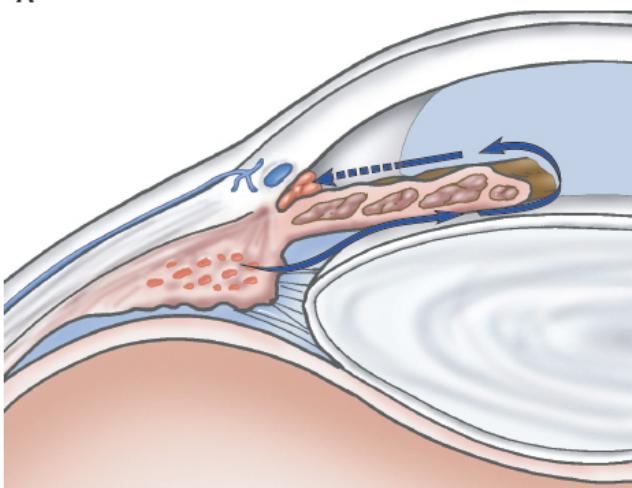
There are several types of glaucoma. Forms of glaucoma are identified as wide-angle glaucoma; narrow-angle glaucoma; congenital glaucoma; and glaucoma associated with other conditions, such as developmental anomalies or corticosteroid use. Glaucoma can be primary or secondary, depending on whether associated factors contribute to the rise in IOP. The two common clinical forms of glaucoma in adults are wide- and narrow-angle glaucoma, which are differentiated by the mechanisms that cause impaired aqueous outflow (Norris, 2019). [Table 58-4](#) summarizes the characteristics of the different types of adult glaucoma.

Clinical Manifestations

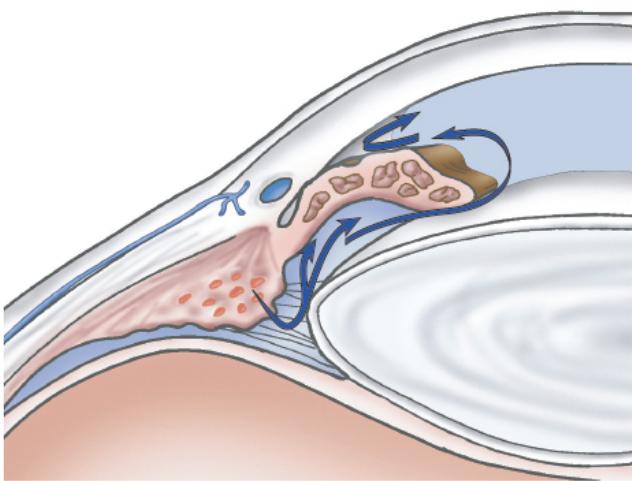
Glaucoma is often called the “silent thief of sight” because most patients are unaware that they have the disease until they have experienced visual changes and vision loss. The patient may not seek health care until they experience blurred vision or “halos” around lights, difficulty focusing, difficulty adjusting eyes in low lighting, loss of peripheral vision, aching or discomfort around the eyes, and headache.



A



B



C

Figure 58-6 • A. Normally, aqueous humor, which is secreted in the posterior chamber, gains access to the anterior chamber by

flowing through the pupil. In the angle of the anterior chamber, it passes through the canal of Schlemm into the venous system. **B.** In wide-angle glaucoma, the outflow of aqueous humor is obstructed at the trabecular meshwork. **C.** In narrow-angle glaucoma, the aqueous humor encounters resistance to flow through the pupil. Increased pressure in the posterior chamber produces a forward bowing of the peripheral iris so that the iris blocks the trabecular meshwork. Reprinted with permission from Norris, T. L. (2019). *Porth's pathophysiology: Concepts of altered health states* (10th ed., Fig. 19.11). Philadelphia, PA: Wolters Kluwer.

TABLE 58-4 Glaucoma Types, Clinical Manifestation, and Treatment

Types of Glaucoma	Clinical Manifestations	Treatment
Wide Angle		
	<i>Usually bilateral, but one eye may be more severely affected than the other. In wide-angle glaucoma, the anterior chamber angle is open and appears normal.</i>	
Normal-tension glaucoma	IOP \leq 21 mm Hg. Optic nerve damage, visual field defects.	If medical treatment is unsuccessful, LT can decrease IOP by 20%. Glaucoma filtering surgery if continued optic nerve damage despite medication therapy and LT.
Ocular hypertension	Elevated IOP. Possible ocular pain or headache.	The best management for normal-tension glaucoma management is yet to be established. Goal is to lower the IOP by at least 30%.
Narrow Angle		
	<i>Obstruction in aqueous humor outflow due to the complete or partial closure of the angle from the forward shift of the peripheral iris to the trabecula. The obstruction results in an increased IOP.</i>	
Acute angle-closure glaucoma	Rapidly progressive visual impairment, periocular pain, conjunctival hyperemia, and congestion. Pain may be associated with nausea, vomiting, bradycardia, and profuse sweating. Reduced central visual acuity, severely elevated IOP, corneal edema. Pupil is vertically oval, fixed in a semidilated position, and unreactive to light and accommodation.	Ocular emergency; administration of hyperosmotics, acetazolamide, and topical ocular hypotensive agents. Possible laser iridotomny (incision in the iris) to release blocked aqueous and reduce IOP. Other eye is also treated with pilocarpine eye drops and/or surgical management to avoid a similar spontaneous attack.
Subacute angle-closure glaucoma	Transient blurring of vision, halos around lights; temporal headaches and/or ocular pain; pupil may be semidilated.	Prophylactic peripheral laser iridotomny. Can lead to acute or chronic angle-closure glaucoma if untreated.
Chronic angle-closure glaucoma	Progression of glaucomatous cupping and significant visual field loss; IOP may be normal or elevated; ocular pain and headache.	Management includes laser iridotomny and medications.

IOP, intraocular pressure; LT, laser trabeculoplasty.

Adapted from McMonnies, C. W. (2017). Glaucoma history and risk factors. *Journal of Optometry*, 10(2), 71–78; Norris, T. (2019). *Porth's pathophysiology: Concepts of altered health status* (10th ed.). Philadelphia, PA: Wolters Kluwer.

Assessment and Diagnostic Findings

The purpose of a glaucoma workup is to establish the diagnostic category, assess the optic nerve damage, and formulate a treatment plan. The patient's ocular and medical history must be detailed to investigate the history of predisposing factors. The types of examinations used in glaucoma include tonometry to measure the IOP, ophthalmoscopy to inspect the optic nerve, and central visual field testing (Glaucoma Research Foundation, 2019; Norris, 2019).

The changes in the optic nerve related to glaucoma are pallor and cupping of the optic nerve disc. The pallor of the optic nerve is caused by a lack of blood supply. Cupping is characterized by exaggerated bending of the blood vessels as they cross the optic disc, resulting in an enlarged optic cup that appears more basinlike compared with a normal cup. The progression of cupping in glaucoma is caused by the gradual loss of retinal nerve fibers and the loss of blood supply.

As the optic nerve damage increases, visual perception decreases. The localized areas of visual loss (i.e., scotomas) represent loss of retinal sensitivity and nerve fiber damage and are measured and mapped on a graph. In patients with glaucoma, the graph has a distinct pattern that is different from other ocular diseases and is useful in establishing the diagnosis. Figure 58-7 shows the visual changes caused by glaucoma.

Medical Management

The aim of all glaucoma treatment is prevention of optic nerve damage. Lifelong therapy is necessary because glaucoma cannot be cured. Treatment focuses on pharmacologic therapy, laser procedures, surgery, or a combination of these approaches, all of which have potential complications and side effects. The object is to achieve the greatest benefit at the least risk, cost, and inconvenience to the patient. Although treatment cannot reverse optic nerve damage, further damage can be controlled. The goal is to maintain an IOP within a range unlikely to cause further damage (Sheybani, Scott, Samuelson, et al., 2020). The initial target for IOP among patients with elevated IOP and those with low-tension glaucoma with progressive visual field loss is typically set at 30% lower than the current pressure. The patient is monitored for changes in the appearance of the optic nerve. If there is evidence of progressive damage, the target IOP is again lowered until the optic nerve shows stability.



Glaucoma

Figure 58-7 • Visual changes associated with glaucoma. Photo courtesy of the National Eye Institute, National Institutes of Health.

TABLE 58-5

Select Medications Used in the Management of
Glaucoma

Medication	Action	Side Effects	Nursing Implications
Cholinergics (miotics) (pilocarpine, carbachol intraocular)	Increase aqueous fluid outflow by contracting the ciliary muscle and causing miosis (constriction of the pupil) and opening of trabecular meshwork	Periorbital pain, blurry vision, difficulty seeing in the dark	Caution patients about diminished vision in dimly lit areas. Pilocarpine can be stored at room temperature for up to 8 wks and then should be discarded.
Beta-blockers (timolol maleate)	Decrease aqueous humor production	Can have systemic effects, including bradycardia, exacerbation of pulmonary disease, and hypotension	Contraindicated in patients with asthma, chronic obstructive pulmonary disease, second- or third-degree heart block, bradycardia, or heart failure; educate patients about punctal occlusion to limit systemic effects (see Chart 58-5).
Alpha-adrenergic agonists (apraclonidine, brimonidine)	Decrease aqueous humor production	Eye redness, dry mouth and nasal passages	Educate patients about punctal occlusion to limit systemic effects (see Chart 58-5).
Carbonic anhydrase inhibitors (acetazolamide, dorzolamide)	Decrease aqueous humor production	Oral medications (acetazolamide) are associated with serious side effects, including anaphylactic reactions, electrolyte loss, depression, lethargy, gastrointestinal upset, impotence, and weight loss; side effects of topical form (dorzolamide) include topical allergy	Do not administer to patients with sulfa allergies; monitor electrolyte levels.
Prostaglandin analogues (latanoprost, bimatoprost)	Increase uveoscleral outflow	Darkening of the iris, conjunctival redness, possible rash	Instruct patients to report any side effects.

Adapted from Comerford, K. C., & Durkin, M. T. (2020). *Nursing 2020 drug handbook*. Philadelphia, PA: Wolters Kluwer.

Pharmacologic Therapy

Medical management of glaucoma relies on systemic and topical ocular medications that lower IOP. Periodic follow-up examinations are essential to monitor IOP, the appearance of the optic nerve, the visual fields, and side effects of medications. Therapy takes into account the patient's health and stage of glaucoma. Comfort, affordability, convenience, lifestyle, and functional ability are factors to consider in the patient's adherence to the medical regimen (Eliopoulos, 2018).

The patient is usually started on the lowest dose of topical medication and then advanced to increased concentrations until the desired IOP level is reached and maintained. Beta-blockers are the preferred initial topical medications because of their efficacy, minimal dosing (can be used once each day), and low cost. One eye is treated first, with the other eye used as a control in determining the efficacy of the medication; once efficacy has been established, treatment of the other eye is started. If the IOP is elevated in both eyes, both are treated. When results are not satisfactory, a new medication is substituted. The main markers of the efficacy of the medication in glaucoma control are lowering of the IOP to the target pressure, stable appearance of the optic nerve head, and the visual field.

Many ocular medications are used to treat glaucoma (see [Table 58-5](#)), including miotics, beta-blockers, alpha₂-agonists (i.e., adrenergic agents), carbonic anhydrase inhibitors, and prostaglandins. Cholinergics (i.e., miotics) increase the outflow of the aqueous humor by affecting ciliary muscle contraction and pupil constriction, allowing flow through a larger opening between the iris and the trabecular meshwork. Beta-blockers and carbonic anhydrase inhibitors decrease aqueous production. Prostaglandin analogues reduce IOP by increasing aqueous humor outflow (Comerford & Durkin, 2020; Norris, 2019).

Surgical Management

Surgery is reserved for patients in whom pharmacologic treatment has not controlled the IOP. This minimally invasive procedure is specifically designed to improve fluid drainage from the eye to balance IOP. By restoring the eye's natural fluid balance, trabeculectomy surgery stabilizes the optic nerve and minimizes further visual field damage (Sheybani et al., 2020). The surgery is performed through a small incision and does not require creation of a permanent hole in the eye wall or an external filtering bleb or an implant.

In laser trabeculoplasty for glaucoma, a laser beam is applied to the inner surface of the trabecular meshwork to open the intratrabecular spaces and

widen the canal of Schlemm, promoting outflow of aqueous humor and decreasing IOP. The procedure is indicated when IOP is inadequately controlled by medications, and it is contraindicated when the trabecular meshwork cannot be fully visualized because of a narrow angle.

In peripheral iridotomy for pupillary block glaucoma, an opening is made in the iris to eliminate the pupillary blockage. Laser iridotomy is contraindicated in patients with corneal edema, which interferes with laser targeting and strength. Potential complications include burns to the cornea, lens, or retina; transient elevated IOP; closure of the iridotomy; uveitis; and blurring.

Filtering procedures for glaucoma are used to create an opening or fistula in the trabecular meshwork to drain aqueous humor from the anterior chamber to the subconjunctival space into a bleb (fluid collection on the outside of the eye), thereby bypassing the usual drainage structures. This allows the aqueous humor to flow and exit by different routes (i.e., absorption by the conjunctival vessels or mixing with tears). Trabeculectomy is the standard filtering technique used to remove part of the trabecular meshwork (Shaw & Lee, 2017). Complications include hemorrhage, an extremely low (hypotony) or extremely elevated IOP, uveitis, cataracts, bleb failure, bleb leak, and **endophthalmitis** (i.e., intraocular infection).

Drainage implants or shunts are tubes implanted in the anterior chamber to shunt aqueous humor to the episcleral plate in the conjunctival space. Implants are used when failure has occurred with one or more trabeculectomies in which antifibrotic agents were used. A fibrous capsule develops around the episcleral plate and filters the aqueous humor, thereby regulating the outflow and controlling IOP.

Nursing Management



Nurses in all settings encounter patients with glaucoma. Even patients with long-standing disease and those with glaucoma as a secondary diagnosis should be assessed for knowledge level and adherence to their prescribed therapeutic regimen.

Promoting Home, Community-Based, and Transitional Care



Educating Patients About Self-Care

The medical and surgical management of glaucoma slows the progression of the disease but does not cure it. The lifelong therapeutic regimen mandates patient education. The nature of the disease and the importance of strict adherence to the medication regimen must be included in an individualized

education plan. A structured self-management program may increase adherence to the treatment regime. A thorough discussion of the medication program, particularly the interactions of glaucoma-control medications with other medications, is essential. For example, the diuretic effect of acetazolamide may have an additive effect on the diuretic effects of other antihypertensive medications (Comerford & Durkin, 2020). The effects of glaucoma-control medications on vision must also be explained. Miotics and sympathomimetics result in altered focus; therefore, patients need to be cautious in navigating their surroundings. [Chart 58-5](#) presents patient education about instilling eye medications and preventing systemic absorption with punctal occlusion. [Chart 58-7](#) contains additional educational information to review with patients with glaucoma.

Continuing and Transitional Care

Patients with severe glaucoma and impaired function may need referral to home, community-based or transitional services that provide assistance in the home. The loss of peripheral vision impairs mobility the most. These patients also benefit from a referral for low vision and rehabilitation services. Patients who meet the criteria for legal blindness should be offered referrals to agencies that can assist them in obtaining federal assistance.

Chart 58-7



PATIENT EDUCATION

Managing Glaucoma

The nurse instructs the patient to:

- Know your intraocular pressure measurement and the desired range.
- Be informed about the extent of your vision loss and optic nerve damage.
- Keep a record of your eye pressure measurements and visual field test results to monitor your own progress.
- Review all of your medications (including over-the-counter and herbal medications) with your ophthalmologist, and mention any side effects each time you visit.
- Ask about potential side effects and drug interactions of your eye medications.
- Ask whether generic or less costly forms of your eye medications are available.
- Review the dosing schedule with your ophthalmologist, and inform them if you have trouble following the schedule.
- Participate in the decision-making process. Let your primary provider know what dosing schedule works for you and other preferences regarding your eye care.
- Have the nurse observe you instilling eye medication to determine whether you are administering it properly (see Chart 58-5).
- Be aware that glaucoma medications can cause adverse effects if used inappropriately. Eye drops are to be given as prescribed, not when eyes feel irritated.
- Ask your ophthalmologist to send a report to your primary provider after each appointment.
- Keep all follow-up appointments.

Reassurance and emotional support are important aspects of care. A lifelong disease involving possible loss of sight has psychological, physical, social, and vocational ramifications. The family must be integrated into the plan of care, and because the disease has a familial tendency, family members should be encouraged to undergo examinations at least once every 2 years to detect glaucoma early.

CATARACTS

A **cataract** is a lens opacity or cloudiness (see Fig. 58-8). Cataracts are responsible for visual disability in 18 million people worldwide (Norris, 2019). By 80 years of age, more than half of all Americans have cataracts. Cataracts are a leading cause of blindness in the world (Prevent Blindness America, 2020).

Pathophysiology

Cataracts can develop in one or both eyes at any age. The three most common types are traumatic, congenital, or senile cataract (Norris, 2019). There are a variety of risk factors, the most common one being age (see [Chart 58-8](#)).



Figure 58-8 • A cataract is a cloudy or opaque lens. On visual inspection, the lens appears gray or milky. Reprinted with permission from Strayer, D. S., Rubin, E., Saffitz, J. E., et al. (2015). *Rubin's pathology: Clinicopathologic foundations of medicine* (7th ed., Fig. 33.2). Philadelphia, PA: Wolters Kluwer.

Clinical Manifestations

Painless, blurry vision is characteristic of cataracts. The person perceives that surroundings are dimmer, as if their glasses need cleaning. Light scattering is common, and the person experiences reduced contrast sensitivity, sensitivity to glare, and reduced visual acuity. Other effects include myopic shift (return of ability to do close work [e.g., reading fine print] without eyeglasses), **astigmatism** (refractive error due to an irregularity in the curvature of the cornea), monocular **diplopia** (double vision), and color changes as lens becomes more brown in color (Eliopoulos, 2018; Shaw & Lee, 2017).

Assessment and Diagnostic Findings

Decreased visual acuity is directly proportionate to cataract density. The Snellen visual acuity test, ophthalmoscopy, and slit-lamp biomicroscopic examination are used to establish the degree of cataract formation. The degree of lens opacity does not always correlate with the patient's functional status.

Some patients can perform normal activities despite clinically significant cataracts. Others with less lens opacification have a disproportionate decrease in visual acuity; hence, visual acuity is an imperfect measure of visual impairment.

Medical Management

No nonsurgical treatment (e.g., medications, eye drops, eyeglasses) cures cataracts or prevents age-related cataracts. Optimal medical management is prevention. Patients should be educated by primary providers about risk reduction strategies such as smoking cessation, weight reduction, optimal blood glucose control for patients with diabetes, and should be advised to wear sunglasses outdoors to prevent early cataract formation (Shaw & Lee, 2017).

Surgical Management

In general, if reduced vision from cataract does not interfere with normal activities, surgery may not be needed. In deciding when cataract surgery is to be performed, the patient's functional and visual status should be a primary consideration (Eliopoulos, 2018). Cataract removal is common, with more than one million such surgeries performed in the United States each year (Prevent Blindness America, 2020). Surgery is performed on an outpatient basis and usually takes less than 1 hour, with the patient being discharged in 30 minutes or less afterward. Although complications from cataract surgery are uncommon, they can have significant effects on vision (see [Table 58-6](#)). Restoration of visual function through a safe and minimally invasive procedure is the surgical goal, which is achieved with advances in topical anesthesia, smaller wound incision (i.e., clear cornea incision), and lens design (i.e., foldable and more accurate intraocular lens [IOL] measurements).

Chart 58-8 RISK FACTORS

Cataract Formation

Aging

- Accumulation of a yellow-brown pigment due to the breakdown of lens protein
- Clumping or aggregation of lens protein (which leads to light scattering)
- Decreased oxygen uptake
- Decrease in levels of vitamin C, protein, and glutathione (an antioxidant)
- Increase in sodium and calcium
- Loss of lens transparency

Associated Ocular Conditions

- Infection (e.g., herpes zoster, uveitis)
- Myopia
- Retinal detachment and retinal surgery
- Retinitis pigmentosa

Toxic Factors

- Alkaline chemical eye burns, poisoning
- Aspirin use
- Calcium, copper, iron, gold, silver, and mercury, which tend to deposit in the pupillary area of the lens
- Cigarette smoking
- Corticosteroids, especially at high doses and in long-term use
- Ionizing radiation

Nutritional Factors

- Obesity
- Poor nutrition
- Reduced levels of antioxidants

Physical Factors

- Blunt trauma, perforation of the lens with a sharp object or foreign body, electric shock
- Dehydration associated with chronic diarrhea, the use of purgatives in anorexia nervosa, and the use of hyperbaric

- oxygenation
- Ultraviolet radiation in sunlight and x-ray

Systemic Diseases and Syndromes

- Diabetes
- Disorders related to lipid metabolism
- Down syndrome
- Musculoskeletal disorders
- Renal disorders

Adapted from Norris, T. (2019). *Porth's pathophysiology: Concepts of altered health status* (10th ed.). Philadelphia, PA: Wolters Kluwer; Prevent Blindness. (2020). Know the risk factors for cataract. Retrieved on 2/16/2020 at: www.preventblindness.org/know-risk-factors-cataract

TABLE 58-6 Potential Complications of Cataract Surgery

Complication	Effects	Management and Outcome
Immediate Preoperative		
Retrobulbar hemorrhage —can result from retrobulbar infiltration of anesthetic agents if the short ciliary artery is located by the injectia	Increased IOP, proptosis, lid tightness, and subconjunctival hemorrhage with or without edema	Emergent lateral canthotomy (slitting of the canthus) is performed to stop central retinal perfusion when the IOP is dangerously elevated. If this procedure fails to reduce IOP, a puncture of the anterior chamber with removal of fluid is considered. The patient must be closely monitored for at least a few hours. Postponement of cataract surgery for 2–4 wks is advised. Complications such as iris prolapse, vitreous loss, and choroidal hemorrhage could result in a catastrophic visual outcome.
Intraoperative		
Rupture of the posterior capsule	May result in loss of vitreous	Anterior vitrectomy is required if vitreous loss occurs.
Suprachoroidal (expulsive) hemorrhage—profuse bleeding into the suprachoroidal space	Extrusion of intraocular contents from the eye or opposition of retinal surfaces	Closure of the incision and administration of a hyperosmotic agent to reduce IOP or corticosteroids to reduce intraocular inflammation. Vitrectomy is performed 1–2 wks later. Visual prognosis is poor; some useful vision may be salvaged on rare occasions.
Early Postoperative		
Acute bacterial endophthalmitis—devastating complication that occurs in about 1 in 1000 cases; the most common causative organisms are <i>Staphylococcus epidermidis</i> , <i>Staphylococcus aureus</i> , <i>Pseudomonas</i> , and <i>Proteus</i> species	Characterized by marked visual loss, pain, lid edema, hypopyon, corneal haze, and chemosis	Managed by aggressive antibiotic therapy. Broad-spectrum antibiotics are given while awaiting culture and sensitivity results. Once results are obtained, the appropriate antibiotics are given via intravitreal injection. Corticosteroid therapy is also given.
Toxic anterior segment syndrome—noninfectious inflammation that is a complication of anterior chamber	Corneal edema occurs <24 h after surgery; symptoms include reduced	If there is no growth of microorganisms, the treatment is topical steroids alone.

surgery; caused by a toxic agent such as an agent used to sterilize surgical instruments

visual acuity and pain

Late Postoperative

Suture-related problems	Toxic reactions or mechanical injury from broken or loose sutures	Suture removal relieves the symptoms. Topical corticosteroids are used when the incision is not healed and sutures cannot be removed.
Malposition of the IOL	Results in astigmatism, sensitivity to glare, or appearance of halos	Miotics are used for mild cases, whereas IOL removal and replacement is necessary for severe cases.
Chronic endophthalmitis	Persistent, low-grade inflammation, and granuloma	Corticosteroids and antibiotics are given systemically. If the condition persists, removal of the IOL and capsular bag, vitrectomy, and intravitreal injection of antibiotics are required.
Opacification of the posterior capsule—most common late complication of extracapsular cataract extraction	Visual acuity is diminished	Nd:YAG laser is used to create a hole in the posterior capsule. Blurred vision is cleared immediately.

IOL, intraocular lens; IOP, intraocular pressure; Nd:YAG, Neodymium: yttrium aluminum garnet.

Adapted from Shaw, M., & Lee, A. (2017). *Ophthalmic nursing* (5th ed.). Boca Raton, FL: CRC Press Taylor & Francis Group.

Injection-free topical and intraocular anesthesia, such as 1% lidocaine gel applied to the surface of the eye, eliminates the hazards of regional (retrobulbar and peribulbar) anesthesia, such as ocular perforation, retrobulbar hemorrhage, optic injuries, diplopia, and ptosis, and is ideal for patients receiving anticoagulants. Furthermore, patients can communicate and cooperate during surgery. IV sedation may be used to minimize anxiety and discomfort.

When both eyes have cataracts, one eye is treated first, with at least several weeks, preferably months, separating the two procedures. Because cataract surgery is performed to improve visual functioning, the delay for the other eye gives time for the patient and the surgeon to evaluate whether the results from the first surgery are adequate to preclude the need for a second operation. The delay also provides time for the first eye to recover; if there are any

complications, the surgeon may decide to perform the second procedure differently.

Phacoemulsification

In this method of extracapsular cataract surgery, a portion of the anterior capsule is removed, allowing extraction of the lens nucleus and cortex while the posterior capsule and zonular support are left intact. An ultrasonic device is used to liquefy the nucleus and cortex, which are then suctioned out through a tube. An intact zonular–capsular diaphragm provides the needed safe anchor for the posterior chamber IOL. The pupil is dilated to 7 mm or greater (Shaw & Lee, 2017). The surgeon makes a small incision on the upper edge of the cornea and a viscoelastic substance (clear gel) is injected into the space between the cornea and the lens. This prevents the space from collapsing and facilitates insertion of the IOL. Because the incision is smaller than the manual extracapsular cataract extraction, the wound heals more rapidly, and there is early stabilization of refractive error and less astigmatism.

Lens Replacement

After removal of the crystalline lens, the patient is referred to as aphakic (i.e., without lens). The lens, which focuses light on the retina, must be replaced for the patient to see clearly. There are three lens replacement options: aphakic eyeglasses, contact lenses, and IOL implants.

Aphakic glasses, although effective, are rarely used. Objects are magnified by 25%, making them appear closer than they actually are. This magnification creates distortion. Peripheral vision is also limited, and **binocular vision** (i.e., ability of both eyes to focus on one object and fuse the two images into one) is impossible if the other eye is aphakic (without a natural lens).

Contact lenses provide patients with almost normal vision, but because contact lenses need to be removed occasionally, the patient also needs a pair of aphakic glasses. Contact lenses are not advised for patients who have difficulty inserting, removing, and cleaning them. Frequent handling and improper disinfection increase the risk of infection.

Insertion of IOLs during cataract surgery is the most common approach to lens replacement (Eliopoulos, 2018). After cataract extraction, or phacoemulsification, the surgeon implants an IOL. Cataract extraction and posterior chamber IOLs are associated with a relatively low incidence of complications (e.g., eye infection, loss of vitreous humor, slipping of the implant) (Eliopoulos, 2018). IOL implantation is contraindicated in patients with recurrent uveitis, proliferative diabetic retinopathy, neovascular glaucoma, or rubeosis iridis.

Nursing Management

Providing Preoperative Care

The patient with cataracts receives the usual preoperative care for ambulatory surgical patients undergoing eye surgery. The standard battery of preoperative tests (e.g., complete blood count, electrocardiogram, urinalysis) commonly performed for most surgeries is prescribed only if indicated by the patient's medical history.

Alpha-antagonists (particularly tamsulosin, which is used for treatment of enlarged prostate) are known to cause a condition called *intraoperative floppy iris syndrome*. Alpha-antagonists can interfere with pupil dilation during the surgical procedure, resulting in miosis and iris prolapse and leading to complications. Intraoperative floppy iris syndrome can occur even though a patient has stopped taking the drug. The nurse needs to ask patients about a history of taking alpha-antagonists. Surgical team members are then alerted to the risk of this complication (Comerford & Durkin, 2020).

Dilating drops are given prior to surgery. Nurses in the ambulatory surgery setting begin patient education about eye medications (antibiotic, corticosteroid, and anti-inflammatory drops) that will need to be self-administered to prevent postoperative infection and inflammation.

Providing Postoperative Care

Before discharge, the patient receives verbal and written education regarding eye protection, administration of medications, recognition of complications, activities to avoid, and obtaining emergency care (see [Chart 58-9](#)). An eye shield is usually worn at night for the first week to avoid injury. The nurse also explains that there should be minimal discomfort after surgery and educates the patient about taking a mild analgesic agent, such as acetaminophen, as needed. Antibiotic, anti-inflammatory, and corticosteroid eye drops or ointments are prescribed postoperatively. Patients prescribed anti-inflammatory or corticosteroid eye drops are monitored for possible increases in IOP (Phulke, Kaushik, Kaur, et al., 2017).

Chart 58-9



HOME CARE CHECKLIST

Intraocular Lens Implant

At the completion of education, the patient and/or caregiver will be able to:

- Name the procedure that was performed and identify any permanent changes in anatomic structure or function as well as changes in ADLs, IADLs, roles, relationships, and spirituality.
- State the name, dose, side effects, frequency, and schedule for all medications.
- Describe ongoing postoperative therapeutic regimen and activities to limit or avoid (e.g., lifting weights, driving a car, engaging in contact sports).
 - Wear glasses or eye shield following surgery as instructed.
 - Always wash hands before touching or cleaning the postoperative eye.
 - Clean postoperative eye with a clean tissue; wipe the closed eye with a single gesture from the inner canthus outward.
 - When bathing or showering, shampoo hair cautiously or seek assistance.
 - Avoid lying on the side of the affected eye the night after surgery.
 - Keep activity light (e.g., walking, reading, watching television). Resume the following activities only as directed by the ophthalmologist: driving, sexual activity, unusually strenuous activity.
 - Avoid lifting, pushing, or pulling objects heavier than 15 lb.
 - Avoid bending or stooping for an extended period.
 - Be careful when climbing or descending stairs.
- Describe signs and symptoms of complications (e.g., vision change, continuous flashing lights appear to the affected eye, redness, swelling or increased pain near the eye, change in amount or type of eye drainage, any eye injury, significant pain not relieved by prescribed analgesia).
- Relate how to reach ophthalmologist with questions or complications.
- State time and date of follow-up appointments.
- Identify sources of assistance (e.g., meals, transportation) and support (e.g., friends, relatives, faith community).
- Identify the need for health promotion, disease prevention, and screening activities

ADLs, activities of daily living; IADLs, instrumental activities of daily living.

Promoting Home, Community-Based, and Transitional Care



Educating Patients About Self-Care

To prevent inadvertent rubbing or poking of the eye, the patient wears a protective eye patch for about the first 24 hours after surgery, followed by eyeglasses worn during the day and an eye shield at night. The nurse educates the patient and family about applying and caring for the eye shield, if one is recommended. Sunglasses should be worn while outdoors during the day because the eye is sensitive to light.

Slight morning discharge, some redness, and a scratchy feeling may be expected for a few days. A clean, damp washcloth may be used to remove slight morning eye discharge. Because cataract surgery increases the risk of retinal detachment, the patient must know to notify the surgeon if new floaters (moving dots) in vision, flashing lights, decrease in vision, pain, or increase in redness occur.

Continuing and Transitional Care

If an eye patch is worn, it is removed after the first follow-up appointment, which should occur within 48 hours of surgery. Nurses should educate patients about the importance of keeping their follow-up appointments, because monitoring of visual status and prompt intervention of postoperative complications enhance good visual outcome. Vision is stabilized when the eye is completely healed, usually within 6 to 12 weeks, when final corrective prescription is completed. Visual correction may still be needed for any remaining refractive errors. Patients who choose multifocal IOLs should be aware that increased night glare and contrast sensitivity may occur.

CORNEAL DISORDERS

Corneal Dystrophies

Corneal dystrophies are inherited as autosomal dominant traits and manifest when the person is about 20 years of age. They are characterized by deposits in the corneal layers. Decreased vision is caused by the irregular corneal surface and corneal deposits. Corneal endothelial decomposition leads to corneal edema and blurring of vision. Persistent edema leads to bullous keratopathy or the formation of blisters that cause pain and discomfort on rupturing. The two main types are keratoconus and Fuchs endothelial dystrophy.

Keratoconus

Keratoconus, the most common type of corneal dystrophy, is characterized by a conical protuberance of the cornea with progressive thinning on protrusion and irregular astigmatism. This hereditary condition has a higher incidence among women. Onset occurs at puberty; the condition may progress for more than 20 years and is bilateral. Corneal scarring occurs in severe cases. Blurred vision is a prominent symptom. Rigid, gas-permeable contact lenses correct irregular astigmatism and improve vision. Advances in contact lens design have reduced the need for surgery. Penetrating keratoplasty (PKP) is indicated when contact lens correction is no longer effective (Allard & Zetterberg, 2018).

Fuchs Endothelial Dystrophy

Fuchs (pronounced *Fooks*) dystrophy is manifested by a slow death of cells in the endothelial cornea. It affects women more than men and is usually not noted until 50 years of age. Corneal endothelial cell death leads to corneal edema and blurring of vision. Persistent edema leads to bullous keratopathy. A bandage contact lens is used to flatten the bullae, protect the exposed corneal nerve endings, and relieve discomfort. Symptomatic treatments, such as hypertonic drops or ointment (5% sodium chloride), may reduce epithelial edema. Currently, the only cure is a corneal transplant (Moshirfar, Ding, & Shah, 2018).

Corneal Surgeries

Among the surgical procedures used to treat diseased corneal tissue are phototherapeutic keratectomy (PTK), PKP and corneal endothelial transplantation, and Descemet's stripping endothelial keratoplasty (DSEK).

Phototherapeutic Keratectomy

PTK is a laser procedure that is used to treat diseased corneal tissue by removing or reducing corneal opacities and smoothing the anterior corneal surface to improve functional vision. PTK is contraindicated in patients with active herpetic keratitis because the ultraviolet rays may reactivate latent virus. Common side effects are induced hyperopia and stromal haze. Complications are delayed re-epithelialization (particularly in patients with diabetes) and bacterial keratitis. Postoperative management consists of oral analgesic agents for eye pain. Re-epithelialization is promoted with a pressure patch or therapeutic soft contact lens. Antibiotic and corticosteroid ointments and NSAIDs are prescribed postoperatively. Follow-up examinations are required for up to 2 years.

Penetrating Keratoplasty

PKP (corneal transplantation or corneal grafting) involves replacing abnormal host tissue with healthy donor (cadaver) corneal tissue. Common indications are keratoconus, corneal scarring from herpes simplex, keratitis, and chemical burns.

Several factors affect the success of the graft: the condition of the ocular structures (e.g., lids, conjunctiva), quality of the tears, adequacy of blinking, and viability of the donor endothelium. Contraindications for the use of donor tissue are outlined in [Chart 58-10](#).

In PKP, the surgeon determines the graft size before the procedure, and the appropriate size is marked on the surface of the cornea. The surgeon prepares the donor cornea and the recipient bed, removes the diseased cornea, places the donor cornea on the recipient bed, and sutures it in place. Sutures remain in place for 12 to 18 months and are then removed. Potential complications include early graft failure due to poor quality of donor tissue, surgical trauma, acute infection, and persistently increased IOP and late graft failure due to rejection.

Chart 58-10

Contraindications to the Use of Donor Tissue for Corneal Transplantation: Donor Characteristics

Systemic Disorders

- Death from unknown cause
- History or suspected history of the following:
 - Acquired immune deficiency syndrome or high risk for human immune deficiency virus infection
 - Creutzfeldt–Jakob disease
 - Eye infection
 - Hepatitis
 - Rabies
 - Systemic infection

Intrinsic Eye Disease

- Disorders of the conjunctiva or corneal surface involving the optical zone of the cornea
- Malignant tumors of anterior segment
- Ocular inflammation
- Retinoblastoma

Other

- Corneal scars
- History of eye trauma
- Previous surgical eye procedures such as corneal graft or LASIK eye surgery

Postoperatively, the patient receives mydriatics for 2 weeks and topical corticosteroids for 12 months (daily doses for 6 months and tapered doses thereafter). These mydriatics and corticosteroid drops should be preservative free to prevent a reactive inflammation. Patients typically describe a sensation of postoperative eye discomfort rather than acute pain.

Additional Keratoplasty Surgeries

For many years, PKP was the standard of care for patients with corneal endothelial failure but with poor refractive results. Subsequently, other techniques have been developed. Anterior lamellar keratoplasty (ALK) involves a partial-thickness graft for disorders such as anterior corneal dystrophies or scarring not involving the endothelial portion of the cornea. Deep anterior lamellar keratoplasty (DALK) involves replacement of only anterior corneal layers and not Descemet's membrane or endothelial layers for

disorders such as keratoconus. Posterior lamellar keratoplasty (PLK or EK) and DSEK (which replaces only the endothelial layer of the cornea) are procedures indicated for conditions such as Fuchs endothelial dystrophy or bullous keratopathy (Moshirfar et al., 2018). In DSEK, layers of the cornea are dissected and selectively replaced by donor cornea tissue. DSEK offers several advantages, such as less postoperative astigmatism, faster visual recovery, and stronger wound integrity. Theoretically, the risk of rejection is less because less of the patient's tissue is replaced.

Keratoprosthesis

An additional therapeutic option for patients with multiple graft failures or severe corneal disease is keratoprosthesis (artificial cornea; e.g., U.S. Food and Drug Administration [FDA]-approved Boston Keratoprosthesis [KPro] and AlphaCor). The design of the keratoprosthesis consists of a central optic core and an outer rim that secures the prosthesis. This procedure has serious potential complications (e.g., glaucoma, endophthalmitis) and requires close follow-up and monitoring (Shalaby Bardan, Al Raqqad, Zarei-Ghanavati, et al., 2018).

Nursing Management

For corneal surgeries, the nurse reinforces instructions regarding visual rehabilitation and visual improvement. A technically successful graft may initially produce disappointing results because the procedure has produced a new optical surface. Only after several months do patients start seeing the natural and true colors of their environment. Correction of a resultant refractive error with eyeglasses or contact lenses determines the final visual outcome. The nurse assesses the patient's support system and their ability to comply with long-term follow-up, which includes frequent clinic visits for several months for tapering of topical corticosteroid therapy, selective suture removal, and ongoing evaluation of the graft site and visual acuity. The nurse also initiates appropriate referrals to community services as indicated.

Because graft failure is an ophthalmic emergency that can occur at any time, the primary goal of nursing care is to educate the patient to identify signs and symptoms of graft failure. The early symptoms are blurred vision, discomfort, tearing, or redness of the eye. Decreased vision results after graft destruction. The patient must contact the ophthalmologist as soon as symptoms occur. Treatment of graft rejection usually involves prompt administration of hourly topical corticosteroids and periocular corticosteroid injections. Systemic immunosuppressive agents may be necessary for severe, resistant cases.

Refractive Surgeries

Refractive surgeries are elective procedures performed to correct refractive errors (myopia or hyperopia) and astigmatism by reshaping the cornea (Gomel, Negari, Frucht-Pery, et al., 2018). Laser vision correction alters the major optical function of the eye and carries risks. Refractive surgery does not alter the normal aging process of the eye. If the reason for the procedure is to meet vision requirements for the patient's occupation, the results must satisfy both the patient and the employer. Precise visual outcome cannot be guaranteed.

The corneal structure must be normal, and the refractive error must be stable. The patient is required to discontinue using contact lenses for a period before the procedure (2 to 3 weeks for soft lenses and 4 weeks for hard lenses). Patients with conditions that are likely to adversely affect corneal wound healing (e.g., corticosteroid use, immunosuppression, elevated IOP) are not good candidates for the procedure. Any superficial eye disease must be diagnosed and fully treated before a refractive procedure.

Patient satisfaction is the ultimate goal; therefore, patient education and counseling about potential risks, complications, and postoperative follow-up are critical. Minimal postoperative care includes topical corticosteroid or NSAID and antibiotic drops.

Laser Vision Correction Photorefractive Keratectomy

Photorefractive keratectomy (PRK) is used to treat myopia and hyperopia with or without astigmatism (Gomel et al., 2018). The excimer laser is applied directly to the cornea according to carefully calculated measurements. For myopia, the relative curvature is decreased; for hyperopia, the relative curvature is increased. A bandage contact lens is placed over the cornea to promote epithelial healing and reduce pain, which is similar to that of a severe corneal abrasion.

Laser-Assisted In Situ Keratomileusis

LASIK involves flattening the anterior curvature of the cornea by removing a stromal lamella or layer. The surgeon creates a corneal flap with a microkeratome, which is an automatic corneal shaper. The surgeon retracts a flap of corneal tissue less than one third the thickness of a human hair to access the corneal stroma and then uses the excimer laser on the stromal bed to reshape the cornea according to calculated measurements (see Fig. 58-9). Data are insufficient to determine whether LASIK or PRK is better at correcting nearsightedness (Gomel et al., 2018). There are few adverse outcomes of either procedure.

Perioperative Complications

Surgically Induced Abnormalities

Corneal surface irregularities can occur after LASIK treatment. These include central islands (central areas of stiffness or elevation), decentered ablations resulting from misalignment of the laser treatment or from involuntary eye movement during laser treatment, and forms of irregular astigmatism. Symptoms of central islands and decentered ablations include monocular diplopia or ghost images, halos, glare, and decreased visual acuity.

Phakic Intraocular Lenses

Increasingly, phakic IOL implantation has been used for patients with moderate to severe myopia (Igarashi, 2019). Phakic IOLs may be used in either the anterior or posterior chamber. The implantation of such devices is reversible because the natural lens is left in place and the normal architecture of the cornea is preserved. This procedure provides more predictable refractive results than procedures that alter the corneal curvature, is safer, and has higher patient satisfaction scores (Igarashi, 2019). Potential complications include cataract, iritis or uveitis, endothelial cell loss, and increased IOP.

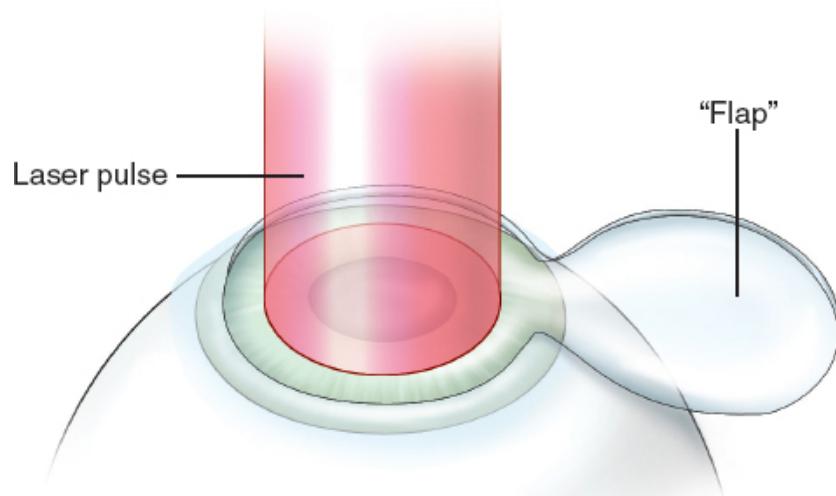


Figure 58-9 • LASIK combines delicate surgical procedures and laser treatment. A flap is surgically created and lifted to one side. A laser is then applied to the cornea to reshape it. With permission from the Wilmer Laser Vision Center, Lutherville, MD.

Conductive Keratoplasty

Another innovation in refractive surgery for the correction of low to mild hyperopia uses the principles of thermal keratoplasty by applying

radiofrequency current to the peripheral cornea using a thin, handheld probe. It does not involve the removal of cornea tissue.

RETINAL DISORDERS

Although the retina is composed of multiple microscopic layers, the two innermost layers—the sensory retina and the retinal pigment epithelium—are most commonly implicated in retinal disorders (Norris, 2019).

Retinal Detachment

Retinal detachment refers to the separation of the retinal pigment epithelium from the neurosensory layer (Norris, 2019). The four types of retinal detachment are rhegmatogenous, traction, a combination of rhegmatogenous and traction, and exudative. *Rhegmatogenous retinal detachment* is the most common form (Liao & Zhu, 2019). In this condition, a hole or tear develops in the sensory retina, allowing some of the liquid vitreous to seep through the sensory retina and detach it from the retinal pigment epithelium (see [Fig. 58-10](#)). People at risk for this type of detachment include those with high myopia or those who have aphakia (absence of the natural lens) after cataract surgery. Trauma may also play a role in rhegmatogenous retinal detachment. Between 5% and 10% of all rhegmatogenous retinal detachments are associated with proliferative retinopathy—a retinopathy associated with diabetic neovascularization (see [Chapter 46](#)).

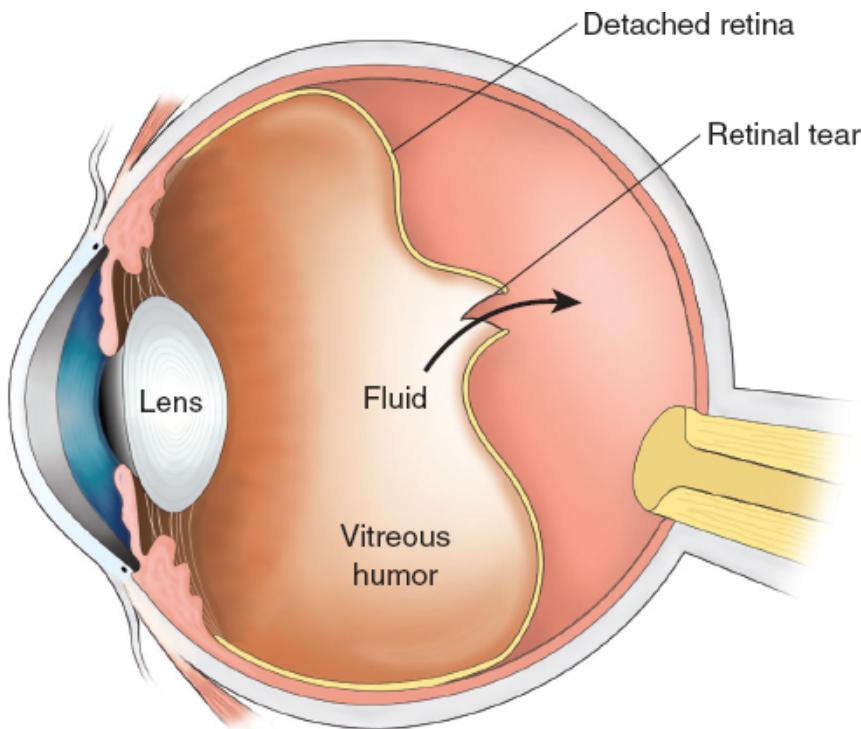


Figure 58-10 • Retinal detachment.

Tension, or a pulling force, is responsible for *traction retinal detachment*. An ophthalmologist must ascertain all of the areas of retinal break and identify and release the scars or bands of fibrous material providing traction on the retina. In general, patients with this condition have developed fibrous scar tissue from conditions such as diabetic retinopathy, vitreous hemorrhage, or the retinopathy of prematurity. The hemorrhages and fibrous proliferation associated with these conditions exert a pulling force on the delicate retina.

Patients can have both rhegmatogenous and traction retinal detachment. *Exudative retinal detachments* are the result of the production of a serous fluid under the retina from the choroid. Conditions such as uveitis and macular degeneration may cause the production of this serous fluid.

Clinical Manifestations

Patients may report the sensation of a shade or curtain coming across the vision of one eye, cobwebs, bright flashing lights, or the sudden onset of a great number of floaters. Patients do not complain of pain but retinal detachment is an ocular emergency, requiring immediate surgical intervention for optimal outcomes.

Assessment and Diagnostic Findings

After visual acuity is determined, the patient must have a dilated fundus examination using an indirect ophthalmoscope as well as slit-lamp biomicroscopy. Stereo fundus photography and fluorescein angiography are commonly used during the evaluation.

Increasingly, optical coherence tomography and ultrasound are used for the complete retinal assessment, especially if the view is obscured by a dense cataract or vitreal hemorrhage. All retinal breaks, all fibrous bands that may be causing traction on the retina, and all degenerative changes must be identified.

Surgical Management

In rhegmatogenous retinal detachment, an attempt is made to surgically reattach the sensory retina to the retinal pigment epithelium. In traction retinal detachment, the source of traction must be removed and the sensory retina reattached. The most commonly used surgical interventions are the scleral buckle and vitrectomy (Park, Lee, & Lee, 2018).

Scleral Buckle

The retinal surgeon compresses the sclera (often with a scleral buckle [see Fig. 58-11] or a silicone band) to indent the scleral wall from the outside of the eye and bring the two retinal layers in contact with each other (Park et al., 2018).

Vitrectomy

A vitrectomy is an intraocular procedure that allows the introduction of a light source through an incision; a second incision serves as the portal for the vitrectomy instrument. The surgeon dissects preretinal membranes under direct visualization while the retina is stabilized by an intraoperative vitreous substitute.

Traction on the retina may be relieved through vitrectomy and may be combined with scleral buckling to repair retinal detachments. A gas bubble, silicone oil, or perfluorocarbon and liquids may be injected into the vitreous cavity to help push the sensory retina up against the retinal pigment epithelium.

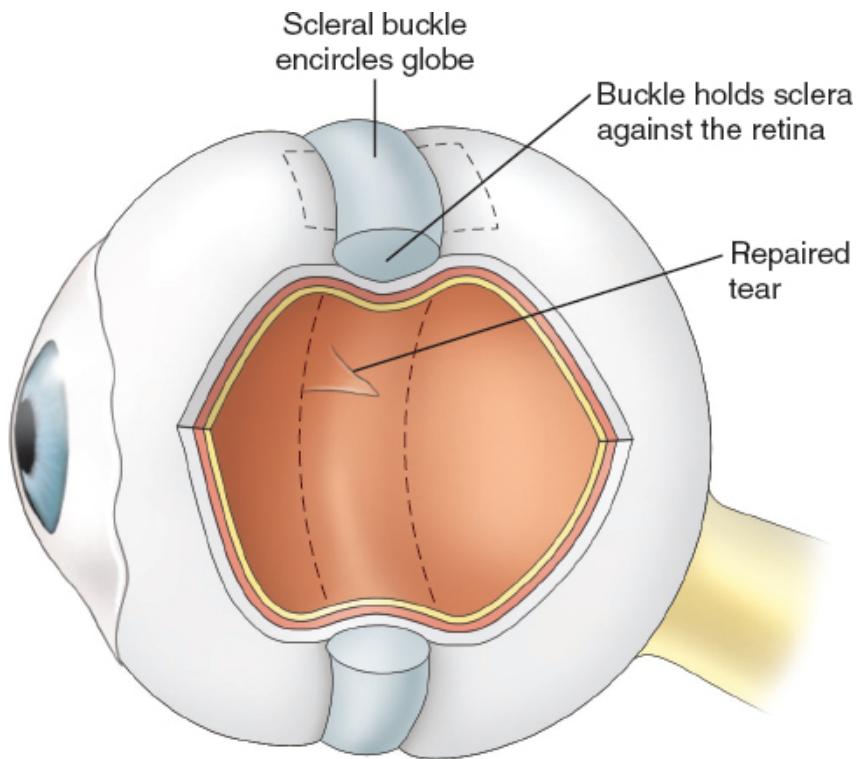


Figure 58-11 • Scleral buckle.

Nursing Management

Nursing management consists of educating the patient and providing supportive care. Postoperative positioning of the patient is critical when a gas bubble is used because the injected bubble must remain in position overlying the area of detachment, providing consistent pressure to reattach the sensory retina. The patient must maintain a prone position that would allow the gas bubble to act as a tamponade for the retinal break (Shaw & Lee, 2017). Patients and family members should be made aware of these needs beforehand so that the patient can be made as comfortable as possible.

In most cases, vitreoretinal procedures are performed on an outpatient basis, and the patient is seen the next day for a follow-up examination. Postoperative complications may include increased IOP, endophthalmitis, retinal detachment, and development of cataracts. Patients must be educated about the signs and symptoms of complications, particularly of increasing IOP and postoperative infection. Contact details for the ophthalmic team are provided and the patient is encouraged to call immediately if complications occur.

Retinal Vascular Disorders

Loss of vision can occur from occlusion of a retinal artery or vein. Such occlusions may result from atherosclerosis, valvular heart disease, venous stasis, hypertension, or increased blood viscosity. Associated risk factors include diabetes, glaucoma, and aging.

Central Retinal Vein Occlusion

Blood supply to and from the ocular fundus is provided by the central retinal artery and vein. Central retinal vein occlusions (CRVOs) are found most often in people older than 50 years. Patients who suffer a CRVO report decreased visual acuity ranging from mild blurring to severely limited.

Direct ophthalmoscopy of the retina shows optic disc swelling, venous dilation and tortuousness, retinal hemorrhages, cotton-wool spots, and a bloody appearance of the retina. The better the initial visual acuity, the better is the general prognosis.

Fluorescein angiography may show extensive areas of capillary closure. The patient should be monitored carefully over the ensuing several months for signs of neovascularization and neovascular glaucoma. Laser panretinal photocoagulation may be necessary to treat the abnormal neovascularization. Neovascularization of the iris may cause neovascular glaucoma. Macular edema, macular nonperfusion, and vitreous hemorrhage from the neovascularization are among the potential complications of CRVO (Schmidt-Erfurth, Garcia-Arumi, Gerendas, et al., 2019).

Branch Retinal Vein Occlusion

Some patients with branch retinal vein occlusion (BRVO) are symptom free, whereas others complain of a sudden loss of vision if the macular area is involved. A more gradual loss of vision may occur if macular edema associated with BRVO develops.

On examination, the ocular fundus appears similar to that found in CRVO. The occlusions generally occur at the arteriovenous crossings. The diagnostic evaluation and follow-up assessments and complications are the same as for CRVO. Associated conditions include glaucoma, systemic hypertension, diabetes, and hyperlipidemia (Schmidt-Erfurth et al., 2019).

Central Retinal Artery Occlusion

Patients with central retinal artery occlusion, a relatively rare disorder that accounts for approximately 1 in 10,000 ophthalmologic visits, present with a sudden loss of vision. Visual acuity is reduced to being able to count the examiner's fingers, or the field of vision is tremendously restricted. A relative afferent pupillary defect is present. Examination of the ocular fundus reveals a

pale retina with a cherry-red spot at the fovea. The retinal arteries are thin, and emboli are occasionally seen in the central retinal artery or its branches. Central retinal artery occlusion is a true ocular emergency. Various treatments may include ocular massage, anterior chamber paracentesis, hyperbaric oxygen therapy, topical ocular hypotensive agents, anticoagulation, and intravenous mannitol and acetazolamide. An aggressive stepwise approach may be beneficial, depending on the underlying cause of the occlusion and the amount of time from onset of occlusion to treatment. Most visual loss associated with central retinal artery occlusion is severe and permanent (Schmidt-Erfurth et al., 2019).

Age-Related Macular Degeneration

AMD is the leading cause of irreversible blindness and visual impairment in the world (Bright Focus Foundation, 2020a). AMD is characterized by drusen beneath the retina (see Fig. 58-12). Most people older than 60 years have at least a few small drusen, which are clusters of debris or waste material. When drusen are located in the macular area, they can affect vision. Patients with AMD have a wide range of visual loss, but only a small portion experience total blindness. Central vision is generally the most affected, with most patients retaining peripheral vision (see Fig. 58-13). There are two types of AMD: the dry type and the wet type (Bright Focus Foundation, 2020a).



Figure 58-12 • Retina showing drusen and age-related macular degeneration.

Between 85% and 90% of people with AMD have the dry (nonneovascular, nonexudative) type of the condition, in which the outer layers of the retina slowly break down. With this breakdown comes the appearance of drusen. When the drusen occur outside of the macular area, patients generally have no symptoms. When the drusen occur within the macula, however, there is a gradual blurring of vision that patients may notice when they try to read.

The second type of AMD, the wet (neovascular, exudative) type, may have an abrupt onset and is more damaging to the vision (Bright Focus Foundation, 2020a). Patients report that straight lines appear crooked and distorted or that letters in words appear broken. This effect results from proliferation of abnormal blood vessels growing under the retina, within the choroid layer of the eye, a condition known as choroidal neovascularization. The affected vessels can leak fluid and blood, elevating the retina. Some patients can be treated with laser therapy to stop leakage from these vessels.



Figure 58-13 • Visual changes resulting from age-related macular degeneration. Photo courtesy of the National Eye Institute, National Institutes of Health.

Chart 58-11



PATIENT EDUCATION

Education for Patients About Preventing Eye Injuries

The nurse instructs the patient in the following measures to prevent eye injuries:

In and Around the House

- Make sure that all spray nozzles are directed away from yourself before pressing down on the handle.
- Read instructions carefully before using cleaning fluids, detergents, ammonia, or harsh chemicals, and wash hands thoroughly after use.
- Use grease shields on frying pans to decrease spattering.
- Use opaque goggles to avoid burns from sunlamps.
- Wear special goggles to shield the eyes from fumes and splashes when using powerful chemicals.

In the Workshop

- Protect the eyes from flying fragments, fumes, dust particles, sparks, and splashed chemicals by wearing safety glasses.
- Read instructions thoroughly before using tools and chemicals, and follow precautions for their use.

Around Children

- Educate children about the correct way to handle potentially dangerous items, such as scissors and pencils.
- Pay attention to age and maturity level of a child when selecting toys and games, and avoid projectile toys, such as darts and pellet guns.
- Supervise children when they are playing with toys or games that can be dangerous.

In the Garden

- Avoid letting anyone stand at the side of or in front of a moving lawn mower.
- Avoid low-hanging branches.
- Direct pesticide spray can nozzles away from the face.
- Pick up rocks and stones before going over them with the lawn mower (stones can be hurled out of the rotary blades and rebound off curbs or walls, causing severe injury to the eye).

Around the Car

- Put out all smoking materials and matches before opening the hood of the car.
- Take standard safety precautions when using jumper cables (wear goggles; make sure the cars are not touching one another; make sure the jumper

cable clamps never touch each other; never lean over the battery when attaching cables; and never attach a cable to the negative terminal of a dead battery).

- Use a flashlight, not a match or lighter, to look at the battery at night.
- Wear goggles when grinding metal or striking metal against metal while performing auto body repair.

In Sports

- Wear protective caps, helmets, or face protectors when appropriate, especially for sports such as ice hockey.
- Wear protective safety glasses, especially for sports such as racquetball, squash, tennis, baseball, and basketball.

Around Fireworks

- Avoid explosive fireworks.
- Avoid standing near others when lighting fireworks.
- Douse firework duds in water instead of attempting to relight them.
- Never allow children to ignite fireworks.
- Wear eyeglasses or safety goggles.

Medical Management

There is no known effective treatment or cure for dry advanced macular degeneration (Bright Focus Foundation, 2020a).

An important component of treatment of wet (neovascular, exudative) AMD targets development and progression of angiogenesis (abnormal blood vessel formation). Vasoproliferation in wet AMD is believed to be caused by an underlying angiogenic stimulus known as vascular endothelial growth factor (VEGF) (Bright Focus Foundation, 2020a). Examples of VEGF inhibitors given by intravitreal injection include ranibizumab and brolucizumab (Bright Focus Foundation, 2020a; Comerford & Durkin, 2020).

Nursing Management

Amsler grids are given to patients to use in their homes to monitor for a sudden onset or distortion of vision. These may provide the earliest sign that macular degeneration is getting worse. Patients should be encouraged to look at these grids, one eye at a time, several times each week with glasses on if needed for corrected near vision. If there is a change in the way the grid appears to the patient (e.g., if the lines or squares appear distorted or faded), the patient should notify the ophthalmologist immediately and should arrange

to be seen promptly. There are digital versions of these grids as well that pick up abnormal changes quicker.

Orbital and Ocular Trauma

Whether affecting the eye or the orbit, trauma to the eye and surrounding structures may have devastating consequences for vision. It is preferable to prevent injury rather than treat it. [Chart 58-11](#) details measures to prevent eye injuries.

Orbital Trauma

Injury to the orbit is usually associated with a head injury; hence, the patient's general medical condition must first be stabilized before conducting an ocular examination (Hickey & Strayer, 2020). Only then is the globe assessed for soft tissue injury. During inspection, the face is meticulously assessed for underlying fractures, which should always be suspected in cases of blunt trauma. To establish the extent of ocular injury, visual acuity is assessed as soon as possible, even if it is only a rough estimate. Soft tissue orbital injuries often result in damage to the optic nerve. Major ocular injuries indicated by a soft globe, prolapsing tissue, ruptured globe, and hemorrhage require immediate surgical attention.

Soft Tissue Injury and Hemorrhage

The signs and symptoms of soft tissue injury from blunt or penetrating trauma include tenderness, ecchymosis, lid swelling, **exophthalmos** (abnormal protrusion of the eyeball), and hemorrhage. Closed injuries lead to contusions with subconjunctival hemorrhage, commonly known as a black eye. Blood accumulates in the tissues of the conjunctiva. Hemorrhage may be caused by a soft tissue injury to the eyelid or by an underlying fracture.

Management of soft tissue hemorrhage that does not threaten vision is usually conservative and consists of thorough inspection, cleansing, and repair of wounds. Cold compresses are used in the early phase, followed by warm compresses. Hematomas that appear as swollen, fluctuating areas may be surgically drained or aspirated; if they are causing significant orbital pressure, they may be surgically evacuated.

Orbital Fractures

Orbital fractures are detected by facial x-rays. Depending on the orbital structures involved, orbital fractures can be classified as blowout, zygomatic or

tripod, maxillary, midfacial, orbital apex, and orbital roof fractures. Blowout fractures result from compression of soft tissue and the sudden increase in orbital pressure when the force is transmitted to the orbital floor, which is the area of least resistance (McQuillan & Makic, 2020).

The inferior rectus and inferior oblique muscles, with their fat and fascial attachments, or the nerve that courses along the inferior oblique muscle may become entrapped, resulting in enophthalmos (inward displacement of the globe). Computed tomography (CT) scanning can identify the muscle and its auxiliary structures that are entrapped. These fractures are usually caused by the blunt force from a fist or baseball (McQuillan & Makic, 2020).

Orbital roof fractures are dangerous because of potential complications to the brain. Surgical management of these fractures requires a neurosurgeon and an ophthalmologist. The most common indications for surgical intervention are displacement of bone fragments disfiguring the normal facial contours, interference with normal binocular vision caused by extraocular muscle entrapment, interference with mastication in zygomatic fracture, and obstruction of the nasolacrimal duct. Surgery is usually nonemergent, and a period of 10 to 14 days gives the ophthalmologist time to assess ocular function, especially the extraocular muscles and the nasolacrimal duct. Emergency surgical repair is usually not performed unless the globe is displaced into the maxillary sinus. Surgical repair is primarily directed at freeing the entrapped ocular structures and restoring the integrity of the orbital floor.

Foreign Bodies

Foreign bodies that enter the orbit are usually tolerated, except for steel, copper, iron, and vegetable materials such as those from plants or trees, which may cause purulent infection. X-rays and CT scans are used to identify the foreign body. A careful history is important, especially if the foreign body has been in the orbit for a period of time and the incident forgotten. It is important to identify metallic foreign bodies because they prohibit the use of magnetic resonance imaging (MRI) as a diagnostic tool.

After the extent of the orbital damage is assessed, the decision to use conservative treatment or surgical removal is made. In general, orbital foreign bodies are removed if they are superficial and anterior in location; have sharp edges that may affect adjacent orbital structures; or are composed of copper, iron, or vegetable material. Surgical intervention is directed at preventing further ocular injury and maintaining the integrity of the affected areas. Cultures are usually obtained, and the patient is placed on prophylactic IV antibiotic medications that are later changed to an oral route.

Ocular Trauma

Ocular trauma is a leading cause of blindness among children and young adults, especially male trauma victims (Buc'án, Matas, Lovric', et al., 2017). Ocular trauma occurs with occupational injuries (e.g., construction industry), contact sports, weapons (e.g., air guns, BB guns), assaults, motor vehicle crashes (e.g., broken windshields), and explosions (e.g., blast fragments).

There are two types of ocular trauma in which the first response is critical: chemical burn and foreign object in the eye. With a chemical burn, the eye should be immediately irrigated with tap water or normal saline. With a foreign body, no attempt should be made to remove the foreign object. The object should be protected from jarring or movement to prevent further ocular damage. No pressure or patch should be applied to the affected eye. All other traumatic eye injuries should be protected using a patch or shield if available or a stiff paper cup until medical treatment can be obtained (see Fig. 58-14).

Assessment and Diagnostic Findings

A thorough history is obtained, particularly assessing the patient's ocular history, such as pre-injury vision in the affected eye or past ocular surgery. Details related to the injury that help in the diagnosis and assessment of the need for further tests include the nature of the ocular injury (i.e., blunt or penetrating trauma); the type of activity that caused the injury to determine the nature of the force striking the eye; and whether onset of vision loss was sudden, slow, or progressive. For chemical eye burns, the chemical agent must be identified and tested for pH if the agent is available. The corneal surface is examined for foreign bodies, wounds, and abrasions, after which the other external structures of the eye are examined. Pupil size, shape, and light reaction of the pupil of the affected eye are compared with the other eye. Ocular motility (ability of the eyes to move synchronously up, down, right, and left) is also assessed.

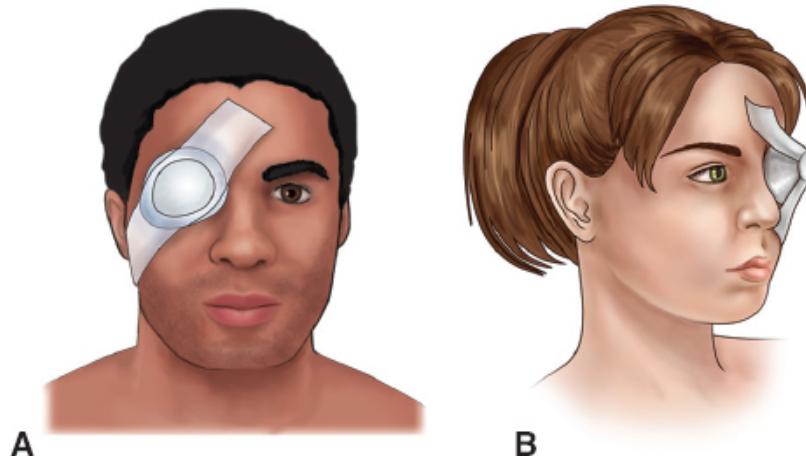


Figure 58-14 • Two kinds of eye patches. **A.** Aluminum shield. **B.** Stiff paper cup shield (innovative substitute when aluminum shield is unavailable).

Medical Management

Splash Injuries

Splash injuries are irrigated with normal saline solution before further evaluation occurs. In cases of a ruptured globe, cycloplegic agents (agents that paralyze the ciliary muscle) or topical antibiotics must be deferred because of potential toxicity to exposed intraocular tissues. Further manipulation of the eye must be avoided until the patient is under general anesthesia. Parenteral, broad-spectrum antibiotics are initiated. Tetanus antitoxin is given, if indicated, as well as analgesic agents. (Tetanus prophylaxis is recommended for full-thickness ocular and skin wounds.) Any topical ophthalmic medication (e.g., anesthetic, dyes) must be sterile.

Foreign Bodies and Corneal Abrasions

After removal of a foreign body from the surface of the eye, an antibiotic ointment is applied and the eye is patched. The eye is examined daily for evidence of infection until the wound is completely healed.

Contact lens wear is a common cause of corneal abrasion. The patient experiences severe pain and **photophobia** (ocular pain on exposure to light). Corneal epithelial defects are treated with antibiotic ointment and, in some instances, a pressure patch to immobilize the eyelids. Topical anesthetic eye drops must not be given to the patient to take home for repeated use after corneal injury because their effects mask further damage, delay healing, and can lead to corneal scarring.

Penetrating Injuries and Contusions of the Eyeball

Sharp penetrating injury or blunt contusion force can rupture the eyeball. When the globe, cornea, and sclera rupture, rapid decompression or herniation of the orbital contents into adjacent sinuses can occur. Blunt traumatic injuries (with an increased incidence of retinal detachment, intraocular tissue avulsion, and herniation) have a worse prognosis than penetrating injuries. Most penetrating injuries result in marked loss of vision with the following signs: hemorrhagic **chemosis** (edema of the conjunctiva), conjunctival laceration, shallow anterior chamber with or without an eccentrically placed pupil, **hyphema** (blood within the anterior chamber), or vitreous hemorrhage.

Hyphema is caused by contusion forces that tear the vessels of the iris and damage the anterior chamber angle. Preventing rebleeding and prolonged increased IOP are the goals of treatment for hyphema. In severe cases, the patient is hospitalized with moderate activity restriction. An eye shield is applied. Topical corticosteroids are prescribed to reduce inflammation. An antifibrinolytic agent (aminocaproic acid) stabilizes clot formation at the site of hemorrhage. Aspirin is contraindicated. A ruptured globe and severe injuries with intraocular hemorrhage require surgical intervention. Vitrectomy is performed for traumatic retinal detachments (Park et al., 2018). Primary **enucleation** (complete removal of the eyeball and part of the optic nerve) is considered only if the globe is irreparable and has no light perception. It is a general rule that enucleation is performed within 2 weeks of the initial injury (in an eye that has no useful vision after sustaining penetrating injury) to prevent the risk of **sympathetic ophthalmia** (an inflammation created in the uninjured eye by the affected eye that can result in blindness of the uninjured eye).

Intraocular Foreign Bodies

A patient who complains of blurred vision and discomfort should be questioned carefully about recent injuries and exposures. Patients may be injured in a number of different situations and experience an intraocular foreign body (IOFB). Precipitating circumstances can include working in construction, striking metal against metal, being involved in a motor vehicle crash with facial injury, a gunshot wound, grinding-wheel work, and explosions.

IOFB is diagnosed and localized by slit-lamp biomicroscopy and indirect ophthalmoscopy, as well as CT or ultrasonography scanning. MRI is contraindicated because most foreign bodies are metallic and magnetic. It is important to determine the composition, size, and location of the IOFB and affected eye structures. Every effort should be made to identify the type of IOFB and whether it is magnetic. Iron, steel, copper, and vegetable matter may cause intense inflammatory reactions. The incidence of endophthalmitis is high. Surgical excision of the foreign body depends on its location and composition and associated ocular injuries. Specially designed IOFB forceps

and magnets are used to grasp and remove the foreign body. Any damaged area of the retina is treated to prevent retinal detachment.

Ocular Burns

Alkali, acid, and other chemically active organic substances, such as Mace and tear gas, cause chemical burns. Alkali burns (e.g., lye, ammonia) result in the most severe injury because they penetrate the ocular tissues rapidly and continue to cause long-term damage. Increased IOP also occurs. Acids (e.g., bleach, car batteries, refrigerant) generally cause less damage because the precipitated necrotic tissue proteins form a barrier to further penetration and damage. Chemical burns may appear as superficial punctate keratopathy (i.e., spotty damage to the cornea), subconjunctival hemorrhage, or complete marbleizing of the cornea.

In treating chemical burns, every minute counts. Immediate irrigation with tap water should be started on site before transport of the patient to an emergency department. A brief history and examination are performed. Critical information, if available, is the name of the substance that went into the eye (the actual container is best). Material safety data sheets (MSDSs) should be accessed for reference (see [Chapter 68](#)). The corneal surfaces and conjunctival fornices are immediately and copiously irrigated with normal saline or any neutral solution. A local anesthetic is instilled, and a lid speculum is applied to overcome blepharospasm (i.e., spasms of the eyelid muscles that result in closure of the lids). Particulate matter must be removed from the fornices using moistened, cotton-tipped applicators and minimal pressure on the globe. Irrigation continues until the conjunctival pH normalizes (between 7.3 and 7.6). The pH of the corneal surface is checked by placing a pH paper strip in the fornix. Antibiotic agents are instilled, and the eye usually is patched.

The goal of intermediate treatment is to prevent tissue ulceration and promote re-epithelialization of the cornea. Intense lubrication using artificial tears without preservatives (to avoid allergic reactions) is essential. Patching or therapeutic soft lenses may also be used to promote corneal healing, and the patient is closely monitored. Prognosis depends on the type of injury and adequacy of the irrigation immediately after exposure. Long-term treatment consists of two phases: restoration of the ocular surface through grafting procedures and surgical restoration of corneal integrity and optical clarity.

Thermal injury is caused by exposure to a hot object (e.g., curling iron, tobacco, ash), whereas photochemical injury results from ultraviolet irradiation or infrared exposure (e.g., exposure to the reflections from snow, sun gazing, viewing an eclipse of the sun without an adequate filter). These injuries can cause corneal epithelial defect, corneal opacity, conjunctival chemosis and **injection** (congestion of blood vessels), and burns of the eyelids and periocular

region. Antibiotic agents and a patch for 24 hours constitute the treatment of mild injuries.

INFECTIOUS AND INFLAMMATORY CONDITIONS

Inflammation and infections of eye structures are common. [Table 58-7](#) summarizes select infections and their treatment.

Dry Eyes

Dry eyes can be caused by decreased tear production or increased tear evaporation, which can be episodic or chronic (Norris, 2019). Decreased tear production (aqueous deficiency) can be caused by systemic disease (Sjögren, connective tissue disease), lacrimal gland obstruction, and systemic drugs (e.g., diuretics, antihistamines, psychotropic drugs). Increased tear evaporation (evaporative dry eye) can be caused by meibomian gland deficiency, lid aperture disorder, vitamin A deficiency, reduced lid blinking rate, preservatives from topical drugs, ocular surface disease (allergy), and contact lens wear. Risk factors include increasing age, smoking, recent refractive surgery, and postmenopausal status (in women). An increase in the intake of omega-3 fatty acids may be beneficial in reducing the risk (Norris, 2019).

Clinical Manifestations

The most common complaints are photophobia, foreign-body sensation, burning and stinging, redness, and decreased tearing.

Assessment and Diagnostic Findings

Chronic dry eyes may result in chronic conjunctival and corneal irritation that can lead to corneal erosion, scarring, ulceration, thinning, or perforation that can seriously threaten vision. Secondary bacterial infection can occur.

Management

Management of dry eyes requires the cooperation of the patient with a regimen that needs to be followed at home for a long period; otherwise, complete relief of symptoms is unlikely. Instillation of artificial tears during the day and an ointment at night is the usual regimen to hydrate and lubricate the eye and preserve a moist ocular surface. Cyclosporine ophthalmic emulsion is an effective agent that increases tear production and is used once daily. Anti-

inflammatory medications are also used, and moisture chambers (e.g., moisture chamber spectacles, swim goggles) may provide additional relief.

Patients may become hypersensitive to chemical preservatives such as benzalkonium chloride and thimerosal. For these patients, preservative-free ophthalmic solutions are used. Management of dry eyes also includes the concurrent treatment of infections, such as chronic blepharitis and acne rosacea, and treating the underlying systemic disease, such as Sjögren syndrome (an autoimmune disease).

TABLE 58-7 Select Infections and Inflammatory Disorders of Eye Structures

Disorder	Description	Management
Hordeolum (stye)	Acute suppurative infection of the glands of the eyelids caused by <i>Staphylococcus aureus</i> . The lid is red and edematous with a small collection of pus in the form of an abscess. There is considerable discomfort.	Warm compresses are applied directly to the affected lid area three to four times a day for 10–15 min. If the condition is not improved after 48 h, incision and drainage may be indicated. Application of topical antibiotics may be prescribed thereafter.
Chalazion	Sterile inflammatory process involving chronic granulomatous inflammation of the meibomian glands; can appear as a single granuloma or multiple granulomas in the upper or lower eyelids.	Warm compresses applied three to four times a day for 10–15 min may resolve the inflammation in the early stages. Most often, however, surgical excision is indicated. Corticosteroid injection to the chalazion lesion may be used for smaller lesions.
Blepharitis	Chronic bilateral inflammation of the eyelid margins. There are two types: staphylococcal and seborrheic. Staphylococcal blepharitis is usually ulcerative and is more serious due to the involvement of the base of hair follicles. Permanent scarring can result.	The seborrheic type is chronic and is usually resistant to treatment, but the milder cases may respond to lid hygiene. Staphylococcal blepharitis requires topical antibiotic treatment. Instructions on lid hygiene (to keep the lid margins clean and free of exudates) are given to the patient.
Bacterial keratitis	Infection of the cornea by <i>S. aureus</i> , <i>Streptococcus pneumoniae</i> , and <i>Pseudomonas aeruginosa</i> .	Fortified (high-concentration) antibiotic eye drops are given every 30 min around the clock for the first few days, then every 1–2 h. Systemic antibiotics may be given. Cycloplegics are given to reduce pain caused by ciliary spasm.
Herpes simplex keratitis	Symptoms are severe pain, tearing, and photophobia. The dendritic ulcer has a branching, linear pattern with feathery edges and terminal bulbs at its ends. Herpes simplex keratitis can lead to recurrent stromal keratitis and persist to 12 mo with residual corneal scarring.	Many lesions heal without treatment and residual effects. The treatment goal is to minimize the damaging effect of the inflammatory response and eliminate viral replication within the cornea. Penetrating keratoplasty is indicated for corneal scarring and must be performed when the herpetic disease has been inactive for many months.

Adapted from Shaw, M., & Lee, A. (2017). *Ophthalmic nursing* (5th ed.). Boca Raton, FL: CRC Press Taylor & Francis Group.

Surgical treatment includes punctal occlusion, grafting procedures, and lateral tarsorrhaphy (uniting the edges of the lids). Punctal plugs are made of silicone material for the temporary or permanent occlusion of the puncta. These help to preserve the volume of natural tears and prolong the effects of artificial tears (Norris, 2019).

Conjunctivitis

Conjunctivitis (inflammation of the conjunctiva) is a common ocular disorder worldwide. It is characterized by a pink appearance (hence the common term *pink eye*) because of subconjunctival blood vessel congestion.

Clinical Manifestations

General symptoms include foreign-body sensation, scratching or burning sensation, itching, and photophobia. Conjunctivitis may be unilateral or bilateral, but the infection usually starts in one eye and then spreads to the other eye by hand contact.

Assessment and Diagnostic Findings

The four main clinical features important to evaluate are the type of discharge (watery, mucoid, purulent, or mucopurulent), type of conjunctival reaction (follicular or papillary), presence of pseudomembranes or true membranes, and presence or absence of lymphadenopathy (enlargement of the preauricular and submandibular lymph nodes where the eyelids drain). Pseudomembranes consist of coagulated exudate that adheres to the surface of the inflamed conjunctiva. True membranes form when the exudate adheres to the superficial layer of the conjunctiva, and removal results in bleeding. Follicles are multiple, slightly elevated lesions encircled by tiny blood vessels; they look like grains of rice. Papillae are hyperplastic conjunctival epithelium in numerous projections that are usually seen as a fine mosaic pattern under slit-lamp examination. Diagnosis is based on the distinctive characteristics of ocular signs, acute or chronic presentation, and identification of any precipitating events. Positive results of swab smear preparations and cultures confirm the diagnosis.

Types of Conjunctivitis

Conjunctivitis is classified according to its cause. The major causes are microbial infection, allergy, and irritating toxic stimuli. A wide spectrum of organisms can cause conjunctivitis, including bacteria (e.g., *Chlamydia*),

viruses, fungus, and parasites. Conjunctivitis can also result from an existing ocular infection or can be a manifestation of a systemic disease.

Microbial Conjunctivitis

Bacterial Conjunctivitis

Bacterial conjunctivitis can be acute or chronic. The acute type can develop into a chronic condition. Signs and symptoms can vary from mild to severe. Chronic bacterial conjunctivitis is usually seen in patients with lacrimal duct obstruction, chronic dacryocystitis, and chronic blepharitis. The most common causative microorganisms are *Streptococcus pneumoniae*, *Haemophilus influenzae*, and *Staphylococcus aureus*.

Bacterial conjunctivitis manifests with an acute onset of redness, burning, and discharge. There is papillary formation, conjunctival irritation, and injection in the fornices. The exudates are variable but are usually present on waking in the morning. The eyes may be difficult to open because of adhesions caused by the exudate. Purulent discharge occurs in severe acute bacterial infections, whereas mucopurulent discharge appears in mild cases. In gonococcal conjunctivitis, the symptoms are more acute. The exudate is profuse and purulent, and there is lymphadenopathy. Pseudomembranes may be present.

Trachoma is an infectious disease caused by the bacterium *Chlamydia trachomatis*, an ancient disease and the leading cause of preventable blindness in the world (Norris, 2019). It is prevalent in areas with hot, dry, and dusty climates and in areas with poor living conditions. It is spread by direct contact or by carrier (e.g., insects such as flies and gnats). The onset of trachoma in children is usually insidious, but it can be acute or subacute in adults. The initial symptoms include red inflamed eyes, tearing, photophobia, ocular pain, purulent exudates, preauricular lymphadenopathy, and lid edema. Initial ocular signs include follicular and papillary formations. At the middle stage of the disease, there is an acute inflammation with papillary hypertrophy and follicular necrosis, after which trichiasis and entropion begin to develop. The lashes that are turned in rub against the cornea and, after prolonged irritation, cause corneal erosion and ulceration. The late stage of the disease is characterized by scarred conjunctiva, subepithelial keratitis, abnormal vascularization of the cornea (pannus), and residual scars from the follicles that look like depressions in the conjunctiva (Herbert pits). Severe corneal ulceration can lead to perforation and blindness.

Inclusion conjunctivitis affects sexually active people who have genital chlamydial infection. Transmission is by oral-genital sex or hand-to-eye transmission. Indirect transmission can occur in inadequately chlorinated swimming pools. The eye lesions usually appear a week after exposure and

may be associated with a nonspecific urethritis or cervicitis. The discharge is mucopurulent, follicles are present, and there is lymphadenopathy.

Viral Conjunctivitis

Viral conjunctivitis can be acute and chronic. The discharge is watery, and follicles are prominent. Severe cases include pseudomembranes. The common causative organisms are adenovirus and herpes simplex virus. Conjunctivitis caused by adenovirus is highly contagious. The condition is usually preceded by symptoms of upper respiratory infection. Corneal involvement causes extreme photophobia. Symptoms include tearing, redness, and foreign-body sensation that can involve one or both eyes. There is lid edema, ptosis, and conjunctival **hyperemia** (red eyes caused by dilation of blood vessels) (see Fig. 58-15). These signs and symptoms vary from mild to severe. Viral conjunctivitis, although self-limited, tends to last longer than bacterial conjunctivitis.

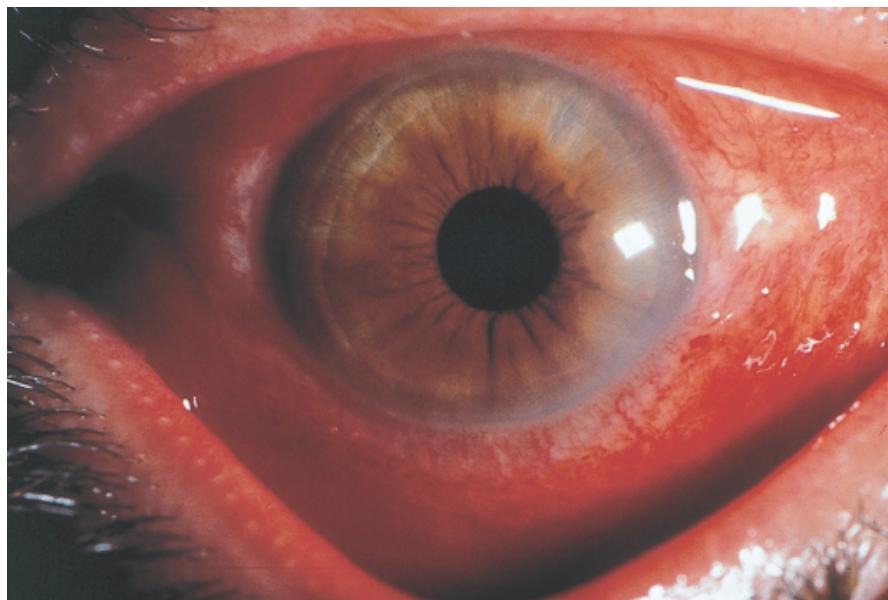


Figure 58-15 • Conjunctival hyperemia in viral conjunctivitis.

Epidemic keratoconjunctivitis is a highly contagious viral conjunctivitis that is easily transmitted from one person to another among household members, schoolchildren, and health care workers. The outbreak of epidemics is seasonal, especially during the summer when people use swimming pools. Epidemic keratoconjunctivitis is most often accompanied by preauricular lymphadenopathy and occasionally periorbital pain. There are marked follicular and papillary formations. This type of conjunctivitis can lead to keratopathy.

Allergic Conjunctivitis

Immunologic or allergic conjunctivitis is a hypersensitivity reaction that occurs as part of allergic rhinitis (hay fever), or it can be an independent allergic reaction. The patient usually has a history of an allergy to pollens and other environmental allergens. There is extreme pruritus, epiphora (excessive secretion of tears), injection, and usually severe photophobia. The stringlike mucoid discharge is usually associated with rubbing the eyes because of severe pruritus. Vernal conjunctivitis is also known as seasonal conjunctivitis because it appears mostly during warm weather. There may be large formations of papillae that have a cobblestone appearance. It is more common in children and young adults. Most affected people have a history of asthma or eczema.

Toxic Conjunctivitis

Chemical conjunctivitis can be the result of medications; chlorine from swimming pools; exposure to toxic fumes among industrial workers; or exposure to other irritants such as smoke, hair sprays, acids, and alkalis.

Medical Management

The management of conjunctivitis depends on the type. Most types of mild and viral conjunctivitis are self-limiting, benign conditions that may not require treatment and laboratory procedures. For more severe cases, topical antibiotic agents, eye drops, or ointments are prescribed. Patients with gonococcal conjunctivitis require urgent antibiotic therapy. If left untreated, this ocular disease can lead to corneal perforation and blindness. The systemic complications can include meningitis and sepsis.

Bacterial Conjunctivitis

Acute bacterial conjunctivitis is almost always self-limiting, lasting 2 weeks if left untreated. If treated with antibiotics, it may last a few days, except for gonococcal and staphylococcal conjunctivitis.

For trachoma, usually broad-spectrum antibiotic agents are given topically and systemically. Surgical management includes the correction of trichiasis to prevent conjunctival scarring.

Adult inclusion conjunctivitis requires 1 week of antibiotics. Prevention of reinfection is important, and affected people and their sexual partners must seek treatment for sexually transmitted infection, if indicated.

Viral Conjunctivitis

Viral conjunctivitis is not responsive to any treatment. Cold compresses may alleviate some symptoms. Viral conjunctivitis, especially epidemic keratoconjunctivitis, is highly contagious. Patients must be made aware of the

contagious nature of the disease, and adequate education must be provided (see [Chart 58-12](#)).

Proper steps must be taken to avoid health care-associated infections. Frequent hand hygiene and procedures for environmental cleaning and disinfection of equipment used for eye examination must be strictly followed at all times. To prevent spread during outbreaks of conjunctivitis caused by adenovirus, health care facilities must set aside specified areas for treating patients diagnosed with or suspected of having conjunctivitis caused by adenovirus. All forms of tonometry must be avoided unless medically indicated. All multidose ophthalmic medications must be discarded at the end of each day or when contaminated. Employees who are infected and others must not be allowed to work or attend school until symptoms have resolved, which can take 3 to 7 days.

Chart 58-12



PATIENT EDUCATION

Education for Patients with Viral Conjunctivitis

Viral conjunctivitis is a highly contagious eye infection that can easily spread from one person to another. The symptoms can be alarming but are not serious.

The nurse instructs the patient about this eye condition and the following self-care strategies:

- Be aware that your eyes will look red and will have watery discharge, and your lids will be swollen for about a week.
- Expect to experience eye pain, a sandy sensation in your eye, and sensitivity to light.
- Keep in mind that symptoms will resolve after about 1 week.
- Use lightweight cold compresses over your eyes for about 10 minutes four to five times a day to soothe the pain.
- Use artificial tears for the sandy sensation in your eye and mild pain medications such as acetaminophen.
- Stay at home and do not go outside. You may return to work or school after 7 days, when the redness and discharge have cleared. Obtain a note from your primary provider to return to work or school.
- Do not share towels, linens, makeup, or any items that have come in contact with your eyes.
- Wash your hands thoroughly with soap and water frequently.
- Use a new tissue every time you wipe the discharge from your eye. Dampen the tissue with clean water to clean the outside of the eye.
- Wash your face and take a shower as you normally do.
- Discard all of your makeup articles and do not apply makeup until the infection has resolved.
- Wear dark glasses if bright lights bother you.
- Note if the discharge from your eye turns yellowish and purulent or if your vision changes and return to the primary provider for an examination.

Allergic Conjunctivitis

Patients with allergic conjunctivitis, especially recurrent vernal or seasonal conjunctivitis, are usually given corticosteroids in ophthalmic preparations. Depending on the severity of the disease, they may be given oral preparations. The use of vasoconstrictors, such as topical epinephrine solution, cold compresses, ice packs, and cool ventilation usually provide comfort by decreasing swelling.

Toxic Conjunctivitis

For conjunctivitis caused by chemical irritants, the eye must be irrigated immediately and profusely with saline or sterile water.

Uveitis

Uveitis, or inflammation of the uveal tract, can affect the iris, the ciliary body, or the choroid. There are two types of uveitis: nongranulomatous and granulomatous.

The more common form of uveitis is the nongranulomatous type, which manifests as an acute condition with pain, photophobia, and a pattern of conjunctival injection, especially around the cornea. The pupil is small or irregular, and vision is blurred. There may be small, fine precipitates on the posterior corneal surface and cells in the aqueous humor (i.e., cell and flare). If the uveitis is severe, a **hypopyon** (accumulation of inflammatory cells in the anterior chamber of the eye) may develop. The condition may be unilateral or bilateral and may be recurrent. Repeated attacks of nongranulomatous anterior uveitis can cause anterior synechiae (peripheral iris adheres to the cornea and impedes outflow of aqueous humor). Posterior synechiae (adherence of the iris and lens) block aqueous outflow from the posterior chamber. Secondary glaucoma can result from either anterior or posterior synechiae. Cataracts may also occur as a sequela to uveitis.

Granulomatous uveitis can have a more insidious onset and can involve any portion of the uveal tract. It tends to be chronic. Symptoms such as photophobia and pain may be minimal. Vision is markedly and adversely affected. Conjunctival injection is diffuse, and there may be vitreous clouding. In a severe posterior uveitis, such as chorioretinitis, there may be retinal and choroidal hemorrhages.

Medical Management

Because photophobia is a common symptom, patients should wear dark glasses outdoors. Ciliary spasm and synechia are best avoided through mydriasis; cyclopentolate and atropine are commonly used. Local corticosteroid drops instilled four to six times a day are also used to decrease inflammation.

If the uveitis is recurrent, a careful history should be initiated to discover any underlying causes. This evaluation should include a complete history, physical examination, and diagnostic tests, including a complete blood count, erythrocyte sedimentation rate, antinuclear antibodies, and Venereal Disease Research Laboratory (VDRL) and Lyme disease titers. Underlying causes include autoimmune disorders such as ankylosing spondylitis and sarcoidosis.

as well as toxoplasmosis, herpes zoster virus, ocular candidiasis, histoplasmosis, herpes simplex virus, tuberculosis, and syphilis.

Orbital Cellulitis

Orbital cellulitis is inflammation of the tissues surrounding the eye that may result from bacterial, fungal, or viral inflammatory conditions of contiguous structures, such as the face, oropharynx, dental structures, or intracranial structures. It can also result from foreign bodies and preexisting ocular infection, such as dacryocystitis and panophthalmitis, or from sepsis. Infection of the sinuses is the most frequent cause. Infection originating in the sinuses can spread easily to the orbit through the thin bony walls and foramina or by means of the interconnecting venous system of the orbit and sinuses. The most common causative organisms are staphylococci and streptococci in adults. The symptoms include pain, eyelid swelling, conjunctival edema, proptosis, and decreased ocular motility. With such edema, optic nerve compression can occur and IOP may increase.

The severe intraorbital tension caused by abscess formation and the impairment of optic nerve function in orbital cellulitis can result in permanent visual loss. Because of the orbit's proximity to the brain, orbital cellulitis can lead to life-threatening complications, such as intracranial abscess and cavernous sinus thrombosis.

Medical Management

Immediate administration of high-dose, broad-spectrum, systemic antibiotics is indicated. Cultures and Gram-stained smears are obtained. Monitoring changes in visual acuity, degree of proptosis, central nervous system function (e.g., nausea, vomiting, fever, cognitive changes), displacement of the globe, extraocular movements, pupillary signs, and the fundus is extremely important. Consultation with an otolaryngologist is necessary, especially when rhinosinusitis is suspected. In the event of abscess formation or progressive loss of vision, surgical drainage of the abscess or sinus is performed. Sinusotomy and antibiotic irrigation are also performed.

ORBITAL AND OCULAR TUMORS

Benign Tumors of the Orbit

Benign tumors can develop from infancy and grow rapidly or slowly and present in later life. Some benign tumors are superficial and are easily identifiable by external presentation, palpation, and x-rays, but some are deep and may require a CT scan to diagnose. There can be significant proptosis, and visual function may be jeopardized. Benign tumors are masses characterized by the lack of infiltration in the surrounding tissues. Examples are cystic dermoid cysts and mucocele, hemangiomas, lymphangiomas, lacrimal tumors, and neurofibromas.

To prevent recurrence, benign masses are excised completely when possible. Excision may be difficult because of the involvement of some portions of the orbital bones, such as deep dermoid cysts, in which dissection of the bone is required. Subtotal resection may be indicated in deep benign tumors that intertwine with other orbital structures, such as optic nerve meningiomas. Complete removal of the tumor may endanger visual function.

Benign Tumors of the Eyelids

There are a wide variety of benign tumors that increase in frequency with age. Nevi may be unpigmented at birth and may enlarge and darken in adolescence or may never acquire any pigment at all. Hemangiomas are vascular capillary tumors that may be bright, superficial, red lesions (formerly known as strawberry angiomas) or bluish and purplish deeper lesions. Milia are small, white, slightly elevated cysts of the eyelid that may occur in multiples. Xanthelasma are yellowish, lipoid deposits on both lids that commonly appear as a result of the aging of the skin or a lipid disorder. Molluscum contagiosum lesions are flat, symmetric growths along the lid margin caused by a virus that can result in conjunctivitis and keratitis if the lesion grows into the conjunctival sac.

Treatment of benign congenital lid lesions is rarely indicated except when visual function is affected. Corticosteroid injection to the hemangioma lesion is usually effective, but surgical excision may be performed. Benign lid lesions usually present aesthetic problems rather than visual function problems. Surgical excision, or electrocautery, is primarily performed for cosmetic reasons, except for cases of molluscum contagiosum, for which surgical intervention is performed to prevent an infectious process that may ensue.

Benign Tumors of the Conjunctiva

Conjunctival nevus, a congenital benign neoplasm, is a flat, slightly elevated, brown spot that becomes pigmented during late childhood or adolescence. This should be differentiated from the pigmented lesion melanosis, which is

acquired at middle age and may become melanoma. Keratin- and sebum-containing dermoid cysts are congenital and can be found in the conjunctiva. Dermolipoma is a congenital tumor that manifests as a smooth, rounded growth in the conjunctiva near the lateral canthus. Papillomas are usually soft with irregular surfaces and appear on the lid margins. Treatment consists of surgical excision.

Malignant Tumors of the Orbit

Rhabdomyosarcoma is the most common malignant primary orbital tumor in childhood; it can also develop in older adults (Tang, Zhang, Lu, et al., 2018). The symptoms of rhabdomyosarcoma include sudden painless proptosis of one eye followed by lid swelling, conjunctival chemosis, and impairment of ocular motility. CT or MRI scans of these tumors establish the size, configuration, location, and stage of the disease; delineates the degree of bone destruction; and is useful in estimating the field for radiation therapy. The most common site of metastasis is the lung.

Management of these primary malignant orbital tumors involves three major therapeutic modalities: surgery, radiation therapy, and adjuvant chemotherapy. The degree of orbital destruction is important in planning the surgical approach. Resection often involves removal of the eyeball. The psychological needs of the patient and family are paramount in planning the management approach.

Malignant Tumors of the Eyelid

Basal cell carcinoma is the most common malignant tumor of the eyelid. Squamous cell carcinoma occurs less frequently but is considered the second most common malignant tumor. Melanoma is rare. Malignant eyelid tumors occur more frequently among people with a fair complexion who have a history of chronic exposure to the sun (Norris, 2019).

Basal cell carcinoma appears as a painless nodule that may ulcerate. The lesion is invasive, spreads to the surrounding tissues, and grows slowly but does not metastasize. It usually appears on the lower lid margin near the inner canthus with a pearly white margin. Squamous cell carcinoma of the eyelids may resemble basal cell carcinoma initially because it also grows slowly and painlessly. It tends to ulcerate and invade the surrounding tissues, but it can metastasize to the regional lymph nodes. Melanoma may not be pigmented and can arise from nevi. It spreads to the surrounding tissues and metastasizes to other organs.

Complete excision of these carcinomas is followed by reconstruction with skin grafting if the surgical excision is extensive. The ocular postoperative site and the graft donor site are monitored for bleeding. Donor graft sites may include the buccal mucosa, the thigh, or the abdomen. The patient is referred to an oncologist for evaluation of the need for radiation therapy and monitoring for metastasis. Early diagnosis and surgical management are the basis of a good prognosis. These conditions have life-threatening consequences, and surgical excisions may result in facial disfigurement. Emotional support is an extremely important aspect of nursing management.

Malignant Tumors of the Conjunctiva

Conjunctival carcinoma most often grows in the exposed areas of the conjunctiva. The typical lesions are usually gelatinous and whitish due to keratin formation. They grow gradually, and deep invasion and metastasis are rare. Melanoma is rare but may arise from a preexisting nevus or acquired melanosis during middle age. Squamous cell carcinoma is also rare but invasive.

The management is surgical incision. Some benign tumors and most malignant tumors recur. To avoid recurrences, patients usually undergo radiation therapy and cryotherapy after the excision of malignant tumors. Cosmetic disfigurement may result from extensive excision when deep invasion by the malignant tumor is involved.

Malignant Tumors of the Globe

Ocular melanoma is a rare malignant choroidal tumor sometimes discovered on a retinal examination. In its early stages, it could be mistaken for a nevus. In addition to a complete physical examination to discover any evidence of metastasis (to the liver, lung, and breast), retinal fundus photography, fluorescein angiography, and ultrasonography are performed. The diagnosis is confirmed at biopsy after enucleation.

Tumors are classified according to boundary lines (apical height and basal diameter) as small, medium, or large. Small tumors are generally monitored, whereas medium and large tumors require treatment. Treatment consists of radiation, enucleation, or both. Radiation therapy may be achieved by external beam performed in repeated episodes over several days or through the implantation of a small plaque that contains radioactive iodine (I-125) pellets over the tumor.

SURGICAL PROCEDURES AND ENUCLEATION

Orbital Surgeries

Orbital surgeries may be performed to repair fractures, remove a foreign body, or remove benign or malignant growths. Surgical procedures involving the orbit and lids affect facial appearance, or cosmesis. The goals are to recover and preserve visual function and to maintain the anatomic relationship of the ocular structures to achieve cosmesis. During the repair of orbital fractures, the orbital bones are realigned to follow the anatomic positions of facial structures.

Orbital surgical procedures involve working around delicate structures of the eye, such as the optic nerve, retinal blood vessels, and ocular muscles. Complications of orbital surgical procedures may include blindness as a result of damage to the optic nerve and its blood supply. Sudden pain and loss of vision may indicate intraorbital hemorrhage or compression of the optic nerve. Ptosis and diplopia may result from trauma to the extraocular muscles during the surgical procedure, but these conditions typically resolve after a few weeks.

Prophylaxis with IV antibiotic agents is the usual postoperative regimen after orbital surgery, especially with repair of orbital fractures and intraorbital foreign-body removal. IV corticosteroids may be used if there is concern about optic nerve swelling. Topical ocular antibiotics are typically instilled, and antibiotic ointments are applied externally to the skin suture sites.

For the first 24 to 48 hours postoperatively, ice compresses are applied over the periocular area to decrease periorbital swelling, facial swelling, and hematoma. The head of the patient's bed should be elevated to a comfortable position (30 to 45 degrees).

Discharge education should include information about oral antibiotic agents, instillation of ophthalmic medications, and application of ice compresses.

Enucleation

Enucleation is removal of the eyeball (globe) from the orbit, leaving the muscles and orbital contents intact. It may be surgically performed for the following conditions:

- Injury resulting in prolapse of uveal tissue or loss of light perception
- A blind, painful, deformed, or disfigured eye, usually caused by glaucoma, retinal detachment, or chronic inflammation
- An eye without useful vision that is producing or has produced sympathetic ophthalmia in the other eye

- Intraocular tumors that are untreatable by other means

The procedure for enucleation involves the separation and cutting of each of the ocular muscles and surrounding soft tissue and cutting of the optic nerve from the eyeball. The insertion of an orbital implant typically follows, and the conjunctiva is closed. A large pressure dressing is applied over the area.

Evisceration involves the removal of the intraocular contents through an incision or opening in the cornea or sclera. Evisceration may be surgically performed to treat severe ocular trauma with ruptured globe, severe ocular inflammation, or severe ocular infection. The optic nerve, sclera, extraocular muscles, and sometimes the cornea are left intact. The main advantage of evisceration over enucleation is that the final cosmetic result and motility after fitting the ocular prosthesis are enhanced.

Exenteration is the surgical removal of the entire contents of the orbit, surrounding soft tissue, and most or all of the eyelids. This surgery is indicated in malignancies of the orbit that are life-threatening or when more conservative modalities of treatment have failed or are inappropriate. An example is squamous cell carcinoma of the paranasal sinuses, skin, and conjunctiva with deep orbital involvement. In its most extensive form, exenteration may include the removal of all orbital tissues and resection of the orbital bones.

Ocular Prostheses

Orbital implants and conformers (ocular prostheses usually made of silicone rubber) maintain the shape of the eye after enucleation or evisceration to prevent a contracted, sunken appearance. The temporary conformer is placed over the conjunctival closure after the implantation of an orbital implant. A conformer is placed after the enucleation or evisceration procedure to protect the suture line, maintain the fornices, prevent contracture of the socket in preparation for the ocular prosthesis, and promote the integrity of the eyelids.

All ocular prosthetics have limitations in their motility. There are two designs of eye prostheses. The anophthalmic ocular prostheses are used in the absence of the globe. Scleral shells look just like the anophthalmic prosthesis (see Fig. 58-16) but are thinner and fit over a globe with intact corneal sensation. An eye prosthesis usually lasts about 6 years, depending on the quality of fit, comfort, and cosmetic appearance. When the anophthalmic socket is completely healed, conformers are replaced with prosthetic eyes.

An ocularist is a specially trained and skilled professional who makes prosthetic eyes. After the ophthalmologist is satisfied that the anophthalmic socket is completely healed and is ready for prosthetic fitting, the patient is referred to an ocularist. The healing period is usually 6 to 8 weeks. It is advisable for the patient to have a consultation with the ocularist before the

fitting. Obtaining accurate information and verbalizing concerns can lessen anxiety about wearing an ocular prosthesis.

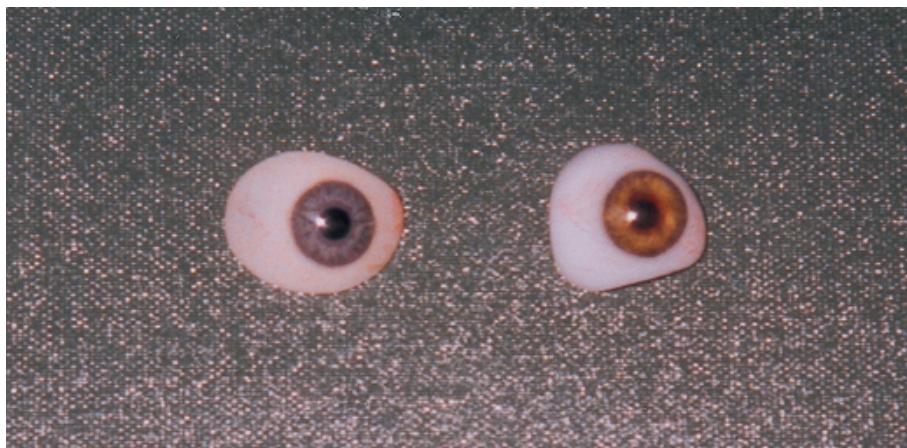


Figure 58-16 • Eye prostheses. (**Left**) Anophthalmic ocular prosthesis. (**Right**) Scleral shell.

Medical Management

Removal of an eye has physical, social, and psychological ramifications for any person. The significance of loss of the eye and vision must be addressed in the plan of care. The patient's preparation should include information about the surgical procedure and placement of orbital implants and conformers and the availability of ocular prosthetics to enhance cosmetic appearance. In some cases, patients may choose to see an ocularist before the surgery to discuss ocular prosthetics.

Nursing Management

Providing Education About Postsurgical and Prosthetic Care

Patients who undergo eye removal need to know that they will usually have a large ocular pressure dressing, which is typically removed after a week, and that an ophthalmic topical antibiotic ointment is applied in the socket three times daily.

After the removal of an eye, there is a loss of depth perception. Patients must be advised to take extra caution in their ambulation and movement to avoid miscalculations that may result in injury. It may take some time to adjust to monocular vision.

The patient must be advised that conformers may inadvertently fall out of the socket. If this happens, the conformer must be washed, wiped dry, and placed back in the socket.

When surgical eye removal is unexpected, such as in severe ocular trauma, leaving no time for the patient and family to prepare for the loss, the nurse's role in providing emotional support is crucial.

Promoting Home, Community-Based, and Transitional Care

The patient with a new prosthetic eye may need referral to home, community-based or transitional services that provide assistance in the home. These patients also benefit from a referral for rehabilitation services.



Educating Patients About Self-Care

Patients need to be educated about how to insert, remove, and care for the prosthetic eye. Proper hand hygiene must be observed before inserting and removing an ocular prosthesis. A suction cup may be used if there are problems with manual dexterity. Precautions, such as draping a towel over the sink and closing the sink drain, must be taken to avoid loss of the prosthesis. When educating patients or family members, a return demonstration is important to assess the level of understanding and ability to perform the procedure.

Before insertion, the inner punctal or outer lateral aspects and the superior and inferior aspects of the prosthesis must be identified by locating the identifying marks, such as a reddish color in the inner punctal area. For people with low vision, other forms of identifying markers, such as dots or notches, are used. The upper lid is raised high enough to create a space and then the patient learns to slide the prosthesis up, underneath, and behind the upper eyelid. Meanwhile, the patient pulls the lower eyelid down to help put the prosthesis in place and to have its inferior edge fall back gradually to the lower eyelid. The lower eyelid is checked for correct positioning.

To remove the prosthesis, the patient cups one hand on the cheek to catch the prosthesis, places the forefinger of the free hand against the midportion of the lower eyelid, and gazes upward. Gazing upward brings the inferior edge of the prosthesis nearer the inferior eyelid margin. With the finger pushing inward, downward, and laterally against the lower eyelid, the prosthesis slides out into the cupped hand.

Continuing and Transitional Care

An eye prosthesis can be worn and left in place for several months. Hygiene and comfort are usually maintained with daily irrigation of the prosthesis in place with normal saline solution, hard contact lens solution, or artificial tears. In the case of dry eye symptoms, the use of ophthalmic ointment lubricants or oil-based drops, such as vitamin E and mineral oil, can be helpful. Removing crusting and mucus discharge that accumulate overnight is performed with the

prosthesis in place. Malpositions may occur when wiping or rubbing the prosthesis in the socket. The prosthesis can be repositioned with the use of clean fingers. Proper wiping of the prosthesis should be a gentle temporal-to-nasal motion to avoid malpositions.

The prosthesis needs to be removed and cleaned when it becomes uncomfortable and when there is increased mucus discharge. The socket should also be rendered free of mucus and inspected for any signs of infection. Any unusual discomfort, irritation, or redness of the globe or eyelids may indicate excessive wear, debris under the shell, or lack of proper hygiene. Any infection or irritation that does not resolve needs medical attention.

OCULAR CONSEQUENCES OF SYSTEMIC DISEASE

Diabetic Retinopathy

Advancements in the treatment of diabetes have enabled patients to have a relatively normal lifespan, but many have complications of long-term diabetes. One of the most serious complications of diabetes is retinopathy. Patients with diabetes are also at higher risk of cataracts and best practices for glycemic control during cataract surgery are not known (Kiziltoprak, Tekin, Inanc, et al., 2019). See [Chapter 46](#) for a detailed discussion of diabetic retinopathy.

Cytomegalovirus Retinitis

Many ophthalmic complications have been associated with AIDS, and CMV is the most common cause of retinal inflammation in patients with AIDS. Early symptoms of CMV retinitis vary from patient to patient. Some patients complain of floaters or a decrease in peripheral vision. Some have a paracentral or central scotoma, whereas others have fluctuations in vision from macular edema. The retina often becomes thin and atrophic and susceptible to retinal tears and breaks.

CMV retinitis generally takes one of three forms: hemorrhagic, brushfire, or granular. In the hemorrhagic type, large areas of white, necrotic retina may be associated with retinal hemorrhage. In the brushfire type, a yellow-white margin begins at the edge of burned-out atrophic retina. This retinitis expands and, if untreated, involves the entire retina. In the granular type, white granular lesions in the periphery of the retina gradually expand. The white, feathery infiltration of the retina destroys sensory retina and leads to necrosis, optic atrophy, and retinal detachment.

Medical Management

Management of CMV retinitis consists of prescribing the appropriate pharmacologic agent.

Pharmacologic Therapy

Pharmacologic agents available for the treatment of CMV retinitis include ganciclovir, foscarnet, and cidofovir.

Ganciclovir is administered IV, orally, or intravitreously in the acute stage of CMV retinitis. The intravitreous form is available as a 4-mm intraocular implant or insert containing the medication embedded in a polymer-based system that slowly releases the medication. The insert is surgically placed in the posterior segment of the eye, and the medication diffuses locally to the site of the infection over a period of 5 to 8 months before the insert must be replaced. When given systemically, ganciclovir is a very potent medication; it can cause neutropenia, thrombocytopenia, anemia, and elevated serum creatinine levels. The surgically implanted sustained-release insert enables higher concentrations of ganciclovir to reach the CMV retinitis, but there are risks and complications associated with the inserts, including endophthalmitis, retinal detachment, and hypotony.

Foscarnet inhibits viral deoxyribonucleic acid (DNA) replication. It may be the medication of choice when ganciclovir is ineffective. It may be administered by IV or intravitreal injections. The combination of foscarnet and ganciclovir has been more effective than either medication alone. Nephrotoxicity may occur with systemic foscarnet, and renal function must be monitored carefully.

Cidofovir impedes CMV replication and is administered IV. Cidofovir has been shown to delay the progression of CMV retinitis significantly. Nephrotoxicity, proteinuria, and increased serum creatinine levels are significant side effects.

A nucleoside analogue such as zidovudine given in combination with one or more protease inhibitors such as ritonavir in the management of patients with AIDS has led to a major therapeutic success, gradually altering the course of the disease and transforming HIV/AIDS into a chronically manageable disease (Jacob, Jacob, & Jugulete, 2017). The immune system can then recover to a functional level. Some patients develop immune recovery uveitis, characterized by intraocular inflammation, cystoid macular edema, and the formation of epiretinal membranes. Immune recovery uveitis is managed by corticosteroids or by injection of corticosteroids into the sub-Tenon area of the eye.

Hypertension-Related Eye Changes

Long-standing hypertension is associated with atherosclerosis, and retinal changes are evidenced by the development of retinal arteriolar changes, such as tortuousness, narrowing, and a change in light reflex (Weber & Kelley, 2018). Fundoscopic examination reveals a copper or silver coloration of the arterioles and venous compression (arteriovenous nicking) at the arteriolar and venous crossings. Intraretinal hemorrhages from hypertension appear flame shaped because they occur in the nerve fiber layer of the retina.

Hypertension can also occur as an acute consequence of conditions such as pheochromocytoma, acute kidney injury, and pregnancy-induced hypertension. The retinopathy associated with these crisis states is extensive, and the manifestations include cotton-wool spots, retinal hemorrhages, retinal edema, and retinal exudates, often clustered around the macula (Weber & Kelley, 2018).

The choroid is also affected by the profound and abrupt rise in blood pressure and resulting vasoconstriction, and ischemia may result in serious retinal detachments and infarction of the retinal pigment epithelium. Ischemic optic neuropathy and papilledema may also result. Blood pressure in these more severe stages should be lowered in a controlled gradual fashion to avoid ischemia of the optic nerve and brain secondary to a too-rapid fall in blood pressure. See [Chapter 27](#) for further information about hypertension.

CRITICAL THINKING EXERCISES

1 pq You are a home health nurse visiting a 72-year-old male who has a long history of uncontrolled diabetes. He reports that recently he has been having trouble with his vision. Define nursing assessment techniques that are important in evaluating this patient. Describe your nursing priorities of care for a patient with low vision. How would these priorities change if the patient were blind?

2 ipc A 45-year-old woman presents to the emergency department complaining she has had progressive loss of vision of her left eye since the morning. She lives alone and is very afraid of going blind. The patient is diagnosed with a retinal detachment and scheduled for surgery. What members of the interdisciplinary team are essential to include? How will you facilitate an interprofessional discussion to develop strategies to decrease her fear?

3 ebp An 80-year-old patient reports to you that he has been diagnosed with AMD. What is the evidence base for offering guidelines for managing this condition? What evidence-based educational information can you share? Identify the criteria used to evaluate the strength of the evidence for these practices.

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Resources

- American Academy of Ophthalmology, www.aao.org
- American Foundation for the Blind (AFB), www.afb.org
- American Society of Ophthalmic Registered Nurses (ASORN), www.asorn.org
- Foundation Fighting Blindness, www.fightingblindness.org
- Glaucoma Research Foundation, www.glaucoma.org
- Lighthouse Guild, www.lighthouseguild.org
- MAB Community Services, www.mabcommunity.org
- Macular Degeneration Foundation, www.eyesight.org
- National Diabetes Information Clearinghouse (NDIC), www.niddk.nih.gov/health-information/diabetes
- National Eye Institute, www.nei.nih.gov
- National Federation of the Blind, www.nfb.org
- Prevent Blindness, www.preventblindness.org
- Research to Prevent Blindness, www.rpbusa.org

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Assessment and Management of Patients with Hearing and Balance Disorders

LEARNING OUTCOMES

On completion of this chapter, the learner will be able to:

1. Describe the anatomy and physiology of the ear as well as the methods used to assess hearing and balance disorders.
2. List the manifestations that may be exhibited by a person with hearing and balance disorders.
3. Identify ways to communicate effectively with a person with a hearing disorder, incorporating the differences between Deaf culture and deafness.
4. Differentiate disorders of the external ear from those of the middle ear and inner ear.
5. Compare the various types of surgical procedures used for managing middle ear disorders and appropriate nursing care.
6. Use the nursing process as a framework for care of the patient undergoing mastoid surgery or of the patient with vertigo.
7. Recognize the different types of inner ear disorders, including the clinical manifestations, diagnosis, and management.

NURSING CONCEPTS

Assessment
Communication
Family
Infection
Inflammation
Sensory Perception
Stress

GLOSSARY

acute otitis media: inflammation in the middle ear lasting less than 6 weeks

cholesteatoma: tumor of the middle ear or mastoid, or both, that can destroy structures of the temporal bone

chronic otitis media: repeated episodes of acute otitis media causing irreversible tissue damage

conductive hearing loss: loss of hearing in which efficient sound transmission to the inner ear is interrupted by some obstruction or disease process

Deaf culture: a community that consists of a group of people who are connected by their use of sign language

deafness: partial or complete loss of the ability to hear

dizziness: altered sensation of orientation in space

endolymphatic hydrops: dilation of the endolymphatic space of the inner ear; the pathologic correlate of Ménière's disease

exostoses: small, hard, protrusions in the lower posterior bony portion of the ear canal

external otitis: inflammation of the external auditory canal (*synonym:* otitis externa)

labyrinthitis: inflammation of the labyrinth of the inner ear

Ménière's disease: **condition of the inner ear characterized by a triad of symptoms:** episodic vertigo, tinnitus, and fluctuating sensorineural hearing loss

middle ear effusion: fluid in the middle ear without evidence of infection

myringotomy: incision in the tympanic membrane (*synonym:* tympanotomy)

nystagmus: involuntary rhythmic eye movement

ossiculoplasty: surgical reconstruction of the middle ear bones to restore hearing

otalgia: sensation of fullness or pain in the ear

otorrhea: drainage from the ear

otosclerosis: a condition characterized by abnormal spongy bone formation around the stapes

presbycusis: progressive hearing loss associated with aging

rhinorrhea: drainage from the nose

sensorineural hearing loss: loss of hearing related to damage to the end organ for hearing or cranial nerve VIII, or both

tinnitus: subjective perception of sound with internal origin; unwanted noises in the head or ear most often described as ringing in the ears

tympanoplasty: surgical repair of the tympanic membrane

vertigo: illusion of movement in which the individual or the surroundings are sensed as moving

The ear is a delicate sensory organ with the dual functions of hearing and balance. The sense of hearing is essential for normal development and maintenance of speech as well as the ability to communicate with others. Balance, or equilibrium, is essential for maintaining body movement, position, and coordination.

The early detection and accurate diagnosis of disorders is necessary for preservation of normal hearing and balance. The diagnosis and treatment of these disorders requires skilled health care professionals such as otolaryngologists, internists, audiologists, and nurses. This chapter provides an overview of the anatomy and physiology of the ear and addresses the general assessment and management of hearing and balance disorders common to adults seen in many health care settings.

ASSESSMENT OF THE EAR

Anatomic and Physiologic Overview

The cranium encloses and protects the brain and surrounding structures, providing attachment for various muscles that control head and jaw movements. The ears are located on either side of the cranium at approximately eye level.

Anatomy of the External Ear

The external ear includes the auricle (pinna) and the external auditory canal (see Fig. 59-1). The external ear is separated from the middle ear by a disc-shaped structure called the *tympanic membrane* (eardrum).

Auricle

The auricle, attached to the side of the head by skin, is composed mainly of cartilage, except for the fat and subcutaneous tissue in the earlobe. The auricle collects the sound waves and directs vibrations into the external auditory canal.

External Auditory Canal

The external auditory canal is approximately 2 to 3 cm long (Norris, 2019). The lateral third is an elastic cartilaginous and dense fibrous framework to

which thin skin is attached. The medial two thirds is bone lined with thin skin. The external auditory canal ends at the tympanic membrane.

The skin of the canal contains hair, sebaceous glands, and ceruminous glands, which secrete a brown, waxlike substance called *cerumen* (ear wax). The ear's self-cleaning mechanism moves old skin cells and cerumen to the outer part of the ear.

Just anterior to the external auditory canal is the temporomandibular joint. The head of the mandible can be felt by placing a fingertip in the external auditory canal while the patient opens and closes the mouth.

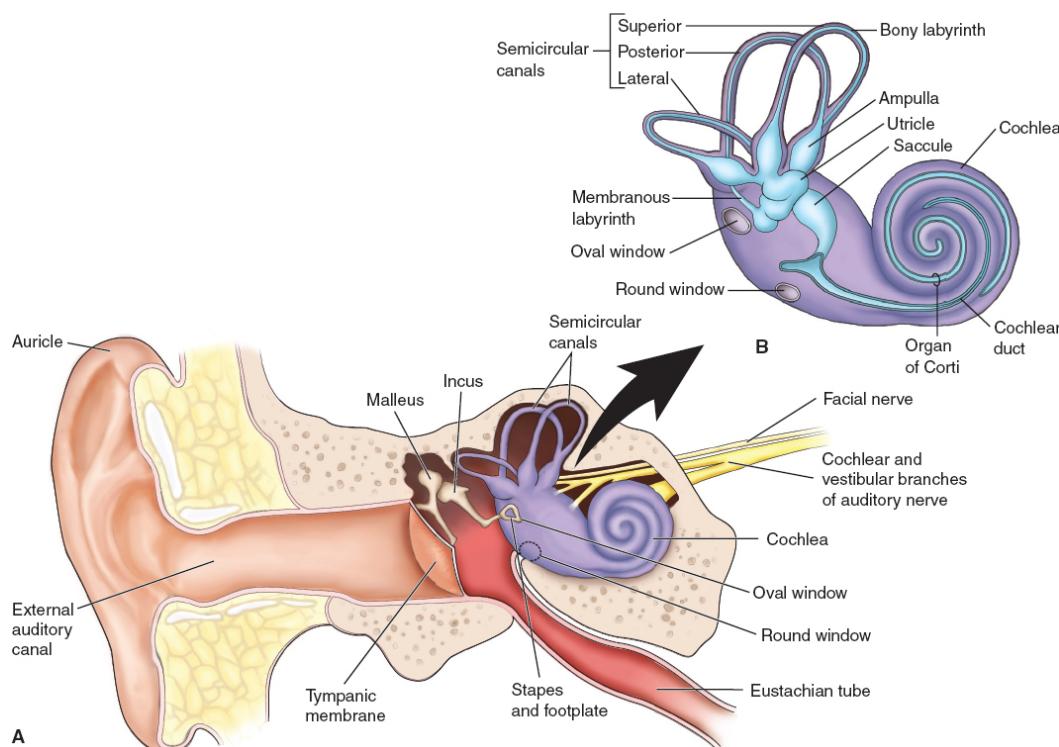


Figure 59-1 • A. Anatomy of the ear. B. The inner ear.

Anatomy of the Middle Ear

The middle ear, an air-filled cavity, includes the tympanic membrane laterally and the otic capsule medially. The middle ear cleft lies between the two. The middle ear is connected to the nasopharynx by the eustachian tube and is continuous with air-filled cells in the adjacent mastoid portion of the temporal bone.

The eustachian tube, which is approximately 1 mm wide and 35 mm long, connects the middle ear to the nasopharynx. Normally, the eustachian tube is closed, but it opens by action of the tensor veli palatini muscle when the person performs a Valsalva maneuver, yawns, or swallows. It drains normal

and abnormal secretions of the middle ear and equalizes pressure in the middle ear with that of the atmosphere.

Tympanic Membrane

The tympanic membrane (eardrum), about 1 cm in diameter and very thin, is normally pearly gray and translucent (Weber & Kelley, 2018). It consists of three layers of tissue: an outer layer, continuous with the skin of the ear canal; a fibrous middle layer; and an inner mucosal layer, continuous with the lining of the middle ear cavity. Approximately 80% of the tympanic membrane is composed of all three layers and is called the *pars tensa*. The remaining 20% lacks the middle layer and is called the *pars flaccida*. The absence of this fibrous middle layer makes the pars flaccida more vulnerable to pathologic disorders than the pars tensa. Distinguishing landmarks include the annulus, the fibrous border that attaches the eardrum to the temporal bone; the short process of the malleus; the long process of the malleus; the umbo of the malleus, which attaches to the tympanic membrane in the center; the pars flaccida; and the pars tensa (see [Fig. 59-2](#)).

The tympanic membrane protects the middle ear and conducts sound vibrations from the external canal to the ossicles. The sound pressure is magnified 22 times as a result of transmission from a larger area to a smaller one.

Ossicles

The middle ear contains the ossicles, the three smallest bones of the body: the malleus, the incus, and the stapes (Norris, 2019). The ossicles, which are held in place by joints, muscles, and ligaments, assist in the transmission of sound. Two small fenestrae (oval and round windows), located in the medial wall of the middle ear, separate the middle ear from the inner ear. The footplate of the stapes sits in the oval window, secured by a fibrous annulus (ring-shaped structure). The footplate transmits sound to the inner ear. The round window, covered by a thin membrane, provides an exit for sound vibrations (see [Fig. 59-1](#)).

Anatomy of the Inner Ear

The inner ear is housed deep within the temporal bone. The organs for hearing (cochlea) and balance (semicircular canals), as well as cranial nerves VII (facial nerve) and VIII (vestibulocochlear nerve), are all part of this complex anatomy (see [Fig. 59-1](#)). The cochlea and semicircular canals are housed in the bony labyrinth. The bony labyrinth surrounds and protects the membranous labyrinth, which is bathed in a fluid called *perilymph*.

Membranous Labyrinth

The membranous labyrinth is composed of the utricle, the saccule, the cochlear duct, the semicircular canals, and the organ of Corti, all of which are surrounded by a fluid called *endolymph*. The three semicircular canals—posterior, superior, and lateral, which lie at 90-degree angles to one another—contain sensory receptor organs that are arranged to detect rotational movement. These receptor end organs are stimulated by changes in the rate or direction of a person's movement. The utricle and saccule are involved with linear movements.

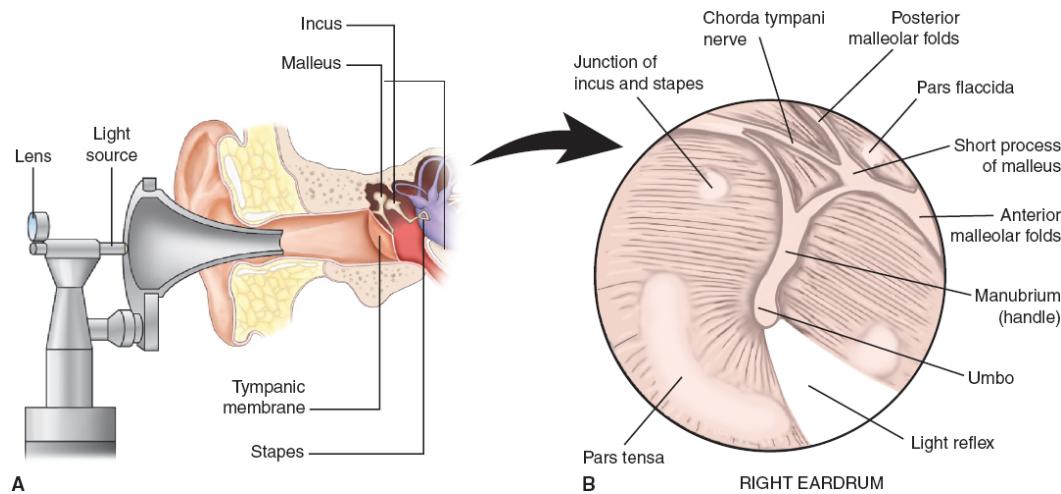


Figure 59-2 • Technique for using the otoscope (A) to see the tympanic membrane (B).

Organ of Corti

The organ of Corti is housed in the cochlea, a snail-shaped, bony tube about 3.5 cm long with two and a half spiral turns. Membranes separate the cochlear duct (scala media) from the scala vestibuli and the scala tympani from the basilar membrane. The organ of Corti is located on the basilar membrane that stretches from the base to the apex of the cochlea. As sound vibrations enter the perilymph at the oval window and travel along the scala vestibuli, they pass through the scala tympani, enter the cochlear duct, and cause movement of the basilar membrane. The organ of Corti, also referred to as the end organ for hearing, transforms mechanical energy into neural activity and separates sounds into different frequencies. This electrochemical impulse travels through the acoustic nerve to the temporal cortex of the brain to be interpreted as meaningful sound. In the internal auditory canal, the cochlear (acoustic) nerve, arising from the cochlea, joins the vestibular nerve, arising from the semicircular canals, utricle, and saccule, to become the vestibulocochlear nerve (cranial nerve VIII). This canal also houses the facial nerve and the blood supply from the ear to the brain.

Function of the Ears

Hearing

Hearing is conducted over two pathways: air and bone. Sounds transmitted by air conduction travel over the air-filled external and middle ear through vibration of the tympanic membrane and ossicles. Sounds transmitted by bone conduction travel directly through bone to the inner ear, bypassing the tympanic membrane and ossicles. Normally, air conduction is the more efficient pathway.

Sound Conduction and Transmission

Sound enters the ear through the external auditory canal and causes the tympanic membrane to vibrate. These vibrations transmit sound through the lever action of the ossicles to the oval window as mechanical energy. This mechanical energy is then transmitted through the inner ear fluids to the cochlea, stimulating the hair cells, and is subsequently converted to electrical energy. The electrical energy travels through the vestibulocochlear nerve to the central nervous system, where it is interpreted in its final form as sound.

Vibrations transmitted by the tympanic membrane to the ossicles of the middle ear are transmitted to the cochlea, located in the labyrinth of the inner ear. The stapes rocks, causing vibrations (waves) in fluids contained in the inner ear. These fluid waves cause movement of the basilar membrane, stimulating the hair cells of the organ of Corti in the cochlea to move in a wavelike manner. The movements of the tympanic membrane initiate electrical currents that stimulate the various areas of the cochlea. The hair cells generate neural impulses that are encoded and then transferred to the auditory cortex in the brain, where they are decoded into a sound message.

The footplate of the stapes receives impulses transmitted by the incus and the malleus from the tympanic membrane. The round window, which opens on the opposite side of the cochlear duct, is protected from sound waves by the intact tympanic membrane, permitting motion of the inner ear fluids by sound wave stimulation. For example, in the normally intact tympanic membrane, sound waves stimulate the oval window first, and a lag occurs before the terminal effect of the stimulus reaches the round window. However, this lag phase is changed when a perforation of the tympanic membrane allows sound waves to impinge on the oval and round windows simultaneously. This effect cancels the lag and prevents the maximal effect of inner ear fluid motility and its subsequent effect in stimulating the hair cells in the organ of Corti. The result is a reduction in hearing ability (see [Fig. 59-3](#)).

Bone conduction occurs by directly stimulating bones of the skull, which send sound to the inner ear. The way in which this occurs can be demonstrated

by striking a tuning fork and placing it directly on the skull above the ear. Sound is transmitted to the inner ear.

Balance and Equilibrium

Body balance is maintained by cooperation of muscles and joints of the body (proprioceptive system), the eyes (visual system), and the labyrinth (vestibular system). These areas send their information about equilibrium, or balance, to the brain (cerebellar system) for coordination and perception in the cerebral cortex. The brain obtains its blood supply from the heart and arterial system. A problem in any of these areas, such as arteriosclerosis or impaired vision, can cause a disturbance of balance. The vestibular apparatus of the inner ear provides feedback regarding the movements and the position of the head and body in space.

Assessment



Assessment of hearing and balance involves inspection of the external, middle, and inner ear. Evaluation of gross hearing acuity also is included in every physical examination.

Inspection of the External Ear

Inspection of the external ear is a simple procedure, but it is often overlooked. The external ear is examined by inspection and direct palpation; the auricle and surrounding tissues should be inspected for deformities, lesions, and discharge, as well as size, symmetry, and angle of attachment to the head. Manipulation of the auricle does not normally elicit pain. If this maneuver is painful, acute external otitis is suspected (Cash & Glass, 2017). Tenderness on palpation in the area of the mastoid may indicate acute mastoiditis or inflammation of the posterior auricular node. Occasionally, sebaceous cysts and tophi (subcutaneous mineral deposits) are present on the pinna. A flaky scaliness on or behind the auricle usually indicates seborrheic dermatitis and can be present on the scalp and facial structures as well.

Otoscopic Examination

The tympanic membrane is inspected with an otoscope and indirect palpation with a pneumatic otoscope. To examine the external auditory canal and tympanic membrane, the otoscope should be held in the examiner's right hand, in a pencil-hold position, with the examiner's hand braced against the patient's face (see Fig. 59-4). This position prevents the examiner from inserting the

otoscope too far into the external canal. Using the opposite hand, the auricle is grasped and gently pulled back to straighten the canal in the adult.

The speculum is slowly inserted into the ear canal, with the examiner's eye held close to the magnifying lens of the otoscope to visualize the canal and tympanic membrane. The largest speculum that the canal can accommodate (usually 5 mm in an adult) is guided gently down into the canal and slightly forward. Because the distal portion of the canal is bony and covered by a sensitive layer of epithelium, only light pressure can be used without causing pain. The external auditory canal is examined for discharge, inflammation, or a foreign body.

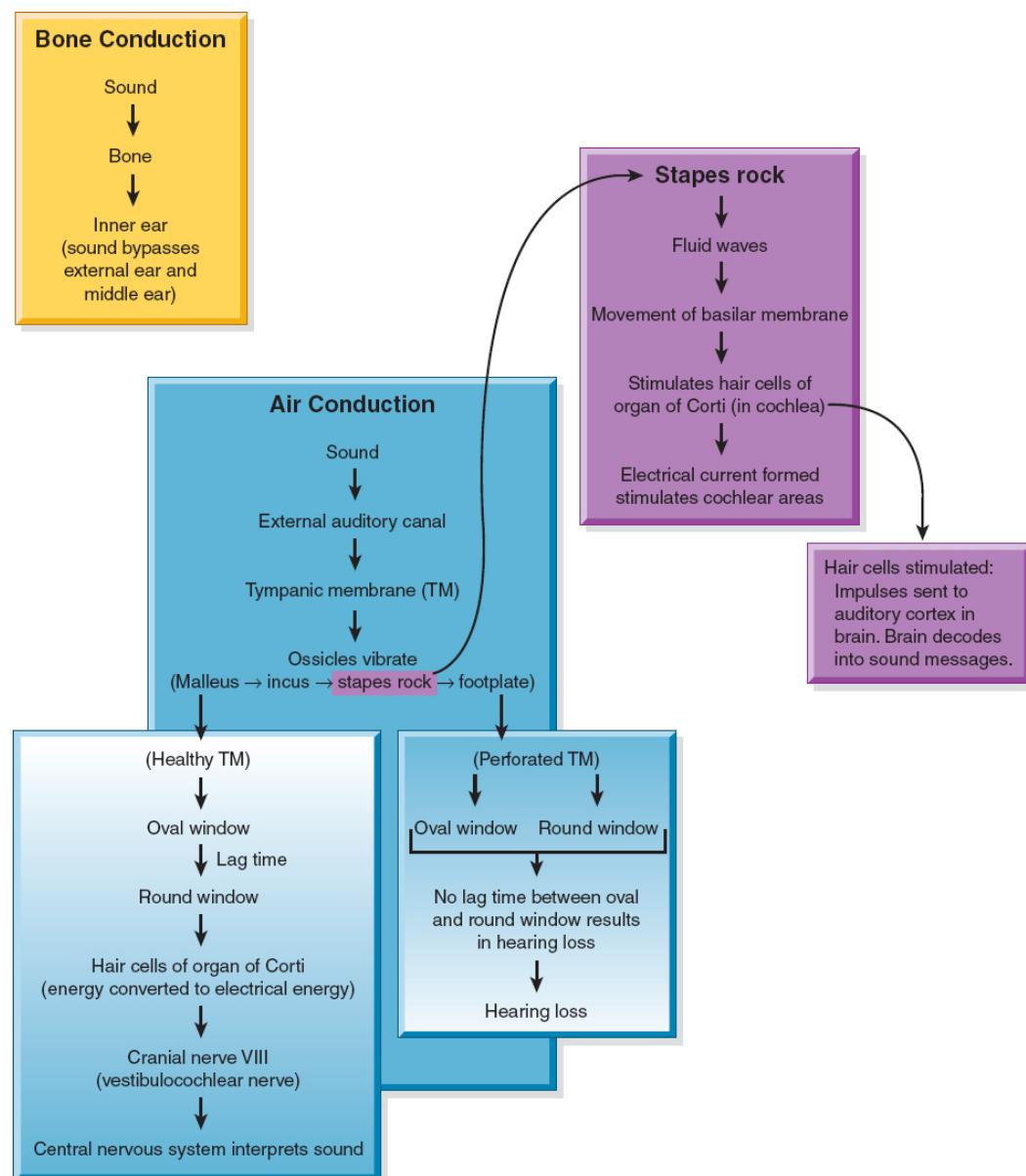


Figure 59-3 • Bone conduction compared to air conduction.

The healthy tympanic membrane is pearly gray and is positioned obliquely at the base of the canal. The following landmarks are identified, if visible (see Fig. 59-2): the pars tensa, the umbo, the manubrium of the malleus, and its short process. A slow, circular movement of the speculum allows further visualization of the malleolar folds and periphery. The position and color of the membrane and any unusual markings or deviations from normal are documented. Presence of fluid, air bubbles, blood, or masses in the middle ear should also be noted.

Proper otoscopic examination of the external auditory canal and tympanic membrane requires that the canal be free of large amounts of cerumen. Cerumen is normally present in the external canal, and small amounts should not interfere with otoscopic examination. If the tympanic membrane cannot be visualized because of cerumen, the cerumen may be removed using several methods (see later discussion of cerumen removal in section Cerumen Impaction). Cerumen buildup is a common cause of local irritation, **tinnitus** (i.e., an unwanted noise commonly described as ringing in the ears), and reversible hearing loss (Norris, 2019).

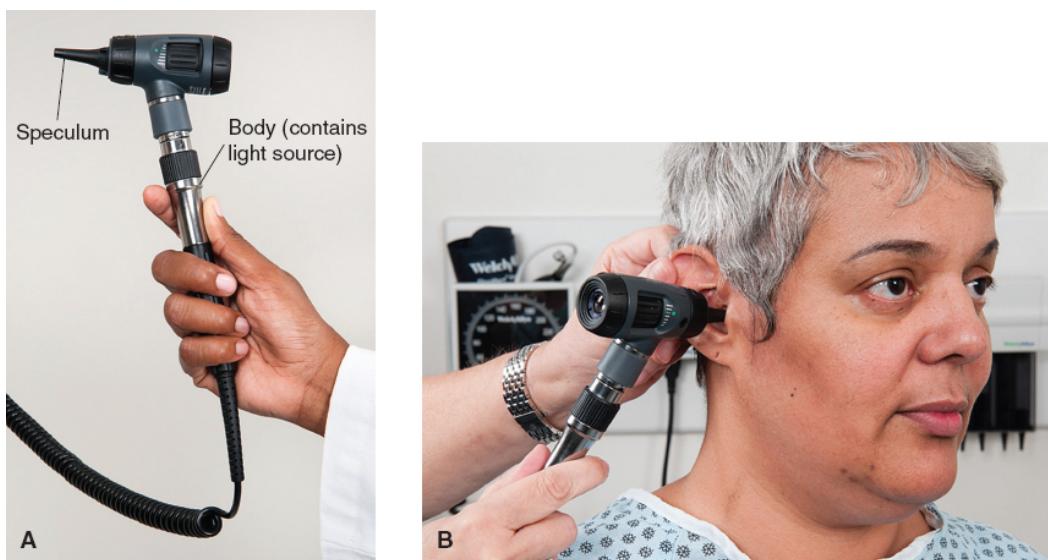


Figure 59-4 • A. The otoscope. **B.** Proper technique for examining the ear. Hold the otoscope in the right or left hand, in a “pencil-hold” position. Reprinted with permission from Weber, J., & Kelley, J. (2018). *Health assessment in nursing* (6th ed.). Philadelphia, PA: Wolters Kluwer.

Evaluation of Gross Auditory Acuity

A general estimate of hearing can be made by assessing the patient’s ability to hear a whispered phrase, testing one ear at a time. The Weber and Rinne tests may be used to distinguish conductive loss from sensorineural loss when

hearing is impaired (Weber & Kelley, 2018). These tests are part of a regular screening physical examination and are useful if a more specific assessment is needed, if hearing loss is detected, or if confirmation of audiometric results is desired.

Whisper Test

To exclude one ear from the testing, the examiner covers the untested ear with the palm of the hand. The examiner then whispers softly from a distance of 1 or 2 feet from the unoccluded ear and out of the patient's sight. The patient with normal acuity can correctly repeat what was whispered.

Weber Test

The Weber test uses bone conduction to test lateralization of sound. A tuning fork (ideally, 512 Hertz [Hz]), set in motion by grasping it firmly by its stem and tapping it on the examiner's knee or hand, is placed on the patient's head or forehead (see [Fig. 59-5A](#)). A person with normal hearing hears the sound equally in both ears or describes the sound as centered in the middle of the head. A person with conductive hearing loss, such as from otosclerosis or otitis media, hears the sound better in the affected ear. A person with sensorineural hearing loss, resulting from damage to the cochlear or vestibulocochlear nerve, hears the sound in the better-hearing ear. The Weber test is useful for detecting unilateral hearing loss (see [Table 59-1](#)).

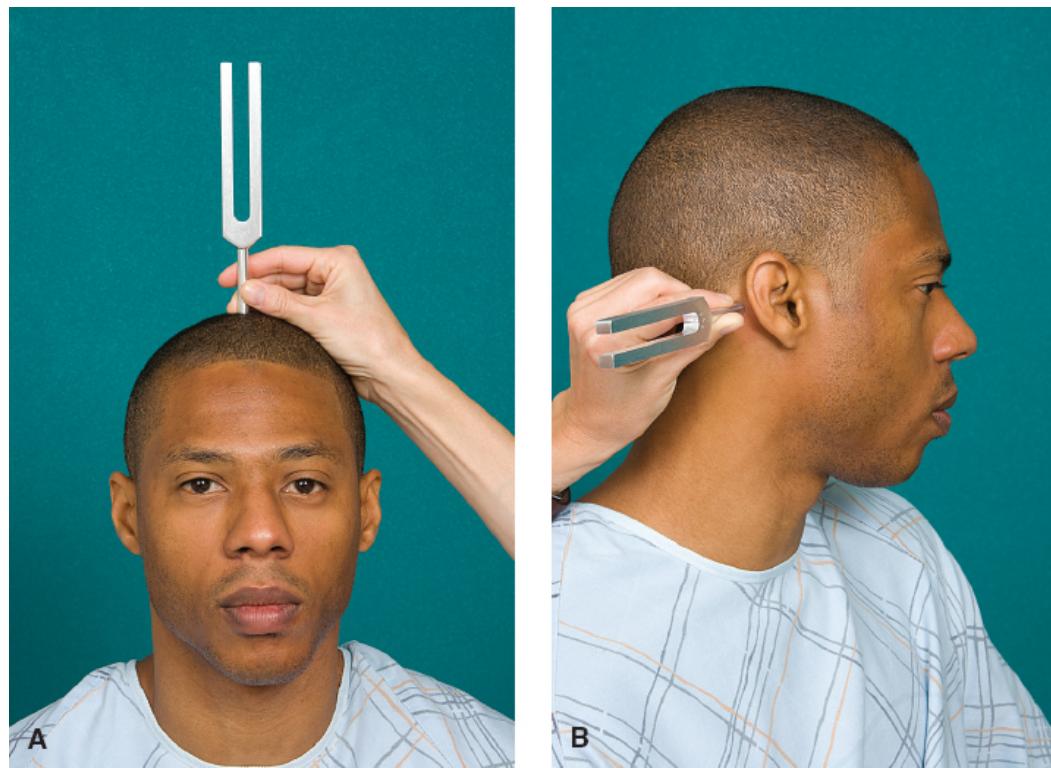


Figure 59-5 • A. The Weber test assesses bone conduction of sound. **B.** The Rinne test assesses both air and bone conduction of sound.

TABLE 59-1 Comparison of Weber and Rinne Tests

Hearing Status	Weber	Rinne
Normal hearing	Sound is heard equally in both ears.	Air conduction is audible longer than bone conduction in both ears.
Conductive hearing loss	Sound is heard best in affected ear (hearing loss).	Sound is heard as long or longer in affected ear (hearing loss).
Sensorineural hearing loss	Sound is heard best in normal hearing ear.	Air conduction is audible longer than bone conduction in affected ear.

Adapted from Weber, J., & Kelley, J. (2018). *Health assessment in nursing* (6th ed.). Philadelphia, PA: Wolters Kluwer.

Results of the Weber test are used to determine whether the patient has conductive hearing loss (sounds are heard better in the affected ear) or sensorineural hearing loss (sounds are heard better in the normal ear) (see later discussion of hearing loss).

Rinne Test

In the Rinne test (pronounced *rin-ay*), the examiner shifts the stem of a vibrating tuning fork between two positions: 2 inches from the opening of the ear canal (for air conduction) and against the mastoid bone (for bone conduction) (see Fig. 59-5B). As the position changes, the patient is asked to indicate which tone is louder or when the tone is no longer audible.

The Rinne test is useful for distinguishing between conductive and sensorineural hearing loss. A person with normal hearing reports that air-conducted sound is louder than bone-conducted sound. A person with a conductive hearing loss hears bone-conducted sound as long as or longer than air-conducted sound. A person with a sensorineural hearing loss hears air-conducted sound longer than bone-conducted sound.

Cultural Considerations

Deaf culture is distinguished with an uppercase *D*, whereas deafness as an audiologic condition is indicated by a lowercase *d*. In a sociocultural context, Deaf refers to individuals who were born with limited hearing or developed hearing loss before developing a spoken language and who use sign language (e.g., American Sign Language [ASL]) as their primary means of communication (Pendergrass, Newman, Jones, et al., 2019). Core cultural values include a sacred respect for and use of the hands, disassociation from speech, complete acceptance of being Deaf as a normal existence, full access to communication and information sharing, and self-determination (Holcomb, 2013; Padden, 1980).

According to the World Federation of the Deaf (World Federation of the Deaf [WFD], 2020), membership in the Deaf community depends on self-identification, acceptance of other members, and proficiency in a signed language. The Deaf community rejects the term *hearing-impaired* as demeaning and use of this term is discouraged (Holcomb, 2013; Moore & Levitan, 2016). Health professionals should be aware of individual differences and use the appropriate terminology when referring to Deaf, deaf, and people who are hard of hearing. Other appropriate terms used to identify Deaf people include Deaf signers, Deaf ASL users, or culturally Deaf adults.

Deaf and people who are hard of hearing are often hampered by communication barriers and an inadequate level of understanding about their language and culture by the hearing majority (Holcomb, 2013). When communicating with Deaf signers, nurses need to know that one's ability to speak is irrelevant and asking about the details of one's audiologic status is considered rude unless the individual offers this information (Mindess, 2014). Although a person who is deaf may have the ability to speak, it is important to remember that speech benefits the hearing population and the individual is in fact, deaf. As patient advocates, nurses should be mindful of the differences and similarities among Deaf signers, nonsigning deaf, and hard of hearing as

well as their own beliefs about these differences and similarities (Lewis & Keele, 2020). See the Nursing Research Profile in [Chart 59-1](#). Although various methods may be necessary, communication should always be based on patient preference.

Diagnostic Evaluation

Many diagnostic procedures are available to measure the auditory and vestibular systems indirectly. These tests are usually performed by a certified audiologist. The nurse educates the patient about the purpose, what to expect, and any possible side effects related to testing. The nurse notes trends in results because they provide information about disease progression as well as the patient's response to therapy.

Audiometry

In detecting hearing loss, audiology is the single most important diagnostic instrument. Audiometric testing is of two kinds: pure-tone audiometry, in which the sound stimulus consists of a pure or musical tone (the louder the tone before the patient perceives it, the greater the hearing loss), and speech audiometry, in which the spoken word is used to determine the ability to hear and discriminate sounds and words.

When evaluating hearing, three characteristics are important: frequency, pitch, and intensity. *Frequency* refers to the number of sound waves emanating from a source per second, measured as cycles per second, or Hertz. The normal human ear perceives sounds ranging in frequency from 20 to 20,000 Hz. The frequencies from 500 to 2000 Hz are important in understanding everyday speech and are referred to as the speech range or speech frequencies. *Pitch* is the term used to describe frequency; a tone with 100 Hz is considered of low pitch, and a tone of 10,000 Hz is considered of high pitch.

The unit for measuring loudness (*intensity* of sound) is the decibel (dB), the pressure exerted by sound. Hearing loss is measured in decibels—a logarithmic function of intensity that is not easily converted into a percentage. The critical level of loudness is approximately 30 dB. The shuffling of papers in quiet surroundings is about 15 dB; a low conversation, 40 dB; and a jet plane 100 feet away, about 150 dB. Sound louder than 80 dB is perceived by the human ear to be harsh and can be damaging to the inner ear. [Table 59-2](#) classifies hearing loss based on decibel level. In surgical treatment of patients with hearing loss, the aim is to improve the hearing level to 30 dB or better within the speech frequencies.

Chart 59-1



NURSING RESEARCH PROFILE

Nurses' Beliefs Toward Deaf and Hard of Hearing Interaction

Lewis, A., & Keele, R. (2020). Development and validation of instrument to measure nurses' beliefs toward Deaf and hard of hearing interaction. *Journal of Nursing Measurement*, 28(2). doi: 10.1891/JNM-D-19-00024

Purpose

Nurse-patient communication has a significant effect on health outcomes and quality of care. Investigating what nurses believe about interacting with Deaf signers, nonsigning deaf, and hard of hearing patients is an important step in minimizing barriers and improving nursing care. The purpose of this research was to develop and test the validity and reliability of an instrument to measure registered nurses' (RNs) beliefs toward interaction with patients who are Deaf signers, nonsigning deaf, and hard of hearing, as well as with certified interpreters.

Design

This study used a quantitative methodologic design.

Findings

Noteworthy findings were associated with the social constructs of power and control. Participant responses strongly indicated the belief that it is acceptable to relinquish the autonomy and self-determination of a Deaf signer, a nonsigning deaf, or a hard of hearing person to a hearing person. This belief is apparent in at least 65% of respondents from three groups of RNs who agreed that "questions or responses for a person who is a Deaf signer, a nonsigning deaf, or hard of hearing should be directed to hearing family members" and at least 60% of respondents from both groups of RNs agreed that "during health care interactions, most Deaf signers prefer to get by without a certified interpreter."

Data collection and analyses resulted in a 25-item D/deaf and Hard of Hearing Interaction Beliefs Scale for Registered Nurses. Psychometric analysis of two separate groups of data concluded that the newly developed scale is a reliable and valid scale to measure nurses' beliefs toward interaction with patients who are either Deaf signers, nonsigning deaf, or hard of hearing. Results of confirmatory factor analysis supported the hypothesized structure of the scale and provided some evidence for its factorial validity.

Nursing Implications

Understanding the beliefs that nurses hold toward interactions with Deaf signers, nonsigning deaf, and hard of hearing persons may shed light on nurses' general beliefs toward individuals who are Deaf signers, nonsigning deaf, and hard of hearing. Furthermore, these beliefs may influence nurses' views about the importance of appropriate and effective interaction with patients who have diverse communication needs. This understanding can

lead to the development of standards of practice and organizational policies that reflect federal laws mandating equal communication access for all. Attention to equal communication access can create a nursing culture that views communication diversity not as a barrier, but as an opportunity to open doors and promote every patient's right to autonomy and self-determination.

With audiometry, the patient wears earphones and signals to the audiologist when a tone is heard. When the tone is applied directly over the external auditory canal, air conduction is measured. When the stimulus is applied to the mastoid bone, bypassing the conductive mechanism (i.e., the ossicles), nerve conduction is tested. For accuracy, testing is performed in a soundproof room. Responses are plotted on a graph known as an audiogram, which differentiates conductive from sensorineural hearing loss.

TABLE 59-2 Severity of Hearing Loss

Loss in Decibels	Interpretation
0–15	Normal hearing
>15–25	Slight hearing loss
>25–40	Mild hearing loss
>40–55	Moderate hearing loss
>55–70	Moderate to severe hearing loss
>70–90	Severe hearing loss
>90	Profound hearing loss

Tympanogram

A tympanogram, or impedance audiometry, measures middle ear muscle reflex to sound stimulation and compliance of the tympanic membrane by changing the air pressure in a sealed ear canal. Compliance is impaired with middle ear disease.

Auditory Brain Stem Response Audiometry

The auditory brain stem response (ABR) audiometry is a detectable electrical potential from cranial nerve VIII and the ascending auditory pathways of the brain stem in response to sound stimulation. Electrodes are placed on the patient's scalp and on each earlobe (Fischbach & Fischbach, 2018). Acoustic stimuli (e.g., clicks) are made in the ear. The resulting electrophysiologic measurements can determine at which decibel level a patient hears and whether there are any impairments along the nerve pathways (e.g., tumor on cranial nerve VIII). Patients are instructed to wash and rinse their hair prior to this study but to avoid applying any other hair product. ABR audiometry

assessment should be used in conjunction with behavioral audiology for the most accurate results (Bhattacharyya, 2017).

Electronystagmography

Electronystagmography is the measurement and graphic recording of the changes in electrical potentials created by eye movements during spontaneous, positional, or calorically evoked nystagmus (see discussion of nystagmus later in this chapter). It is also used to assess the oculomotor and vestibular systems and their corresponding interaction. It helps to diagnose causes of unilateral hearing loss of unknown origin, vertigo, or tinnitus. Any vestibular suppressants, such as caffeine and alcohol, are withheld for 48 hours before testing. Medications such as tranquilizers, stimulants, or antivertigo agents are withheld for 5 days before the test (Fischbach & Fischbach, 2018).

Platform Posturography

Platform posturography is recommended for patients with dizziness and balance disorders (American Academy of Otolaryngology—Head and Neck Surgery, 2014). It can be used to determine if a patient's vertigo is worsening or to evaluate a patient's response to treatment. The integration of visual, vestibular, and proprioceptive cues (i.e., sensory integration) with motor response output and coordination of the lower limbs is tested. The patient stands on a platform, surrounded by a screen, and different conditions such as a moving platform with a moving screen or a stationary platform with a moving screen are presented. The responses from the patient are measured and indicate which of the anatomic systems may be impaired. Preparation for the testing is the same as for electronystagmography.

Sinusoidal Harmonic Acceleration

Sinusoidal harmonic acceleration, or a rotary chair, is used to assess the vestibuloocular system by analyzing compensatory eye movements in response to the clockwise and counterclockwise rotation of the chair. Although such testing cannot identify the side of the lesion in unilateral disease, it helps to identify the disease (e.g., Ménière's disease and tumors of the auditory canal) and evaluate the course of recovery. The same patient preparation is required as that for electronystagmography.

Middle Ear Endoscopy

Using instruments called endoscopes that have very small diameters and acute angles, the ear can be examined by an endoscopist specializing in

otolaryngology. Middle ear endoscopy is performed safely and effectively as an office procedure to evaluate suspected perilymphatic fistula and new-onset conductive hearing loss, the anatomy of the round window before transtympanic treatment of Ménière's disease, and the tympanic cavity before ear surgery to treat chronic middle ear and mastoid infections.

The tympanic membrane is anesthetized topically for about 10 minutes before the procedure. Then, the external auditory canal is irrigated with sterile normal saline solution. With the aid of a microscope, a tympanotomy is created with a laser beam or a myringotomy knife so that the endoscope can be inserted into the middle ear cavity. Video and photo documentation can be accomplished through the scope.

HEARING LOSS

In the United States, hearing loss has been reported to occur in approximately 2 to 3 of every 1000 births (U.S. Department of Health and Human Services [HHS], 2016). More than half of the 4000 infants born deaf each year have a hereditary disorder associated with genetic sensorineural hearing loss (Antonio, 2018). Genetic syndromes associated with hearing impairment include Waardenburg syndrome, Usher syndrome, Pendred syndrome, and Jervell and Lange-Nielsen syndrome (Antonio, 2018). [Chart 59-2](#) contains more information about hearing disorders that have a genetic cause. Hearing loss may also be acquired; causes include TORCH infections (TOxoplasmosis, Rubella, Cytomegalovirus, Herpes) during pregnancy as well as trauma or chronic exposure to loud noise (Antonio, 2018). Most hospitals or birthing centers offer universal hearing screenings for newborns after birth and prior to discharge.

Chart 59-2



GENETICS IN NURSING PRACTICE

Hearing Disorders

Several hearing disorders are associated with genetic mutations and have various patterns of inheritance:

Autosomal dominant:

- Branchiootorenal syndrome
- Neurofibromatosis type 2
- Otosclerosis
- Stickler syndrome
- Waardenburg syndrome

Autosomal recessive:

- Connexin 26 gene hearing loss (majority of cases are recessive; however, there is an autosomal dominant form that occurs less commonly)
- Jervell and Lange-Nielsen syndrome
- Pendred syndrome
- Refsum disease
- Usher syndrome

X-linked syndromic hearing loss:

- Alport syndrome

Nursing Assessments

Refer to [Chapter 4, Chart 4-2: Genetics in Nursing Practice: Genetic Aspects of Health Assessment](#)

Family History Assessment Specific to Hearing Loss

- Assess for other family members in several generations with hearing loss (autosomal dominant hearing loss).
- Inquire about genetic relatedness (e.g., individuals who are related, such as first cousins, have a higher chance to share the same recessive genes—autosomal recessive hearing loss).
- Inquire about age at onset of hearing loss.

Patient Assessment Specific to Genetic-Related Hearing Loss

- Assess for:
 - Dizziness
 - Facial numbness or weakness
 - Headaches
 - Tinnitus
- Assess for related genetic conditions, such as vision impairment (e.g., retinitis pigmentosa in Usher syndrome; thyroid disorder in Pendred

- syndrome).
- Assess for iris, pigment, and hair alterations (white forelock) seen in Waardenburg syndrome.
 - Assess for exposure to loud noises (e.g., industrial).
 - Assess for presence of rubella, toxoplasmosis, herpes, or cytomegalovirus during pregnancy.
 - Determine if patient had taken medications associated with ototoxicity.

Genetics Resources

Genetics of Hearing Loss, www.cdc.gov/ncbddd/hearingloss/genetics.html

Hear-It, www.Hear-it.org/Genetic-hearing-loss

Neurofibromatosis Network, www.nfnetwork.org/

See Chapter 6, Chart 6-7 for components of genetic counseling.

Hearing loss occurs in men more often than in women. Approximately 2% of adults between the ages of 45 and 54 years have disabling hearing loss. This percentage increases to 8.5% in the 55 to 64 age group, to 25% of adults in the 65 to 74 age group, and up to 50% for those over 75 years of age (HHS, 2016). Hearing loss is an important health issue; and as people age, hearing screening and treatment are recommended.

Many people are exposed on a daily basis to noise levels that produce high-frequency hearing loss. Occupations such as carpentry, plumbing, and coal mining have the highest risk of noise-induced hearing loss. Wise Ears was developed by the National Institute on Deafness and Other Communication Disorders (NIDCD) and the National Institute for Occupational Safety and Health (NIOSH). It aims to educate the public about noise-induced hearing loss and ways to prevent this hearing loss (NIDCD, 2010).

Conductive hearing loss usually results from an external ear disorder, such as impacted cerumen, or a middle ear disorder, such as otitis media or otosclerosis. In such instances, the efficient transmission of sound by air to the inner ear is interrupted. A **sensorineural hearing loss** involves damage to the cochlea or vestibulocochlear nerve.

Mixed hearing loss and functional hearing loss also may occur. Patients with mixed hearing loss have conductive loss and sensorineural loss, resulting from dysfunction of air and bone conduction. A functional (or psychogenic) hearing loss is nonorganic and unrelated to detectable structural changes in the hearing mechanisms; it is usually a manifestation of an emotional reaction.

Clinical Manifestations

Deafness is the partial or complete loss of the ability to hear. Early manifestations may include tinnitus, increasing inability to hear when in a group, and a need to turn up the volume of the television. Hearing loss can also

trigger changes in attitude, the ability to communicate, the awareness of surroundings, and even the ability to protect oneself, thus affecting a person's quality of life. In a classroom, a student with hearing loss may be uninterested and inattentive and have failing grades. A pedestrian with hearing loss may attempt to cross the street and fail to hear an approaching car. People with hearing loss may miss parts of conversations, and may gradually interact with others less, leading to feelings of isolation. Many people are unaware of their gradual hearing loss. Often, it is not the person with the hearing loss but the people with whom they are communicating that recognizes the change (see [Chart 59-3](#)).

For various reasons, some people with hearing loss refuse to seek medical attention or wear a hearing aid. They may feel self-conscious about wearing a hearing aid. Other people, however, may feel comfortable asking those with whom they are trying to communicate to let them know whether difficulties in communication exist. The attitudes and behaviors of patients who need hearing assistance should be taken into account when counseling them. The decision to wear a hearing aid is a personal one that is affected by these attitudes and behaviors.

Chart 59-3



ASSESSMENT

Assessing for Hearing Loss

The nurse should be alert to the following:

Speech deterioration: The person who slurs words, drops word endings, or produces flat-sounding speech may not be hearing correctly. Hearing guides the voice, both in loudness and in pronunciation.

Fatigue: If a person tires easily when listening to conversation or to a speech, fatigue may be the result of straining to hear. Under these circumstances, a person may become easily irritable.

Indifference: It is easy for the person who cannot hear what others say to become depressed and disinterested in life in general.

Social withdrawal: Not being able to hear what is going on causes a person who is hard of hearing to withdraw from situations that might prove embarrassing.

Insecurity: Lack of self-confidence and fear of mistakes create a feeling of insecurity in many people who are hard of hearing. No one likes to say the wrong thing or do anything that might appear foolish.

Indecision and procrastination: Loss of self-confidence makes it increasingly difficult for a person who is hard of hearing to make decisions.

Suspiciousness: The person who is hard of hearing—who often hears only part of what is being said—may suspect that others are talking about them, or that portions of the conversation are deliberately spoken softly so that they will not hear them.

False pride: The person who is hard of hearing wants to conceal the hearing loss and thus often pretends to be hearing when they actually is not.

Loneliness and unhappiness: Although everyone wishes for quiet now and then, enforced silence can be boring and even somewhat frightening. People with a hearing loss often feel isolated.

Tendency to dominate the conversation: Many people who are hard of hearing tend to dominate the conversation, knowing that as long as it is centered on them and they can control it, they are unlikely to be embarrassed by some mistake.

Prevention

Many environmental factors have an adverse effect on the auditory system and with time result in permanent sensorineural hearing loss. The most common is noise. Noise (unwanted and unavoidable sound) has been identified as one of today's environmental hazards. The volume of noise that surrounds us daily has increased into a potentially dangerous source of physical and psychological damage.

Loud, persistent noise has been found to cause constriction of peripheral blood vessels, increased blood pressure and heart rate (because of increased secretion of adrenalin), and increased gastrointestinal activity. Although research is needed to address the overall effects of noise on the human body, a

quiet environment is more conducive to peace of mind. A person who is ill feels more at ease when noise is kept to a minimum.

Chart 59-4 RISK FACTORS

Hearing Loss

- Congenital malformations of the cranial structure (ear)
- Family history of sensorineural impairment
- Low birth weight (<1500 g)
- Perforation of the tympanic membrane
- Recurrent ear infections
- Use of ototoxic medications (e.g., gentamycin, loop diuretics)

Adapted from Norris, T. (2019). *Porth's pathophysiology: Concepts of altered health status* (10th ed.). Philadelphia, PA: Wolters Kluwer.

Numerous factors contribute to hearing loss (see [Chart 59-4](#)). *Noise-induced hearing loss* refers to hearing loss that follows a long period of exposure to loud noise (e.g., heavy machinery, engines, artillery, rock-band music). *Acoustic trauma* refers to hearing loss caused by a single exposure to an extremely intense noise, such as an explosion. Usually, noise-induced hearing loss occurs at a high frequency (about 4000 Hz). However, with continued noise exposure, the hearing loss can become more severe and include adjacent frequencies. The minimum noise level known to cause noise-induced hearing loss, regardless of duration, is about 85 to 90 dB.

Noise exposure is inherent in many jobs (e.g., mechanics, printers, pilots, flight attendants, musicians) and in hobbies such as woodworking and hunting. Occupational noise level regulations are based on the amount of noise a person is exposed to over an 8-hour work shift, with the maximum legal limits being 85 dB per the NIOSH or 90 dB per the Occupational Safety and Health Administration (OSHA), with a peak sound pressure of 135 dB (Centers for Disease Control and Prevention [CDC], 2018). NIOSH recommends and OSHA requires that workers wear ear protection to prevent noise-induced hearing loss when exposed to noise above the legal limits. Ear protection against noise is the most effective preventive measure available. Hearing loss due to noise is permanent because the hair cells in the organ of Corti are destroyed.



Gerontologic Considerations

With aging, changes occur in the ear that may eventually lead to hearing deficits. Although few changes occur in the external ear, cerumen tends to become harder and drier, posing a greater chance of impaction. In the middle ear, the tympanic membrane may atrophy or become sclerotic. In the inner ear, cells at the base of the cochlea degenerate. A familial predisposition to sensorineural hearing loss is also seen, manifested by inability to hear high-frequency sounds, followed in time by the loss of middle and lower frequencies. The term **presbycusis** is used to describe this progressive hearing loss (Eliopoulos, 2018).

In addition to age-related changes, other factors can affect hearing in the older adult population, such as lifelong exposure to loud noises. Psychogenic factors, other disease processes (e.g., diabetes), and medications may be partially responsible for sensorineural hearing loss. Certain medications, such as aminoglycosides, aspirin, loop diuretics, and platinum-based antineoplastic medications have ototoxic effects when kidney changes result in delayed medication excretion and increased levels of the medications in the blood.

Even with the best health care, people with hearing loss must learn to adjust. Care of older patients includes recognizing emotional reactions related to hearing loss, such as suspicion of others because of an inability to hear adequately; frustration and anger, with repeated statements such as “I didn’t hear what you said!”; and feelings of insecurity because of the inability to hear the telephone or alarms. The Americans with Disabilities Act (ADA) of 1990 requires that all emergency services are accessible to people who have text message telephones (teletypewriters [TTYs]). In addition, all 911 centers in the United States must be accessible to people with TTYs.

Depression, isolation, and a decrease in cognitive function can have a negative impact on quality of life in the older adult with hearing loss. Feelings of social isolation, confusion, alterations in daily living activities, and increasing risk of falls have all been associated with hearing loss in older adults (Shukla, Reed, Armstrong, et al., 2019). Hearing loss has also been identified as a factor associated with a significant increase in the risk of hospitalization, readmission, and increased mortality (Hsu, McKee, Roscigno, et al., 2019). Additionally, hearing loss can interfere with relationships due to a loss of communication. A hearing screening is recommended as one component of the physical examination for the older adult when joining Medicare for the first time. “Welcome to Medicare” examinations and annual screenings are also important.

Medical Management

If a hearing loss is permanent or untreatable or if the patient elects not to be treated, aural rehabilitation (discussed at the end of the chapter) may be beneficial.

Nursing Management

Early detection of hearing loss is in the plans for *Healthy People 2030*, and nurses are in a position to assist in meeting this goal if included (Haskins, 2017; HHS, 2017). Another possible objective is for people diagnosed with hearing loss or deafness to use rehabilitation services and supplemental devices to improve communication with hearing people. Resources are available in workplaces and in schools. Questions used to assess for hearing loss may include:

- Have you experienced any hearing loss in the past?
- Are you experiencing any hearing loss now?
- Do your family members think that you are having difficulty hearing or experiencing any hearing loss?

These questions should be included in any routine nursing assessment and referrals made for further evaluation as needed.

Nurses who understand the different types of hearing loss are more successful in adopting a communication style to fit the needs and preferences of each patient. Trying to speak in a loud voice to a person who cannot hear high-frequency sounds only makes understanding more difficult. However, strategies such as talking into the better-hearing ear and using gestures and facial expressions can help (see [Chart 59-5](#)).

Chart 59-5

Communicating with People Who Are Deaf or Have Severe Hearing Loss

For the Person Who Is Hard of Hearing Whose Speech Is Difficult to Understand

- Determine how the person prefers to communicate with others. Do not assume that writing, gestures, or other means are the best or preferred technique.
- Consider if the person uses sign language. Interpreters are available from American Sign Language Services, Inc. (ASLI). These specialists provide the best means of communication, providing accurate, professional services.
- Devote full attention to what the person is saying. Look and listen—do not try to attend to another task while listening.
- Engage the speaker in conversation when it is possible for you to anticipate the replies. This enables you to become accustomed to any peculiarities in speech patterns.
- Try to determine the essential context of what is being said; you can often fill in the details from context.
- Do not try to appear as if you understand if you do not.
- If you cannot understand at all or have serious doubt about your ability to understand what is being said, have the person write the message rather than risk misunderstanding. Having the person repeat the message in speech, after you know its content, also aids you in becoming accustomed to the person's pattern of speech.
- Written communication is an excellent resource. Material should be written at a third-grade level so that the majority of people can understand it.

For the Person Who Is Hard of Hearing Who Speech Reads

- Be aware that speech or lip reading is ineffective because the majority of English sounds are not clearly visible on the lips and even the most proficient lip readers understand less than 30% of verbal communication.
- When speaking, always face the person as directly as possible.
- Make sure that your face is as clearly visible as possible. Locate yourself so that your face is well lighted; avoid being silhouetted against strong light. Do not obscure the person's view of your mouth in any way; avoid talking with any object held in your mouth.
- Be sure that the patient knows the topic or subject before going ahead with what you plan to say. This enables the person to use contextual clues in speech reading.
- Speak slowly and distinctly, pausing more frequently than you would normally.

- If you question whether some important direction or instruction has been understood, check to be certain that the patient has the full meaning of your message.
- If, for any reason, your mouth must be covered (as with a mask), you must direct or instruct the patient, write or communicate the message by another means.

A major issue for many people who are deaf or hard of hearing is that they have other health problems that often do not receive attention, in large part because of communication barriers with their health care providers. To meet the health care needs of these patients, providers are legally obligated to make accommodations for the most effective patient communication and understanding. Providing certified interpreters for those who can communicate through sign language is essential in many situations to ensure effective communication.

During health care and screening procedures, the provider (e.g., dentist, physician, nurse) must be aware that patients who are deaf or hard of hearing are unable to read lips, see a signer, or read written materials in the dark rooms required during some diagnostic tests. The same situation exists if the provider is wearing a mask or is not in sight (e.g., x-ray studies, magnetic resonance imaging [MRI], colonoscopy).

Nurses and other health care providers must work with patients who are deaf or hard of hearing and their families to identify practical and effective means of communication. Nurses can serve as catalysts throughout the health care system to ensure that accommodations are made to meet the communication needs of every patient.

CONDITIONS OF THE EXTERNAL EAR

Cerumen Impaction

Cerumen normally accumulates in the external canal in various amounts and colors. Although wax does not usually need to be removed, impaction occasionally occurs, causing **otalgia** (a sensation of fullness or pain in the ear) with or without a hearing loss. Accumulation of cerumen as a cause of hearing loss is especially significant in older adult patients (Eliopoulos, 2018). Attempts to clear the external auditory canal with matches, hairpins, and other implements are dangerous because trauma to the skin, infection, and damage to the tympanic membrane can occur.

Management

Cerumen can be removed by irrigation, suction, or instrumentation. Unless the patient has a perforated eardrum or an inflamed external ear (i.e., otitis externa), gentle irrigation with warm water usually helps remove impacted cerumen, particularly if it is not tightly packed in the external auditory canal. For successful removal, the water stream must flow behind the obstructing cerumen to move it first laterally and then out of the canal. To prevent injury, the lowest effective pressure should be used. However, if the eardrum behind the impaction is perforated, water can enter the middle ear, producing acute vertigo and infection. If irrigation is unsuccessful, direct visual, mechanical removal can be performed by a trained health care provider on a patient who is cooperative.



Quality and Safety Nursing Alert

Warm water (never cold or hot) and gentle (not forceful) irrigation should be used to remove cerumen. Irrigation that is too forceful can cause perforation of the tympanic membrane, and ice water causes vomiting.

Instilling a few drops of warmed glycerin, mineral oil, or half-strength hydrogen peroxide into the ear canal for 30 minutes prior to irrigation can soften cerumen before its removal. Ceruminolytic agents, such as peroxide in glycercyl, are available. The use of any softening solution two or three times a day for several days is generally sufficient. If cerumen cannot be dislodged by these methods, instruments, such as a cerumen curette, aural suction, and a binocular microscope for magnification, can be used. The use of instruments such as a cerumen curette for cerumen removal is reserved for otolaryngologists and nurses with specialized training because of the danger of perforating the tympanic membrane or excoriating the external auditory canal.

Foreign Bodies

Some objects are inserted intentionally into the ear by adults who may have been trying to clean the external canal or relieve itching, or by children who introduce peas, beans, pebbles, toys, and beads. Insects may also enter the ear canal. In either case, the effects may range from no symptoms to profound pain and decreased hearing.

Management

Removing a foreign body from the external auditory canal can be quite challenging. The three standard methods for removing foreign bodies are the same as those for removing cerumen: irrigation, suction, and instrumentation. The contraindications for irrigation are also the same. Foreign vegetable bodies and insects tend to swell; thus, irrigation is contraindicated. Usually, an insect can be dislodged by instilling mineral oil, which will kill the insect and allow it to be removed.

Attempts to remove a foreign body from the external canal may be dangerous in unskilled hands. The object may be pushed completely into the bony portion of the canal, lacerating the skin and perforating the tympanic membrane. In rare circumstances, the foreign body may have to be extracted in the operating room with the patient under general anesthesia.

External Otitis (Otitis Externa)

External otitis (i.e., otitis externa) refers to an inflammation of the external auditory canal. Causes include water in the ear canal (swimmer's ear); trauma to the skin of the ear canal, permitting entrance of organisms into the tissues; and systemic conditions, such as vitamin deficiency and endocrine disorders. Bacterial or fungal infections are most frequently encountered. The most common bacterial pathogens associated with external otitis are *Staphylococcus aureus* and *Pseudomonas* species. The most common fungus isolated in both normal and infected ears is *Aspergillus* (Norris, 2019). External otitis is often caused by a dermatosis such as psoriasis, eczema, or seborrheic dermatitis. Even allergic reactions to hair spray, hair dye, and permanent wave lotions can cause dermatitis, which clears when the offending agent is removed.

Clinical Manifestations

Patients usually report pain; discharge from the external auditory canal; aural tenderness (usually not present in middle ear infections); and occasionally fever, cellulitis, and lymphadenopathy. Other symptoms may include pruritus and hearing loss or a feeling of fullness in the ear. On otoscopic examination, the ear canal is erythematous and edematous. Discharge may be yellow or green and foul smelling. In fungal infections, hairlike black spores may be visible.

Medical Management

The principles of therapy are aimed at relieving the discomfort, reducing the swelling of the ear canal, and eradicating the infection. Patients may require analgesic medications for the first 48 to 96 hours. Treatment most often

includes antimicrobial or antifungal otic medications given by dropper at room temperature. In bacterial infection, a combination antibiotic and corticosteroid agent may be used to soothe inflamed tissues (Norris, 2019).

Nursing Management

Nurses should instruct patients not to clean the external auditory canal with cotton-tipped applicators and to avoid events that traumatize the external canal, such as scratching the canal with the fingernail or other objects. Trauma may lead to infection of the canal. Patients should also avoid getting the canal wet when swimming or shampooing the hair. A cotton ball or lamb's wool can be covered in a water-insoluble gel such as petrolatum jelly and placed in the ear as a barrier to the canal getting wet. Infection can be prevented by using antiseptic otic preparations after swimming (e.g., Swim-Ear, Ear Dry), unless there is a history of tympanic membrane perforation or a current ear infection (see [Chart 59-6](#)).

Malignant External Otitis

A more serious, although rare, external ear infection is malignant external otitis (temporal bone osteomyelitis). This is a progressive, debilitating, and occasionally fatal infection of the external auditory canal, the surrounding tissue, and the base of the skull. *Pseudomonas aeruginosa* is usually the infecting organism in patients with low resistance to infection (e.g., patients with acquired immune deficiency syndrome (AIDS). Successful treatment includes administration of antibiotics (usually intravenously [IV]) and aggressive local wound care. Standard parenteral antibiotic treatment includes the combination of an antipseudomonal agent and an aminoglycoside, both of which have potentially serious side effects. Because aminoglycosides are nephrotoxic and ototoxic, serum aminoglycoside levels and kidney and auditory function must be monitored during therapy. Local wound care includes limited débridement of the infected tissue, including bone and cartilage, depending on the extent of the infection.

Chart 59-6



PATIENT EDUCATION

Prevention of Otitis Externa

The nurse instructs the patient to:

- protect the external canal when swimming, showering, or washing hair. Use ear plugs or place a cotton ball covered in petrolatum jelly in the ear, and wear a swim cap. The external canal may be dried afterward with a hair dryer on low heat.
- place alcohol drops in the external canal to act as an astringent to help prevent infection after water exposure.
- prevent trauma to the external canal. Procedures, foreign objects (e.g., bobby pin), scratching, or any other trauma to the canal that breaks the skin integrity may cause infection.
- be aware that if otitis externa is diagnosed, refrain from any water sport activity for approximately 7 to 10 days to allow the canal to heal completely. Recurrence is highly likely unless you allow the external canal to heal completely.

Masses of the External Ear

Exostoses are small, hard, bony protrusions found in the lower posterior bony portion of the ear canal; they usually occur bilaterally. The skin covering the exostosis is normal. It is believed that exostoses are caused by exposure to cold water, as in scuba diving or surfing. The usual treatment, if any, is surgical excision.

Malignant tumors also may occur in the external ear. Most common are basal cell carcinomas on the pinna and squamous cell carcinomas in the ear canal. If untreated, squamous cell carcinoma may spread through the temporal bone, causing facial nerve paralysis and hearing loss. Carcinomas must be treated surgically.

CONDITIONS OF THE MIDDLE EAR

Tympanic Membrane Perforation

Perforation of the tympanic membrane is usually caused by infection or trauma. Sources of trauma include skull fracture, injury from explosion, or a severe blow to the ear. Less frequently, perforation is caused by foreign objects (e.g., cotton-tipped applicators, hairpins, keys) that have been pushed too far into the external auditory canal. In addition to tympanic membrane perforation,

injury to the ossicles and even the inner ear may result from this type of trauma. During infection, the tympanic membrane can rupture if the pressure in the middle ear exceeds the atmospheric pressure in the external auditory canal.

Medical Management

Although most tympanic membrane perforations heal spontaneously within weeks after rupture, some may take several months to heal. Some perforations persist because scar tissue grows over the edges of the perforation, preventing extension of the epithelial cells across the margins and final healing. In the case of a head injury or temporal bone fracture, a patient is observed for evidence of cerebrospinal fluid **otorrhea** or **rhinorrhea**—a clear, watery drainage from the ear or nose, respectively. While healing, the ear must be protected from water entering the ear canal.

Surgical Management

Perforations that do not heal on their own may require surgery. The decision to perform a tympanoplasty (see later section in this chapter) is usually based on the need to prevent potential infection from water entering the ear or the desire to improve the patient's hearing. Performed on an outpatient basis, tympanoplasty may involve a variety of surgical techniques. In all techniques, tissue (commonly from the temporalis fascia) is placed across the perforation to allow healing. Surgery is usually successful in closing the perforation permanently and improving hearing.

Acute Otitis Media

Ear infections can occur at any age; however, they are most commonly seen in children. **Acute otitis media (AOM)** is an acute infection of the middle ear, lasting less than 6 weeks. Pathogens that cause AOM are usually bacterial or viral and enter the middle ear after eustachian tube dysfunction caused by obstruction related to upper respiratory infections, inflammation of surrounding structures (e.g., rhinosinusitis, adenoid hypertrophy), or allergic reactions (e.g., allergic rhinitis) (Norris, 2019). Bacteria can enter the eustachian tube from contaminated secretions in the nasopharynx and the middle ear from a tympanic membrane perforation. A purulent exudate is usually present in the middle ear, resulting in a conductive hearing loss.

Clinical Manifestations

Symptoms of otitis media vary with the severity of the infection. The condition, usually unilateral in adults, may be accompanied by otalgia. The pain is relieved after spontaneous perforation or therapeutic incision of the tympanic membrane. Other symptoms may include drainage from the ear, fever, and hearing loss. [Table 59-3](#) differentiates acute otitis externa from AOM. Risk factors for AOM include younger age, chronic upper respiratory infections, medical conditions that predispose the patient to ear infections (e.g., Down syndrome, cystic fibrosis, cleft palate), and chronic exposure to secondhand cigarette smoke.

Medical Management

The outcome of AOM depends on the efficacy of therapy (the prescribed dose of an oral antibiotic and the duration of therapy), the virulence of the bacteria, and the physical status of the patient. With early and appropriate broad-spectrum antibiotic therapy, otitis media may resolve with no serious sequelae. If drainage occurs, an antibiotic otic preparation is usually prescribed. The condition may become subacute (lasting 2 weeks to 3 months) with persistent purulent discharge from the ear. Rarely does permanent hearing loss occur. Secondary complications involving the mastoid and other serious intracranial complications, such as meningitis or brain abscess, although rare, can occur.

TABLE 59-3 Clinical Features of Otitis

Feature	Acute Otitis Externa	Acute Otitis Media
Otorrhea	May or may not be present	Present if tympanic membrane perforates; discharge is profuse
Otalgia	Persistent; may awaken patient at night	Relieved if tympanic membrane ruptures
Aural tenderness	Present on palpation of auricle	Usually absent
Systemic symptoms	Absent	Fever, upper respiratory infection, rhinitis
Edema of external auditory canal	Present	Absent
Tympanic membrane	May appear normal	Erythema, bulging, may be perforated
Hearing loss	Conductive type	Conductive type

Adapted from Weber, J., & Kelley, J. (2018). *Health assessment in nursing* (6th ed.). Philadelphia, PA: Wolters Kluwer.

Surgical Management

A **myringotomy** (i.e., tympanotomy) is an incision in the tympanic membrane. The tympanic membrane is numbed with a local anesthetic agent such as

phenol or by iontophoresis (i.e., in which electrical current flows through a lidocaine and epinephrine solution to numb the ear canal and tympanic membrane). The procedure is painless and takes less than 15 minutes. Under microscopic guidance, an incision is made through the tympanic membrane to relieve pressure and to drain serous or purulent fluid from the middle ear.

Normally, this procedure is unnecessary for treating AOM, but it may be performed if pain persists. Myringotomy also allows the drainage to be analyzed (by culture and sensitivity testing) so that the infecting organism can be identified and appropriate antibiotic therapy prescribed. The incision heals within 24 to 72 hours.

If AOM recurs and there is no contraindication, a ventilating, or pressure-equalizing, tube may be inserted. The ventilating tube, which temporarily takes the place of the eustachian tube in equalizing pressure, is retained for 6 to 18 months. The ventilating tube is then extruded with normal skin migration of the tympanic membrane, with the hole healing in nearly every case. Ventilating tubes are used to treat recurrent episodes of AOM.

Serous Otitis Media

Middle ear effusion, or serous otitis media, involves the presence of fluid, without evidence of active infection, in the middle ear. In theory, this fluid results from a negative pressure in the middle ear caused by eustachian tube obstruction. When this condition occurs in adults, an underlying cause for the eustachian tube dysfunction must be sought. Middle ear effusion is frequently seen in patients after radiation therapy or barotrauma and in patients with eustachian tube dysfunction from a concurrent upper respiratory infection or allergy. Barotrauma results from sudden pressure changes in the middle ear caused by changes in barometric pressure, as in scuba diving or airplane descent. A carcinoma (e.g., nasopharyngeal cancer) obstructing the eustachian tube should be ruled out in adults with persistent unilateral serous otitis media.

Clinical Manifestations

Patients may complain of hearing loss, fullness in the ear or a sensation of congestion, or popping and crackling noises that occur as the eustachian tube attempts to open. The tympanic membrane appears dull on otoscopy, and air bubbles may be visualized in the middle ear. Usually, the audiogram shows a conductive hearing loss.

Management

Serous otitis media need not be treated medically unless infection (i.e., AOM) occurs. If the hearing loss associated with middle ear effusion is significant, a myringotomy can be performed, and a tube may be placed to keep the middle ear ventilated. Corticosteroids in small doses may decrease the edema of the eustachian tube in cases of barotrauma. Decongestants have not proved to be effective. A Valsalva maneuver, which forcibly opens the eustachian tube by increasing nasopharyngeal pressure, may be cautiously performed; this maneuver may cause worsening pain or perforation of the tympanic membrane.

Chronic Otitis Media

Chronic otitis media is recurrent AOM that causes irreversible tissue pathology. Chronic infections of the middle ear damage the tympanic membrane, destroy the ossicles, and involve the mastoid but are rare in developed countries.

Clinical Manifestations

Symptoms may be minimal, with varying degrees of hearing loss and a persistent or intermittent, foul-smelling otorrhea. Pain is not usually experienced, except in cases of acute mastoiditis, when the postauricular area is tender and may be erythematous and edematous. Otoscopic examination may show a perforation, and cholesteatoma can be identified as a white mass behind the tympanic membrane or coming through to the external canal from a perforation.

Cholesteatoma is a cystlike lesion of the external layer of the eardrum into the middle ear. It is generally caused by a chronic retraction pocket of the tympanic membrane, creating a persistently high negative pressure of the middle ear. The skin forms a sac that fills with degenerated skin and sebaceous materials. The sac can attach to the structures of the middle ear, mastoid, or both.

Chronic otitis media can cause chronic mastoiditis and lead to the formation of cholesteatoma. The location will dictate the type of surgery to be performed. If untreated, cholesteatoma will continue to enlarge, possibly causing damage to the facial nerve and horizontal canal and destruction of other surrounding structures.

Cholesteatomas are cystlike lesions of the middle ear (Norris, 2019). They usually do not cause pain; however, if treatment or surgery is delayed, they may burst or destroy the mastoid bone. Cholesteatomas found in older adult patients generally develop in the external canal.

Cholesteatomas may be asymptomatic, or they may cause hearing loss, facial pain and paralysis, tinnitus, or vertigo. Audiometric tests often show a conductive or mixed hearing loss. Based on presenting symptoms, diagnosis may be made by visual examination or by computed tomography (CT) or MRI scan. Therapy includes treatment of the acute infection and surgical removal of the mass to restore hearing.

Medical Management



Local treatment for chronic otitis media consists of careful suctioning of the ear under otoscopic guidance. Instillation of antibiotic drops or application of antibiotic powder is used to treat purulent discharge. Systemic antibiotic agents are prescribed only in cases of acute infection.

Surgical Management

Surgical procedures, including tympanoplasty, ossiculoplasty, and mastoidectomy, are used if medical treatments are ineffective.

Tympanoplasty

The most common surgical procedure for chronic otitis media is **tympanoplasty**, or surgical reconstruction of the tympanic membrane. Reconstruction of the ossicles may also be required. The purposes of a tympanoplasty are to reestablish middle ear function, close the perforation, prevent recurrent infection, and improve hearing.

There are five types of tympanoplasties. The simplest surgical procedure, type I (myringoplasty), is designed to close a perforation in the tympanic membrane. The other procedures, types II through V, involve more extensive repair of middle ear structures. The structures and the degree of involvement can differ, but all tympanoplasty procedures include restoring the continuity of the sound conduction mechanism.

Tympanoplasty is performed through the external auditory canal with a transcanal approach or through a postauricular incision. The contents of the middle ear are carefully inspected, and the ossicular chain (malleus and incus unit) is evaluated. Ossicular interruption is most common in chronic otitis media, but problems of reconstruction can also occur with malformations of the middle ear and ossicular dislocations due to head injuries. Dramatic improvement in hearing can result from closure of a perforation and reestablishment of the ossicles. Surgery is usually performed in an outpatient facility under moderate sedation or general anesthesia.

Ossiculoplasty

Ossiculoplasty is the surgical reconstruction of the middle ear bones to restore hearing. Prostheses made of materials such as Teflon, stainless steel, and hydroxyapatite are used to reconnect the ossicles, thereby reestablishing the sound conduction mechanism. However, the greater the damage, the lower the success rate for restoring normal hearing.

Mastoidectomy

The objectives of mastoid surgery are to remove the cholesteatoma, gain access to diseased structures, and create a dry (noninfected) and healthy ear. If possible, the ossicles are reconstructed during the initial surgical procedure. Occasionally, extensive disease or damage dictates that this be performed as part of a two-stage operation.

A mastoidectomy is usually performed through a postauricular incision. Infection is eliminated by removing the mastoid air cells. A second mastoidectomy may be necessary to check for recurrent or residual cholesteatoma. The hearing mechanism may be reconstructed at this time. The success rate for correcting this conductive hearing loss is approximately 75%. Surgery is usually performed in an outpatient setting. The patient has a mastoid pressure dressing, which can be removed 24 to 48 hours after surgery. Although infrequently injured, the facial nerve, which runs through the middle ear and mastoid, is at some risk for injury during mastoid surgery. As the patient awakens from anesthesia, any evidence of facial paresis should be reported to the primary provider.

NURSING PROCESS

The Patient Undergoing Mastoid Surgery

Although several otologic surgical procedures are performed under moderate sedation, mastoid surgery is performed using general anesthesia.

Assessment

The health history includes a complete description of the ear disorder, including infection, otalgia, otorrhea, hearing loss, and vertigo. Data are collected about the duration and intensity of the disorder, its causes, and previous treatments. Information is obtained about other health problems and all medications that the patient is taking. Medication allergies and family history of ear disease also should be obtained.

Physical assessment addresses erythema, edema, otorrhea, lesions, and characteristics such as odor and color of discharge. The results of the audiogram are reviewed.

Nursing Diagnoses

Based on the assessment data, major nursing diagnoses may include the following:

- Anxiety associated with surgical procedure, potential loss of hearing, potential taste disturbance, and potential loss of facial movement
- Acute pain associated with mastoid surgery
- Risk for infection associated with mastoidectomy; placement of grafts, prostheses, and electrodes; and surgical trauma to surrounding tissues and structures
- Impaired verbal communication associated with ear disorder, surgery, or packing
- Risk for injury associated with impaired balance or vertigo during the immediate postoperative period, dislodgment of the graft or prosthesis, or injury to facial nerve (cranial nerve VII) and chorda tympani nerve
- Lack of knowledge about mastoid disease, surgical procedure, and postoperative care and expectations

Planning and Goals

Major goals of caring for a patient undergoing mastoidectomy include reduction of anxiety, freedom from pain and discomfort, prevention of infection, stable or improved hearing and communication, absence of vertigo and related injury, and increased knowledge regarding the disease, surgical procedure, and postoperative care.

Nursing Interventions

REDUCING ANXIETY

The nurse reinforces the information discussed by the otologic surgeon with the patient, including anesthesia, the location of the incision (postauricular), and expected surgical results (e.g., hearing, balance, taste, facial movement). The patient also is encouraged to discuss any anxieties and concerns about the surgery.

RELIEVING PAIN

Although most patients complain very little about incisional pain after mastoid surgery, they do have some ear discomfort. Aural fullness or pressure after surgery is caused by residual blood or fluid in the middle ear. The prescribed analgesic medication may be taken for the first 24 hours after surgery and then only as needed.

A wick or external auditory canal packing is used if tympanoplasty was performed at the time of the mastoidectomy. For the next 2 to 3 weeks after surgery, the patient may experience sharp, shooting pains intermittently as the eustachian tube opens and allows air to enter the middle ear. Constant, throbbing pain accompanied by fever may indicate infection and should be reported to the primary provider.

PREVENTING INFECTION

Measures are initiated to prevent infection in the operated ear. The external auditory canal wick, or packing, may be impregnated with an antibiotic solution before instillation. Prophylactic antibiotic agents are given as prescribed, and the patient is instructed to prevent water from entering the external auditory canal for 6 weeks. A cotton ball or lamb's wool covered with a water-insoluble substance (e.g., petroleum jelly) and placed loosely in the ear canal usually prevents water from entering the ear canal and should be used when the patient showers or washes their hair, or in any situations in which water may enter the ear canal. The postauricular incision should be kept dry for the first 2 days. Signs of infection such as an elevated temperature and purulent drainage should be reported. Some serosanguineous drainage from the external auditory canal is normal after surgery.

IMPROVING HEARING AND COMMUNICATION

Hearing in the operated ear may be reduced for several weeks because of edema, accumulation of blood and tissue fluid in the middle ear, and dressings or packing. Measures are initiated to improve hearing and communication, such as reducing environmental noise, facing the patient when speaking, speaking clearly and distinctly without shouting, providing good lighting, and using nonverbal clues (e.g., facial expression, pointing, gestures), writing, picture boards, electronic tablets, and other forms of communication. Family members or significant others are instructed about

effective ways to communicate with the patient. If the patient uses assistive hearing devices, one can be used in the unaffected ear.

PREVENTING INJURY

Vertigo may occur after mastoid surgery if the semicircular canals or other areas of the inner ear are traumatized. Antiemetic or antivertiginous medications (e.g., antihistamines) can be prescribed if a balance disturbance or vertigo occurs. Safety measures such as assisted ambulation are implemented to prevent falls and injury. The patient is instructed to avoid heavy lifting, straining, exertion, and nose blowing for 2 to 3 weeks after surgery to prevent dislodging the tympanic membrane graft or ossicular prosthesis.

Facial nerve injury is a potential, although rare, complication of mastoid surgery. The patient is instructed to report immediately any evidence of facial nerve (cranial nerve VII) weakness, such as drooping of the mouth on the operated side, slurred speech, decreased sensation, and difficulty swallowing. A more frequent occurrence is a temporary disturbance in the chorda tympani nerve, which is a small branch of the facial nerve that runs through the middle ear. Patients experience a taste disturbance and dry mouth on the side of surgery for several months until the nerve regenerates.

Chart 59-7



PATIENT EDUCATION

Self-Care After Middle Ear or Mastoid Surgery

Postoperative instructions for patients who have had middle ear and mastoid surgery may vary among otolaryngologists. The nurse instructs the patient in the following general guidelines:

- Take antibiotics and other medications as prescribed.
- Avoid nose blowing for 2 to 3 weeks after surgery.
- Sneeze and cough with the mouth open for a few weeks after surgery.
- Avoid heavy lifting (>10 lb), straining, and bending over for a few weeks after surgery.
- Be aware that popping and crackling sensations in the operative ear are normal for approximately 3 to 5 weeks after surgery.
- Note that temporary hearing loss is normal in the operative ear due to fluid, blood, or packing in the ear.
- Report excessive or purulent ear drainage to the physician.
- Avoid getting water in the operative ear for 2 weeks after surgery. You may shampoo the hair 2 to 3 days postoperatively if the ear is protected from water by saturating a cotton ball with petroleum jelly (or some other water-insoluble substance) and loosely placing it in the ear. If the postauricular suture line becomes wet, pat (not rub) the area and cover it with a thin layer of antibiotic ointment.

PROMOTING HOME, COMMUNITY-BASED, AND TRANSITIONAL CARE



Educating Patients About Self-Care. Patients require education about medication therapy, such as analgesic and antivertiginous agents (e.g., antihistamines) prescribed for balance disturbance. Education includes information about the expected effects and potential side effects of the medication. Patients also need instruction about any activity restrictions. Possible complications such as infection, facial nerve weakness, or taste disturbances, including the signs and symptoms to report immediately, are included (see [Chart 59-7](#)).

Continuing and Transitional Care. Some patients, particularly older adult patients, who have had mastoid surgery may require the services of a home, community-based, or transitional care nurse for a few days after returning home. However, most people find that assistance from a family member or a friend is sufficient. The caregiver and patient are cautioned that the patient may experience some vertigo and will therefore require help with ambulation to avoid falling. Any symptoms of complications are to be reported promptly to the primary provider. The importance of scheduling and keeping follow-up appointments is also stressed.

Evaluation

Expected patient outcomes may include:

1. Demonstrates reduced anxiety about surgical procedure
 - a. Verbalizes and exhibits less stress, tension, and irritability
 - b. Verbalizes acceptance of results of surgery and adjustment to possible hearing loss
2. Remains free of discomfort or pain
 - a. Exhibits no facial grimacing, moaning, or crying, and reports absence of pain
 - b. Uses analgesic agents appropriately
3. Demonstrates no signs or symptoms of infection
 - a. Has normal vital signs, including temperature
 - b. Demonstrates absence of purulent drainage from the external auditory canal
 - c. Describes method for preventing water from contaminating packing
4. Exhibits signs that communication and hearing have stabilized or improved
 - a. Describes surgical goal for hearing and judges whether the goal has been met
 - b. Verbalizes that hearing has improved
5. Remains free of injury and trauma
 - a. Reports absence of vertigo or balance disturbance
 - b. Experiences no injury or fall
 - c. Avoids activities that can cause dislodgement of graft or prosthesis
 - d. Reports no taste disturbance, mouth dryness, or facial weakness
6. Verbalizes the reasons for and methods of care and treatment
 - a. Discusses the discharge plan formulated with the nurse with regard to rest periods, medication, and activities permitted and restricted
 - b. Lists symptoms that should be reported to the primary provider
 - c. Keeps follow-up appointments

Otosclerosis

Otosclerosis involves the stapes and is thought to result from the formation of new, abnormal spongy bone, especially around the oval window, with resulting fixation of the stapes (Norris, 2019). The efficient transmission of sound is prevented because the stapes cannot vibrate and carry the sound as conducted from the malleus and incus to the inner ear. Otosclerosis is more common in

women, is a familial condition, and can progress to complete deafness (Eliopoulos, 2018; Norris, 2019).

Clinical Manifestations

Otosclerosis may involve one or both ears and manifests as a progressive conductive or mixed hearing loss. The patient may or may not complain of tinnitus. Otoscopic examination usually reveals a normal tympanic membrane. Bone conduction is better than air conduction on Rinne testing. The audiogram confirms conductive hearing loss or mixed loss, especially in the low frequencies.

Medical Management

The management of otosclerosis can be surgical or medical. Amplification with a hearing aid may help (Norris, 2019).

Surgical Management

One of two surgical procedures may be performed: the stapedectomy or the stapedotomy. A stapedectomy involves removing the stapes superstructure and part of the footplate and inserting a tissue graft and a suitable prosthesis (see Fig. 59-6). In a stapedotomy, the surgeon drills a small hole into the stapes, instead of removing it, to hold a prosthesis. In both procedures, the prosthesis bridges the gap between the incus and the inner ear, providing better sound conduction. The majority of patients experience resolution of conductive hearing loss following stapes surgery. The use of sodium fluoride in the postoperative period increases the success rate and hearing ability postoperatively (Norris, 2019). Balance disturbance or true vertigo may occur during the postoperative period for several days. Long-term balance disorders are rare.

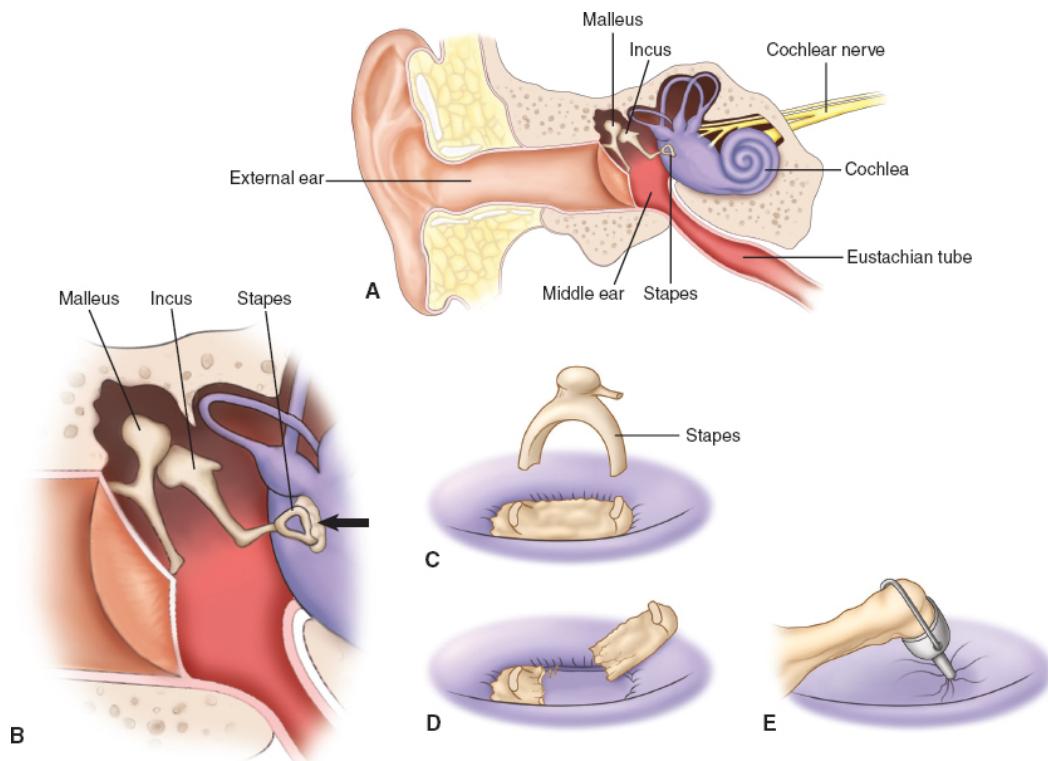


Figure 59-6 • Stapedectomy for otosclerosis. **A.** Normal anatomy. **B.** Arrow points to sclerotic process at the foot of the stapes. **C.** Stapes broken away surgically from its diseased base. The hole in the footplate provides an area where an instrument can grasp the plate. **D.** The footplate is removed from its base. Some otosclerotic tissue may remain, and tissue is placed over it. **E.** Robinson stainless steel prosthesis in position.

Middle Ear Masses

Other than cholesteatoma, masses in the middle ear are rare. Glomus tympanicum is a tumor that arises from Jacobson nerve (in the temporal bone of the skull) and remains limited to the middle ear. On otoscopy, a red blemish on or behind the tympanic membrane is seen. Glomus jugulare tumors are rarely malignant; however, because of their location, treatment may be necessary to relieve symptoms. The treatment is surgical excision, except in patients who are poor surgical candidates, in whom radiation therapy is used.

A facial nerve neuroma is a tumor on cranial nerve VII. These types of tumors are usually not visible on otoscopic examination but are suspected when a patient presents with a facial nerve paresis. X-ray evaluation is used to identify the site of the tumor along the facial nerve. The treatment is surgical removal.

CONDITIONS OF THE INNER EAR

Disorders of balance are common and dizziness may increase the risk of falls (NIDCD, 2018). The term **dizziness** is used frequently by patients and health care providers to describe any altered sensation of orientation in space and is more commonly referred to as lightheadedness (Weber & Kelley, 2018). **Vertigo** is the misperception or illusion of motion of the person or the surroundings. Most patients with vertigo describe a spinning sensation or say they feel as though objects are moving around them. Ataxia is a failure of muscular coordination and may be present in patients with vestibular disease. Syncope, fainting, and loss of consciousness are not forms of vertigo and usually indicate disease in the cardiovascular system.

Nystagmus is an involuntary rhythmic movement of the eyes. Nystagmus occurs normally when a person watches a rapidly moving object (e.g., through the side window of a moving car or train). However, pathologically, it is an ocular disorder associated with vestibular dysfunction. Nystagmus can be horizontal, vertical, or rotary and can be caused by a disorder in the central or peripheral nervous system.

Motion Sickness

Motion sickness is a disturbance of equilibrium caused by a conflict in motion receptor stimuli. For example, it can occur aboard a ship, in a car, while riding on a merry-go-round or swing, or even while viewing virtual motion on a large screen (Brainard & Gresham, 2014).

Clinical Manifestations

The syndrome manifests itself in sweating, pallor, nausea, and vomiting caused by vestibular overstimulation. These manifestations may persist for several hours after the stimulation stops.

Management

Over-the-counter antihistamines such as dimenhydrinate or meclizine may provide some relief of nausea and vomiting by blocking the conduction of the vestibular pathway of the inner ear. Anticholinergic medications, such as scopolamine patches, may also be effective because they antagonize the histamine response. These must be applied 4 hours before exposure to motion and replaced every 3 days (Brainard & Gresham, 2014). Side effects such as dry mouth and drowsiness may occur. Potentially hazardous activities such as

driving a car or operating heavy machinery should be avoided if drowsiness occurs.

Ménière's Disease



Ménière's disease is an abnormality in inner ear fluid balance caused by a malabsorption in the endolymphatic sac or a blockage in the endolymphatic duct (NIDCD, 2017b). **Endolymphatic hydrops** (dilation of the endolymphatic space) frequently occurs, causing either increased pressure in the system or rupture of the inner ear membrane, producing symptoms of Ménière's disease (van Steekelenburg, van Weijnen, de Pont, et al., 2020).

Ménière's disease affects 10 to 12 of 1000 people in the United States. It is estimated that there are 615,000 cases in the United States, with approximately 45,500 new cases diagnosed annually (NIDCD, 2017b). More common in adults, onset is generally seen when adults reach their 40s, with symptoms usually beginning between the ages of 20 and 60 years. Ménière's disease appears to be equally common in men and women, and is usually bilateral (Norris, 2019).

Clinical Manifestations

Ménière's disease is characterized by a triad of symptoms: episodic vertigo, tinnitus, and fluctuating sensorineural hearing loss (Luryi, Morse, & Michaelides, 2019). It may also include a feeling of pressure or fullness in the ear and incapacitating vertigo, often accompanied by nausea and vomiting (NIDCD, 2017b). These symptoms range in severity from a minor nuisance to extreme disability, especially if the attacks of vertigo are severe. At the onset of the disease, usually only one or two of the symptoms are manifested.

The disease may be characterized into two subsets: cochlear and vestibular. Cochlear Ménière's disease is recognized as a fluctuating, progressive sensorineural hearing loss associated with tinnitus and aural pressure in the absence of vestibular symptoms or findings. Vestibular Ménière's disease is characterized as the occurrence of episodic vertigo associated with aural pressure but no cochlear symptoms. Patients may experience either cochlear or vestibular disease symptoms at first; however, eventually all of these symptoms develop.

Assessment and Diagnostic Findings

Vertigo is usually the most troublesome complaint related to Ménière's disease. A careful history is taken to determine the frequency, duration,

severity, and character of the vertigo attacks. Vertigo may last minutes to hours, possibly accompanied by nausea or vomiting. Diaphoresis and a persistent feeling of imbalance or disequilibrium may awaken patients at night. Some patients report that these feelings last for days. However, they usually feel well between attacks. Hearing loss may fluctuate, with tinnitus and aural pressure waxing and waning with changes in hearing. These feelings may occur during or before attacks, or they may be constant.

Physical examination findings are usually normal, with the exception of those of cranial nerve VIII. Sounds from a tuning fork (Weber test) may lateralize to the ear opposite the hearing loss, the one affected with Ménière's disease. An audiogram typically reveals a sensorineural hearing loss in the affected ear. This can be in the form of a pattern that looks like a hill or mountain. A sensorineural loss in the low frequencies occurs as the disease progresses. The electronystagmogram may be normal or may show reduced vestibular response.

Medical Management

Most patients with Ménière's disease can be successfully treated with diet and medication. Many patients can control their symptoms by adhering to a low-sodium (1000 to 1500 mg/day or less) diet. [Chart 59-8](#) describes dietary guidelines that may be useful in Ménière's disease. The amount of sodium is one of many factors that regulate the balance of fluid within the body. Sodium and fluid retention disrupts the delicate balance between endolymph and perilymph in the inner ear. Psychological evaluation and cognitive therapy may be indicated if a patient is anxious, uncertain, fearful, or depressed (NIDCD, 2017b).

Chart 59-8



PATIENT EDUCATION

Dietary Guidelines for Patients with Ménière's Disease

The nurse instructs the patient to:

- limit foods high in salt or sugar. Be aware of foods with hidden salts and sugars.
- eat meals and snacks at regular intervals to stay hydrated. Missing meals or snacks may alter the fluid level in the inner ear.
- eat fresh fruits, vegetables, and whole grains. Limit the amount of canned, frozen, or processed foods with high sodium content.
- drink plenty of fluids daily. Water, milk, and low-sugar fruit juices are recommended. Limit intake of coffee, tea, and soft drinks. Avoid caffeine because of its diuretic effect.
- limit alcohol intake. Alcohol may change the volume and concentration of the inner ear fluid and may worsen symptoms.
- avoid monosodium glutamate (MSG), which may increase symptoms.
- pay attention to the intake of foods containing potassium (e.g., bananas, tomatoes, oranges) if taking a diuretic that causes potassium loss.
- avoid aspirin and aspirin-containing medications. Aspirin may increase tinnitus and dizziness.

Adapted from National Institute on Deafness and Other Communication Disorders (NIDCD). (2017b). Ménière's disease. Retrieved on 2/22/2020 at: www.nidcd.nih.gov/health/menieres-disease

Pharmacologic Therapy

Pharmacologic therapy for Ménière's disease consists of antihistamines, such as meclizine, which shortens the attack (NIDCD, 2017b). Tranquilizers such as diazepam may be used in acute instances to help control vertigo. Antiemetic agents such as promethazine suppositories help control the nausea and vomiting and the vertigo because of their antihistamine effect. Diuretic therapy (e.g., hydrochlorothiazide, triamterene, spironolactone) may relieve symptoms by lowering the pressure in the endolymphatic system (Norris, 2019). Intratympanic injection of gentamicin is used to cause ablation of the vestibular hair cells; however, the risk of significant hearing loss is high (NIDCD, 2017b).

Surgical Management

Although most patients respond well to conservative therapy, some continue to have disabling attacks of vertigo. If these attacks reduce their quality of life, patients may elect to undergo surgery for relief. Surgical procedures include endolymphatic sac procedures and vestibular nerve section (NIDCD, 2017b). However, hearing loss, tinnitus, and aural fullness may continue, because the

surgical treatment of Ménière's disease is aimed at eliminating the attacks of vertigo.

Endolymphatic Sac Decompression

Endolymphatic sac decompression, or shunting, theoretically equalizes the pressure in the endolymphatic space. A shunt or drain is inserted in the endolymphatic sac through a postauricular incision. This procedure is favored by many otolaryngologists as a first-line surgical approach to treat the vertigo of Ménière's disease because it is relatively simple and safe and can be performed on an outpatient basis.

Vestibular Nerve Sectioning

Vestibular nerve sectioning provides the greatest success rate (approximately 98%) in eliminating the attacks of vertigo. It can be performed by a translabyrinthine approach (i.e., through the hearing mechanism) or in a manner that can conserve hearing (i.e., suboccipital or middle cranial fossa), depending on the degree of hearing loss. Most patients with incapacitating Ménière's disease have little or no effective hearing. Cutting the nerve prevents the brain from receiving input from the semicircular canals. This procedure may require a brief hospital stay. A plan of nursing care for the patient with vertigo is presented in [Chart 59-9](#).

Benign Paroxysmal Positional Vertigo

Benign paroxysmal positional vertigo is a brief period of incapacitating vertigo that occurs when the position of the patient's head is changed with respect to gravity, typically by placing the head back with the affected ear turned down (Muñoz, Moreno, Balboa, et al., 2019; NIDCD, 2017b). The onset is sudden and followed by a predisposition for positional vertigo, usually for hours to weeks but occasionally for months or years.

Benign paroxysmal positional vertigo is thought to be due to the disruption of debris within the semicircular canal. This debris is formed from small crystals of calcium carbonate from the inner ear structure (the utricle). This is frequently stimulated by head trauma, infection, or other events. In severe cases, vertigo may easily be induced by any head movement. The vertigo is usually accompanied by nausea and vomiting; however, hearing loss does not generally occur.

Chart 59-9



PLAN OF NURSING CARE

Care of the Patient with Vertigo

NURSING DIAGNOSIS: Risk for fall-related injury associated with impaired balance, gait disturbance, and vertigo

GOAL: Remains free of physical trauma associated with imbalance and falls

Nursing Interventions	Rationale	Expected Outcomes
<ol style="list-style-type: none"> 1. Assess for balance disturbance or vertigo, including history, onset, description of attacks, duration, frequency, and any associated ear symptoms (hearing loss, tinnitus, aural fullness). 2. Perform examination for nystagmus, positive Romberg, and inability to perform tandem Romberg. 3. Assess extent of disability (i.e., visual acuity and proprioceptive deficits) in relation to activities of daily living. 4. Instruct or reinforce vestibular/balance therapy as prescribed. 5. Administer, or educate about administration of, antivertiginous medications or vestibular sedation medication; instruct patient about side effects. 6. Encourage patient to sit down and restrict activity when dizzy. 7. Place pillow on each side of head to restrict movement. 8. Assist patient in identifying aura that 	<ol style="list-style-type: none"> 1. Health history provides basis for interventions. 2. Peripheral vestibular disorders cause these signs and symptoms. 3. Extent of disability indicates risk of falling. Balance depends on visual, vestibular, and proprioceptive systems. 4. Exercises hasten labyrinthine compensation, which may decrease vertigo and gait disturbance. 5. Alleviates acute symptoms of vertigo. 6. Decreases possibility of falling and injury. 7. Movement aggravates vertigo. 8. Recognition of aura may trigger the need to take medication before an attack occurs, 	<ul style="list-style-type: none"> • Experiences no physical trauma due to balance disturbance • Visual and proprioceptive risks identified • Activity level increases • Performs exercises as prescribed • Takes prescribed medications appropriately • Assumes safe position when vertigo is present • Keeps head still when vertigo is present • Identifies a characteristic fullness or sense of pressure in the ear as occurring before an attack occurs • Reports measures that help reduce vertigo

suggests an impending attack.	thereby minimizing the severity of effects.	<ul style="list-style-type: none"> • Home environment free of hazards • Has adapted home environment or uses rehabilitative devices to reduce risk of falling
9. Recommend that the patient keep eyes open and stare straight ahead when lying down and experiencing vertigo.	9. Sensation of vertigo decreases and motion decelerates if eyes are kept in a fixed position.	
10. Help identify hazards or use rehabilitative devices in home environment.	10. Adaptation of home environment can reduce risk of falls.	

NURSING DIAGNOSIS: Risk for negative quality of life associated with unpredictability of vertigo

GOAL: Modifies lifestyle to decrease disability and exert maximum control and independence within limits posed by chronic vertigo

Nursing Interventions	Rationale	Expected Outcomes
<ol style="list-style-type: none"> 1. Encourage patient to identify personal strengths and roles that can still be fulfilled. 2. Provide information about vertigo and what to expect. 3. Include family and significant others in rehabilitative process. 4. Encourage patient to maintain sense of control by making decisions and assuming more responsibility for care. 	<ol style="list-style-type: none"> 1. Maximizes sense of regaining control and independence. 2. Reduces fear and anxiety. 3. Perceived beliefs of significant others are important for patient's adherence to medical regimen. 4. Reinforces positive psychological and social outcomes. 	<ul style="list-style-type: none"> • Exerts maximum control of environment and independence within limits imposed by vertigo • Is informed about condition • Family and significant others are included in rehabilitation process • Uses strengths and potentials to engage in the most independent

and
constructive
lifestyle

NURSING DIAGNOSIS: Risk for hypovolaemia associated with increased fluid output, altered intake, and medications

GOAL: Maintains normal fluid and electrolyte balance

Nursing Interventions	Rationale	Expected Outcomes
<ol style="list-style-type: none"> Assess, or have patient assess, intake and output (including emesis, liquid stools, urine, and diaphoresis). Monitor laboratory values of electrolytes. Assess indicators of dehydration, including blood pressure (orthostasis), pulse, skin turgor, mucous membranes, and level of consciousness. Encourage oral fluids as tolerated; discourage beverages containing caffeine (a vestibular stimulant). Administer, or educate about administration of, antiemetic medications as prescribed and needed. Educate patient about side effects. 	<ol style="list-style-type: none"> Accurate records provide basis for fluid replacement. Prompt recognition of dehydration allows early intervention. Oral replacement is begun as soon as possible to replace losses. Antiemetic medications reduce nausea and vomiting, reducing fluid losses and improving oral intake. 	<ul style="list-style-type: none"> Laboratory values within normal limits Alert and oriented; vital signs within normal limits, skin turgor normal; electrolytes normal Mucous membranes are moist Vomiting has stopped; usual oral intake is resumed

NURSING DIAGNOSIS: Anxiety associated with threat of, or change in, health status and disability effects of vertigo

GOAL: Experiences less or no anxiety

Nursing Interventions	Rationale	Expected Outcomes
<ol style="list-style-type: none"> Assess level of anxiety. Help patient identify 	<ol style="list-style-type: none"> Guides therapeutic 	<ul style="list-style-type: none"> Fear and anxiety about

coping skills used successfully in the past.	interventions and participation in self-care. Past coping skills can relieve anxiety.	attacks of vertigo reduced or eliminated
2. Provide information about vertigo and its treatment.	2. Increased knowledge helps to decrease anxiety.	• Acquires knowledge and skills to deal with vertigo
3. Encourage patient to discuss anxieties and explore concerns about vertigo attacks.	3. Promotes awareness and understanding of relationship between anxiety level and behavior.	• Feels less tension, apprehension, and uncertainty
4. Educate patient about stress management techniques or make appropriate referral.	4. Improved stress management can reduce the frequency and severity of some vertiginous attacks.	• Uses stress management techniques when needed
5. Provide comfort measures and avoid stress-producing activities.	5. Stressful situations may exacerbate symptoms of the condition.	• Avoids upsetting encounters
6. Instruct patient in aspects of treatment regimen.	6. Patient knowledge helps to decrease anxiety.	• Repeats instructions given and verbalizes understanding of treatments

NURSING DIAGNOSIS: Impaired self feeding, impaired ability to perform hygiene, impaired ability to dress, and impaired self toileting associated with labyrinth dysfunction and episodes of vertigo

GOAL: Able to care for self

Nursing Interventions	Rationale	Expected Outcomes
1. Administer, or educate about administration of, antiemetic and other prescribed medications to relieve nausea and vomiting associated with vertigo.	1. Antiemetic and sedative-type medications depress stimuli in the cerebellum. 2. Spacing activities is important because episodes	• Carries out necessary functions during symptom-free periods and takes medications to

2. Encourage patient to perform self-care when free of vertigo.	of vertigo vary in occurrence.	relieve nausea, vomiting, or vertigo
3. Review diet with patient and caregivers. Offer fluids as necessary.	3. Sodium restriction helps improve an inner ear fluid imbalance in some patients, thereby decreasing vertigo. Fluids help prevent dehydration.	<ul style="list-style-type: none"> • Carries out daily activities • Accepts dietary plan and reports its effectiveness • Drinks fluids in sufficient amounts

NURSING DIAGNOSIS: Risk for powerlessness associated with illness regimen and feeling helpless in certain situations due to vertigo/balance disturbance

GOAL: Experiences increased sense of control over life and activities despite vertigo/balance disturbance

Nursing Interventions	Rationale	Expected Outcomes
<ol style="list-style-type: none"> 1. Assess patient's needs, values, attitudes, and readiness to initiate activities. 2. Provide opportunities for patient to express feelings about self and illness. 3. Help patient identify previous coping behaviors that were successful. 	<ol style="list-style-type: none"> 1. Involving patient in planning activities and care enhances potential for mastery. 2. Expressing feelings increases understanding of individual coping styles and defense mechanisms. 3. Awareness increases understanding of stressors that trigger feeling of powerlessness. Awareness of past successes enhances self-confidence. 	<ul style="list-style-type: none"> • Does not restrict activities unnecessarily due to vertigo • Verbalizes positive feelings about own ability to achieve a sense of power and control • Identifies previous successful coping behaviors

Bed rest is recommended for patients with acute symptoms. Repositioning techniques can be used to treat vertigo. The canalith repositioning procedure is commonly used (Bhattacharyya, Gubbels, Schwartz, et al., 2017). This noninvasive procedure, which involves quick movements of the body, rearranges the debris in the canal. The procedure is performed by placing the patient in a sitting position, turning the head to a 45-degree angle on the affected side, and then quickly moving the patient to the supine position. The procedure is safe, inexpensive, and easy to perform.

Patients with acute vertigo may be treated with meclizine for 1 to 2 weeks. After this time, the meclizine is stopped and the patient is reassessed. Patients who continue to have severe positional vertigo may be premedicated with prochlorperazine 1 hour before the canalith repositioning procedure is performed.

Vestibular rehabilitation can be used in the management of vestibular disorders. This strategy promotes active use of the vestibular system through an interdisciplinary team approach, including medical and nursing care, stress management, biofeedback, vocational rehabilitation, and physical therapy. A physical therapist prescribes balance exercises that help the brain compensate for the impairment to the balance system.

Tinnitus

Tinnitus may be a symptom of an underlying disorder of the ear that is associated with hearing loss or it may be benign. This condition affects approximately 25 million people in the United States and is most prevalent in adults (NIDCD, 2017c). The severity of tinnitus may range from mild to severe. Patients describe tinnitus as a roaring, buzzing, or hissing sound in one or both ears. Numerous factors may contribute to the development of tinnitus, including several ototoxic substances (see [Chart 59-10](#)). Underlying disorders that contribute to tinnitus may include cardiovascular disease, thyroid disease, hyperlipidemia, vitamin B₁₂ deficiency, psychological disorders (e.g., depression, anxiety), fibromyalgia, otologic disorders (Ménière's disease, acoustic neuroma), and neurologic disorders (head injury, multiple sclerosis).

A physical examination should be performed to determine the cause of tinnitus. Diagnostic testing determines if hearing loss is present. An audiograph speech discrimination test or a tympanogram may be used to help determine the cause. Some forms of tinnitus are irreversible; therefore, patients may need education and counseling about ways of adjusting to their treatment and dealing with tinnitus in the future.

Labyrinthitis

Labyrinthitis, an inflammation of the labyrinth of the inner ear, can be bacterial or viral in origin. Bacterial labyrinthitis is rare because of antibiotic therapy, but it sometimes occurs as a complication of otitis media. The infection can spread to the inner ear by penetrating the membranes of the oval or round windows. Viral labyrinthitis is a common diagnosis, but little is known about this disorder, which affects hearing and balance. The most common viral causes are mumps, rubella, rubeola, and influenza. Viral illnesses of the upper respiratory tract and herpetiform disorders of the facial and acoustic nerves (i.e., Ramsay Hunt syndrome) also cause labyrinthitis.

Chart 59-10

Select Ototoxic Substances

- **Aminoglycoside antibiotic agents:** amikacin, gentamicin, kanamycin, netilmicin, neomycin, streptomycin, tobramycin
- **Anti-inflammatory agents:** salicylates (aspirin), indomethacin
- **Antimalarial agents:** quinine, chloroquine
- **Chemicals:** alcohol, arsenic
- **Chemotherapeutic (antineoplastic) agents:** cisplatin, nitrogen mustard, carboplatin
- **Loop diuretic agents:** ethacrynic acid, furosemide, acetazolamide, torsemide, azosemide, ozolinone, indacrinone, piretanide
- **Metals:** gold, mercury, lead
- **Other antibiotic agents:** erythromycin, azithromycin, clarithromycin, minocycline, polymyxin B, vancomycin

Adapted from Mudd, P. (2019). Ototoxicity. *Medscape*. Retrieved on 3/7/2020 at: emedicine.medscape.com/article/857679-overview

Clinical Manifestations

Labyrinthitis is characterized by a sudden onset of incapacitating vertigo, usually with nausea and vomiting, various degrees of hearing loss, and possibly tinnitus. The first episode is usually the worst; subsequent attacks, which usually occur over a period of several weeks to months, are less severe.

Management

Treatment of bacterial labyrinthitis includes IV antibiotic therapy, fluid replacement, and administration of an antihistamine (e.g., meclizine) and antiemetic medications. Treatment of viral labyrinthitis is based on the patient's symptoms.

Ototoxicity

A variety of medications may have adverse effects on the cochlea, vestibular apparatus, or cranial nerve VIII. All but a few, such as aspirin and quinine, cause irreversible hearing loss. Aspirin toxicity can produce bilateral tinnitus. IV medications, especially the aminoglycosides, are a common cause of ototoxicity, because they destroy the hair cells in the organ of Corti (see [Chart 59-10](#)). Antineoplastic agents also cause hair cell death in the cochlea, which can lead to hearing loss (Mudd, 2019). These medications can be found in the body several months later; side effects are dose dependent, with higher doses causing increased ototoxicity. Therefore, hearing loss may occur at any time, even several months after the last dose of the medication was given.

To prevent loss of hearing or balance, patients receiving potentially ototoxic medications should be counseled about their side effects. These medications should be used with caution in individuals that are at high risk for complications, such as children, older adults, patients who are pregnant, patients with kidney or liver problems, and patients with current hearing disorders. Blood levels of the medications should be monitored, and patients receiving long-term IV antibiotics should be monitored with an audiogram twice each week during therapy.

Acoustic Neuroma

Acoustic neuromas, also referred to as vestibular schwannomas, are slow-growing, benign tumors of cranial nerve VIII, usually arising from the Schwann cells of the vestibular portion of the nerve. Acoustic tumors typically arise within the internal auditory canal and extend into the cerebellopontine angle to press on the brain stem, possibly destroying the vestibular nerve. Most acoustic neuromas are unilateral, except in von Recklinghausen disease (neurofibromatosis type 2), in which bilateral tumors occur (Norris, 2019).

Acoustic neuromas develop in one of every 100,000 people in the United States per year (NIDCD, 2017d). These neuromas account for 8% of all intracranial tumors and seem to occur with equal frequency in men and women at any age, although most occur during middle age (Carlson, Tveiten, Driscoll, et al., 2015).

Assessment and Diagnostic Findings

The most common assessment findings of patients with acoustic neuromas are unilateral tinnitus and hearing loss with or without vertigo or balance disturbance. It is important to identify asymmetry in audiovestibular test results so that further workup can be performed to rule out an acoustic neuroma. Although conflicting data exist, the only known risk factor for

acoustic neuroma is cell phone usage (Park, Vernick, & Ramakrishna, 2019). An MRI scan with a contrast agent (i.e., gadolinium or gadopentetate) is the imaging study of choice. If the patient is claustrophobic or cannot undergo an MRI scan for other reasons, or if the scan is unavailable, a CT scan with contrast dye is performed. However, MRI is more sensitive than CT in delineating a small tumor.

Management

Three options for managing an acoustic neuroma include: (1) surgical removal, (2) radiation, and (3) observation (Carlson et al., 2015; NIDCD, 2017d). Conservative treatment and routine monitoring are recommended for patients with tumors less than 1.5 cm and for those who are older. For patients who are at low risk, surgical removal of the acoustic tumor is the treatment of choice because these tumors do not respond well to radiation or chemotherapy. Because treatment of acoustic tumors crosses several specialties, the interdisciplinary treatment approach involves a neurologist and a neurosurgeon. The objective of the surgery is to remove the tumor while preserving facial nerve function. Most acoustic tumors have damaged the cochlear portion of cranial nerve VIII resulting in hearing loss. In these patients, surgery is performed using a translabyrinthine approach, and the hearing mechanism is destroyed. If hearing is still good before surgery, a suboccipital or middle cranial fossa approach to removing the tumor may be used. This procedure exposes the lateral third of the internal auditory canal and preserves hearing (Park et al., 2019).

Potential complications of surgery include facial nerve paralysis, cerebrospinal fluid leakage, meningitis, and cerebral edema. Death from acoustic neuroma surgery is rare (Park et al., 2019).

AURAL REHABILITATION

If hearing loss is permanent or cannot be treated by medical or surgical means or if the patient elects not to undergo surgery, aural rehabilitation may be beneficial. The purpose of aural rehabilitation is to maximize the communication skills of the person with hearing loss. Aural rehabilitation includes auditory training, speech reading, speech training, and the use of hearing aids and hearing guide dogs.

Auditory training emphasizes listening skills, so the person with hearing loss practices concentrating on the speaker. Speech reading (also known as lip reading) can help fill the gaps left by missed or misheard words. The goals of speech training are to conserve, develop, and prevent deterioration of current communication skills.

It is important to identify the type of hearing loss so that rehabilitative efforts can be appropriate for each individual. Surgical correction may be all that is necessary to treat and improve a conductive hearing loss by eliminating the cause of the hearing loss. Advances in hearing aid technology have greatly improved amplification for patients with sensorineural hearing loss.

Hearing Aids

A hearing aid is a device through which speech and environmental sounds are received by a microphone, converted to electrical signals, amplified, and reconverted to acoustic signals. Many aids available for sensorineural hearing loss depress the low frequencies, or tones, and enhance hearing for the high frequencies. A general guideline for assessing the patient's need for a hearing aid is a hearing loss exceeding 30 dB in the range of 500 to 2000 Hz in the better-hearing ear.

A hearing aid makes sounds louder, but it does not improve a patient's ability to discriminate words or understand speech. People who have low discrimination scores (i.e., 20%) on audiograms may derive little benefit from a hearing aid. Hearing aids amplify all sounds, including background noise, which may be particularly disturbing to the first-time wearer. Chart 59-11 identifies additional problems associated with hearing aid use. Computerized hearing aids are available to compensate for background noise or allow amplification at certain programmed frequencies rather than at all frequencies. Occasionally, depending on the type of hearing loss, binaural aids (i.e., one for each ear) may be indicated. Chart 59-12 provides tips for hearing aid care.

Chart 59-11

Hearing Aid Problems

Whistling Noise

- Loose ear mold
- Improperly made
- Improperly worn
- Worn out

Improper Aid Selection

- Too much power required in aid, with inadequate separation between microphone and receiver
- Open mold used inappropriately
- Inadequate amplification
- Dead batteries
- Cerumen in ear
- Cerumen or other material in mold
- Wires or tubing disconnected from aid
- Aid turned off or volume too low
- Improper mold
- Improper aid for degree of loss

Pain From Mold

- Improperly fitted mold
- Ear skin or cartilage infection
- Middle ear infection
- Ear tumor
- Unrelated conditions of the temporomandibular joint, throat, or larynx

Chart 59-12



PATIENT EDUCATION

Tips for Hearing Aid Care

The nurse instructs the patient how to clean a hearing aid, check for malfunctions, and recognize complications:

Cleaning

- Keep in mind that the ear mold is the only part of the hearing aid that may be washed frequently.
- Wash the ear mold daily with soap and water.
- Allow the ear mold to dry completely before it is snapped into the receiver.
- Clean the cannula with a small pipe cleaner-like device.
- Note that properly caring for the ear device and keeping the ear canal clean and dry can prevent complications.

Checking for Malfunctions

- Be aware that inadequate amplification, a whistling noise, or pain from the mold can occur when a hearing aid is not functioning properly.
- Check for malfunctions:
 - Is the switch on properly?
 - Are the batteries charged and positioned correctly?
 - Is the ear mold clogged with cerumen? Ear wax can be easily removed with pin, pipe cleaner, or wax loop.
- Notify the hearing aid dealer if the hearing aid is still not working properly.
- Keep in mind that if the unit requires extended time for repair, the dealer may lend you a hearing aid until the repair can be accomplished.

Recognizing Complications

- Understand that common medical complications include external otitis media and pressure injury in the external auditory canal. Signs and symptoms of these infections include painful ear, especially when the external ear is touched; canal swelling; redness; difficulty hearing; pain radiating to the jaw area; and fever.
- Notify your health care provider for evaluation if any of these symptoms are present. You may need medication to treat infection, pain, or both.

A hearing aid should be fitted according to the patient's needs (e.g., type of hearing loss, manual dexterity, and preference), rather than brand name, by a certified audiologist licensed to dispense hearing aids. Many states have consumer protection laws that allow the hearing aid to be returned after a trial use if the patient is not completely satisfied. In addition, to protect the health

and safety of people with hearing loss, the U.S. Food and Drug Administration (FDA) has established certain regulations. A medical evaluation of the hearing loss by a physician must be obtained within 6 months before the purchase of a hearing aid. However, the written statement from a physician may be waived if the patient (a fully informed adult 18 years or older) signs a document to this effect. Health care professionals who dispense hearing aids are required to refer prospective users to a physician if any of the following otologic conditions are evident:

- Visible congenital or traumatic deformity of the ear
- Active drainage from the ear within the previous 90 days
- Sudden or rapidly progressive hearing loss within the previous 90 days
- Complaints of dizziness or tinnitus
- Unilateral hearing loss that has occurred suddenly or within the previous 90 days
- Audiometric air–bone gap of 15 dB or more at 500, 1000, and 2000 Hz
- Significant accumulation of cerumen or a foreign body in the external auditory canal
- Pain or discomfort in the ear

A user instruction brochure is provided with every hearing aid device. In this brochure, the following information is presented:

- Notification that good health practice requires a medical evaluation before purchasing a hearing aid
- Notification that any of the otologic conditions listed previously should be investigated by a physician before purchase of a hearing aid
- Instructions for proper use, maintenance, and care of the hearing aid, as well as instructions for replacing or recharging the batteries
- Repair service information
- Description of avoidable conditions that could damage the hearing aid
- List of any known side effects that may warrant physician consultation (e.g., skin irritation, accelerated cerumen accumulation)

The evolution in technology has led to the availability of many smaller and more effective devices as well as different options and features of hearing aids (FDA, 2018a) (see Chart 59-13). The majority of hearing aids sold today are behind-the-ear, in-the-ear, or in-the-canal types (see Table 59-4). One model is the Lyric, which is placed in the ear canal just 4 mm from the tympanic membrane. Its volume is controlled by a magnet, and when its batteries no longer function (1 to 4 months), a physician can remove it with the magnet and reinsert a new device. This device does not have many of the problems (e.g., feedback noise, overamplification of background noise) associated with other hearing aids, and it does not involve the expense and uncertainty of surgical procedures. However, Lyric is not an option if the ear canal is too narrow to accommodate the device.

Chart 59-13

Options and Features of Hearing Aids to Consider

- *T-Coil*: May improve hearing on the telephone by switching the settings from normal to telephone setting. This feature also assists in amplifying voices when the patient is in larger areas, such as theaters, auditoriums, and gymnasiums. Background noise may be toned down to adequately hear close conversation.
- *Directional Microphone*: Useful in environments with a lot of background noise and activity. The microphone may be directed toward the speaker and amplifies conversation while diminishing background noise.
- *Direct Audio Input*: Allows direct connection from a remote microphone or an FM assistive listening system to devices such as a computer, television, or stereo.
- *Feedback Suppression*: Suppresses whistling feedback noise.

Adapted from U.S. Food and Drug Administration. (2018a). Medical devices: Types of hearing aids. Retrieved on 2/22/2020 at: www.fda.gov/medical-devices/hearing-aids/types-hearing-aids

TABLE 59-4 Hearing Aids

Site (Range of Hearing Loss)	Advantages	Disadvantages
Body, usually on the trunk (mild–profound)	Separation of receiver and microphone prevents acoustic feedback, allowing high amplification; generally used in a school setting	Bulky; requires long wire, which may be cosmetically displeasing; some loss of high-frequency response
Behind the ear (mild–profound)	Economical; powerful, with no long wires; easily used by children—adapts easily as the child grows, with only the ear mold needing replacement	Large size
In the ear (mild–moderately severe)	One-piece custom fit to contour of ear; no tubes or cords; miniature microphone located in the ear, which is a more natural placement; more cosmetically appealing due to easy concealment	Smaller size limits output; patients who have arthritis or cannot perform tasks requiring good manual dexterity may have difficulty with the small size of aid or battery; can require more repair than the behind-the-ear aid
In the canal (mild–moderately severe)	Same as in-the-ear aids; less visible, so more cosmetically pleasing	Even smaller than in-the-ear aids; requires good manual dexterity and good vision

Implanted Hearing Devices

There are several types of implanted hearing devices, ranging from implantable to semi-implantable devices (FDA, 2018b).

Bone conduction devices, which transmit sound through the skull to the inner ear, are used in patients with a conductive hearing loss if a hearing aid is contraindicated (e.g., those with chronic infection). The device is implanted postauricularly under the skin into the skull, and an external device—worn above the ear, not in the canal—transmits the sound through the skin. There are two types of implantable hearing aids. The bone-anchored hearing aid (BAHA) is implanted behind the ear in the mastoid area. The middle ear implant (MEI) is implanted in the middle ear cavity. The BAHA is used for conductive or mixed hearing loss, whereas the MEI is used for sensorineural hearing loss (FDA, 2018b).

The implantable middle ear hearing device (IMEHD) comes in two styles: piezoelectric and electromagnetic, which are partially or totally implanted. Patients must be 18 years or older, must be diagnosed with mild to severe

sensorineural loss, and must have tried other conventional devices with poor results to be considered candidates for this type of device. The implantable device has several advantages—for example, it may eliminate feedback, achieve good cosmetic results, and allow the patient to perform most preferred leisure activities (e.g., dancing, swimming). Disadvantages are that this device is expensive, necessitates surgery, requires periodic recharging of batteries, and has unpredictable power output (FDA, 2018b).

The FDA has also approved the semi-implantable Vibrant Soundbridge (electromagnetic) and the total implantable Envoy Esteem (piezoelectric) devices. The Vibrant Soundbridge has an external device attached to the postauricular bone that transmits sound to the magnet in the middle ear that is attached to the long process of the incus. The magnet surrounds the long axis of the stapes, which in turn vibrates and sound is heard. The Envoy Esteem works similarly to the natural ear. The piezoelectric transducer is located at the head of the incus, which sends a signal that is amplified, filtered, and then converted back to a vibration signal. This vibration is delivered by the driver (piezoelectric transducer) and attached to the stapes capitulum; then, via the stapes bone, the inner ear receives the signal and is converted into a nerve impulse and translated into sound by the brain. The incus is removed prior to insertion of this device to prevent feedback from the sensor. Clinical trials reported that in best-fit aided conditions, speech reception thresholds improved from 41.2 dB to 29.4 dB and word recognition score at 50 dB hearing level improved from best-fit aided of 46.3% to 68.9% with the Envoy Esteem (Kraus, Shohet, & Catalano, 2011). A 5-year follow-up study of the same group found that word recognition scores were improved by 17%; word recognition score at 50 dB hearing level improved in 49%, and remained the same in 41%; and speech reception threshold improved at every annual follow-up (Shohet, Kraus, Catalano, et al., 2018).

A cochlear implant is an auditory prosthesis used for people with profound sensorineural hearing loss bilaterally who do not benefit from conventional hearing aids. The cochlear implant directly stimulates the auditory nerve and has made it possible for people who have severe hearing loss or who are deaf to hear sound (NIDCD, 2017a). The hearing loss may be congenital or acquired. An implant does not restore normal hearing; rather, it helps the person detect medium to loud environmental sounds and conversation. The implant provides stimulation directly to the auditory nerve, bypassing the nonfunctioning hair cells of the inner ear. The microphone and signal processor, worn outside the body, transmit electrical stimuli to the implanted electrodes. The electrical signals stimulate the auditory nerve fibers and then the brain, where they are interpreted.

Worldwide, more than 324,200 people have received cochlear implants. In the United States, approximately 58,000 adults and 38,000 children have received cochlear implants (NIDCD, 2017a). Studies report that older adult

patients with cochlear implants experience improved understanding of speech and better cognition. Patients with cochlear implants were also noted to have more social interactions with others and improved quality of life. Research has reported that hearing improvement with a cochlear implant delays the occurrence as well as the progression of dementia (Sarant, Harris, Busby, et al., 2019). Candidates for a cochlear implant, who are usually at least 1 year of age, are selected after careful screening by otologic history, physical examination, audiologic testing, x-rays, and psychological testing. Criteria for choosing adults who may benefit from a cochlear implant include:

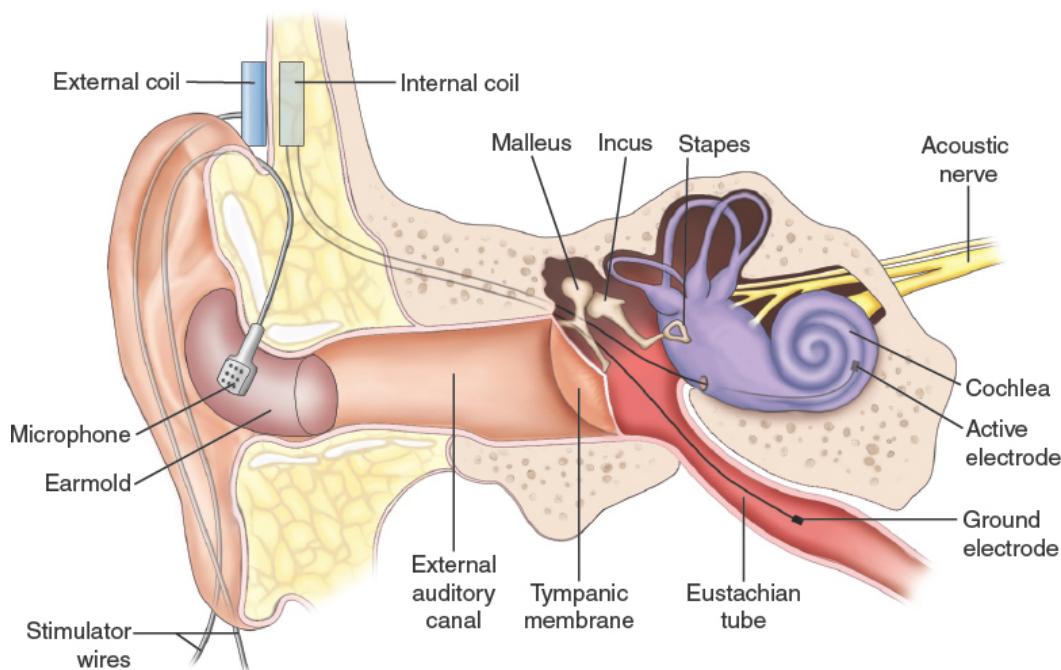


Figure 59-7 • The cochlear implant. The internal coil has a stranded electrode lead. The electrode is inserted through the round window into the scala tympani of the cochlea. The external coil (the transmitter) is held in alignment with the internal coil (the receiver) by a magnet. The microphone receives the sound. The stimulator wire receives the signal after it has been filtered, adjusted, and modified so that the sound is at a comfortable level for the patient. Sound is passed by the external transmitter to the inner coil receiver by magnetic conduction and is then carried by the electrode to the cochlea.

- Profound sensorineural hearing loss in both ears
- Inability to hear and recognize speech well with hearing aids
- No medical contraindication to a cochlear implant or general anesthesia
- Indications that being able to hear would enhance the patient's life

The surgery involves implanting a small receiver in the temporal bone through a postauricular incision and placing electrodes into the inner ear (see Fig. 59-7). The microphone and transmitter are worn on an external unit. The patient undergoes extensive cochlear rehabilitation with the multidisciplinary team, which includes an audiologist and speech pathologist. Several months may be needed to learn to interpret the sounds heard. Children and adults who lost their hearing before they learned to speak take much longer to acquire speech. There are wide variations of success with cochlear implants, and there is also controversy about their use, especially among the Deaf community. Patients who have had a cochlear implant are cautioned that an MRI scan will inactivate the implant; MRI should be used only when there is no other diagnostic option.

Hearing Guide Dogs

Specially trained dogs (service dogs) are available to assist the person with a hearing loss. People who live alone are eligible to apply for a dog trained by International Hearing Dog, Inc. The dog reacts to the sound of a telephone, a doorbell, an alarm clock, a baby's cry, a knock at the door, a smoke alarm, or an intruder. The dog alerts its master by physical contact; the dog then runs to the source of the noise. In public, the dog positions itself between the person who is deaf or has severe hearing loss and any potential hazard that the person cannot hear, such as an oncoming vehicle or a loud, hostile person. A certified hearing guide dog is legally permitted access to public transportation, public eating places, and stores, including food markets.

CRITICAL THINKING EXERCISES

1  ebp A 65-year-old woman visits the clinic where you work for a routine well-woman examination. She mentions that her friend was recently diagnosed with hearing loss and fitted for a hearing aid. She has no perceived hearing loss, but she wonders whether she needs a hearing aid. What evidence-based recommendations would you make for this patient about hearing loss screening and diagnosis? What is the strength of the evidence for your recommendations?

2  pc A 40-year-old man has complained of hearing loss that has gradually worsened over the past few months. He has difficulty hearing the television and having a telephone conversation. After seeing the primary provider today, he was diagnosed with cerumen impaction. Discuss how you will educate the patient about the diagnosis of cerumen impaction. What is your priority for care of this patient? Give your rationale.

3  ipc A 54-year-old man had a cochlear implant placed 5 years ago and has been admitted to the medical-surgical unit due to implant malfunction. Develop a plan of action for communicating with this patient. What should you know about cochlear implants to communicate effectively with the patient? What environmental conditions should be considered to promote patient safety and well-being? Which members of the health care team should be informed about this patient's communication needs?

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*Asterisk indicates nursing research.

**Double asterisk indicates classic reference.

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Resources

- Acoustic Neuroma Association (ANA), www.anausa.org
- Alexander Graham Bell Association for the Deaf and Hard of Hearing, www.agbell.org
- American Academy of Audiology, www.audiology.org
- American Academy of Facial Plastic and Reconstructive Surgery, www.aafprs.org
- American Academy of Otolaryngology—Head and Neck Surgery, www.entnet.org
- American Board of Audiology, www.boardofaudiology.org
- American Cochlear Implant Alliance (ACI Alliance), www.acialliance.org
- American Speech-Language-Hearing Association, www.asha.org
- American Tinnitus Association (ATA), www.ata.org
- Association of Late-Deafened Adults, Inc. (ALDA), www.alda.org
- Association of Medical Professionals with Hearing Losses (AMPHL), www.amphl.org
- Center for Hearing and Communication, www.chcheating.org
- Hearing Health Foundation, www.hearinghealthfoundation.org
- Hearing Loss Association of America (HLAA), www.hearingloss.org
- International Federation of Hard of Hearing People (IFHOH), www.ifhoh.org
- International Hearing Dog, Inc., www.hearingdog.org
- National Association of the Deaf (NAD), www.nad.org
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- Usher Syndrome Coalition, www.usher-syndrome.org
- Vestibular Disorders Association, www.vestibular.org
- World Federation of the Deaf (WFD), www.wfdeaf.org