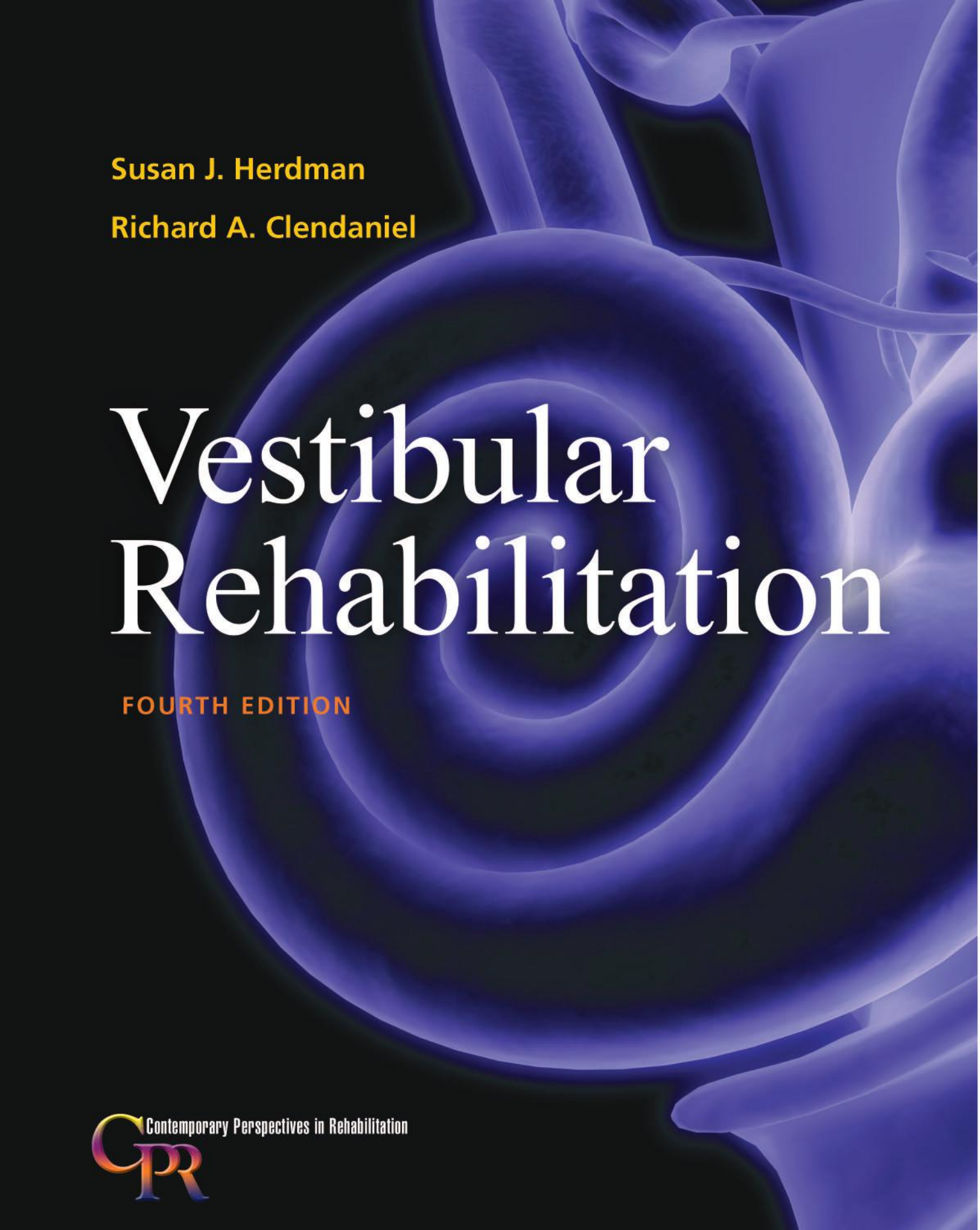
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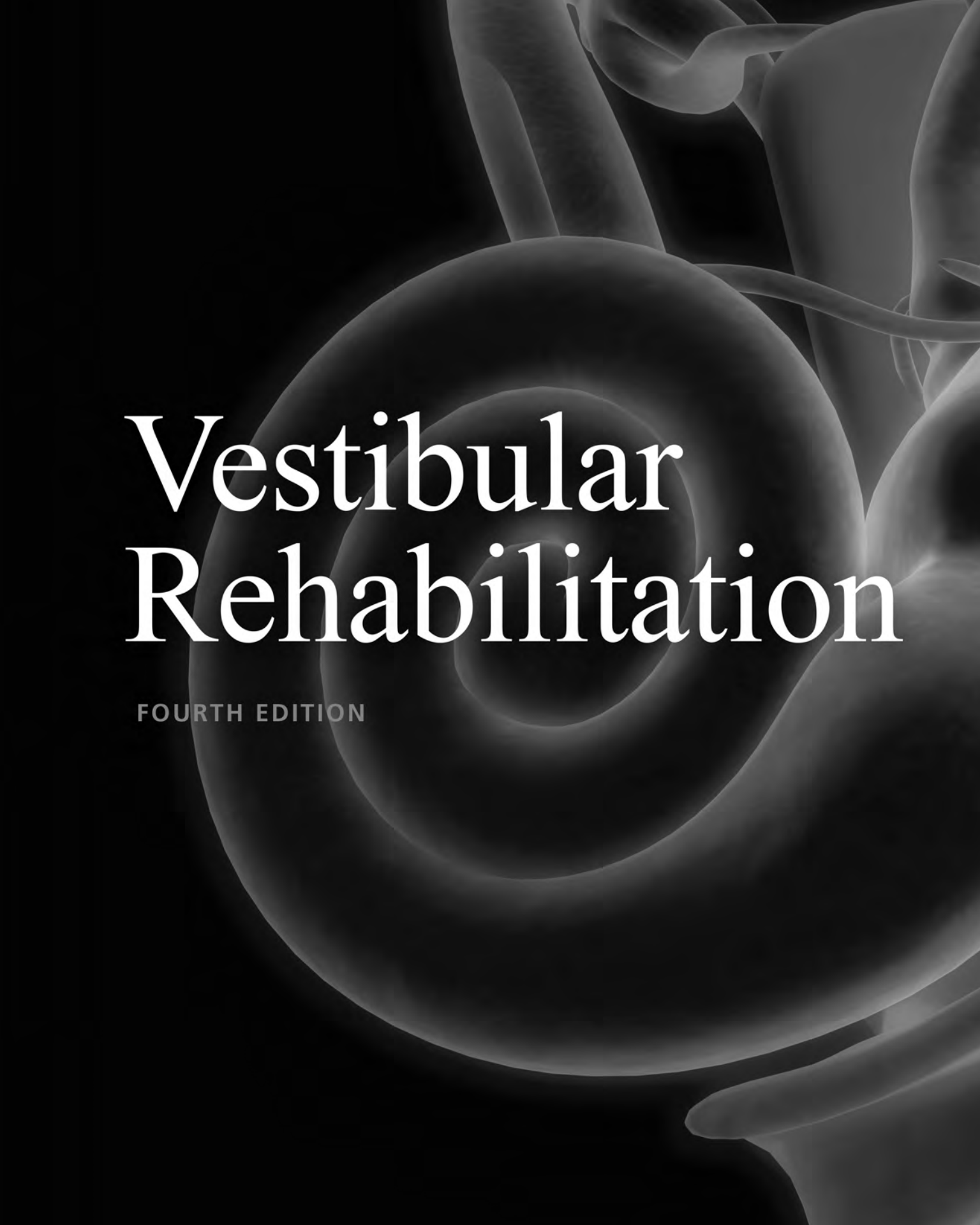

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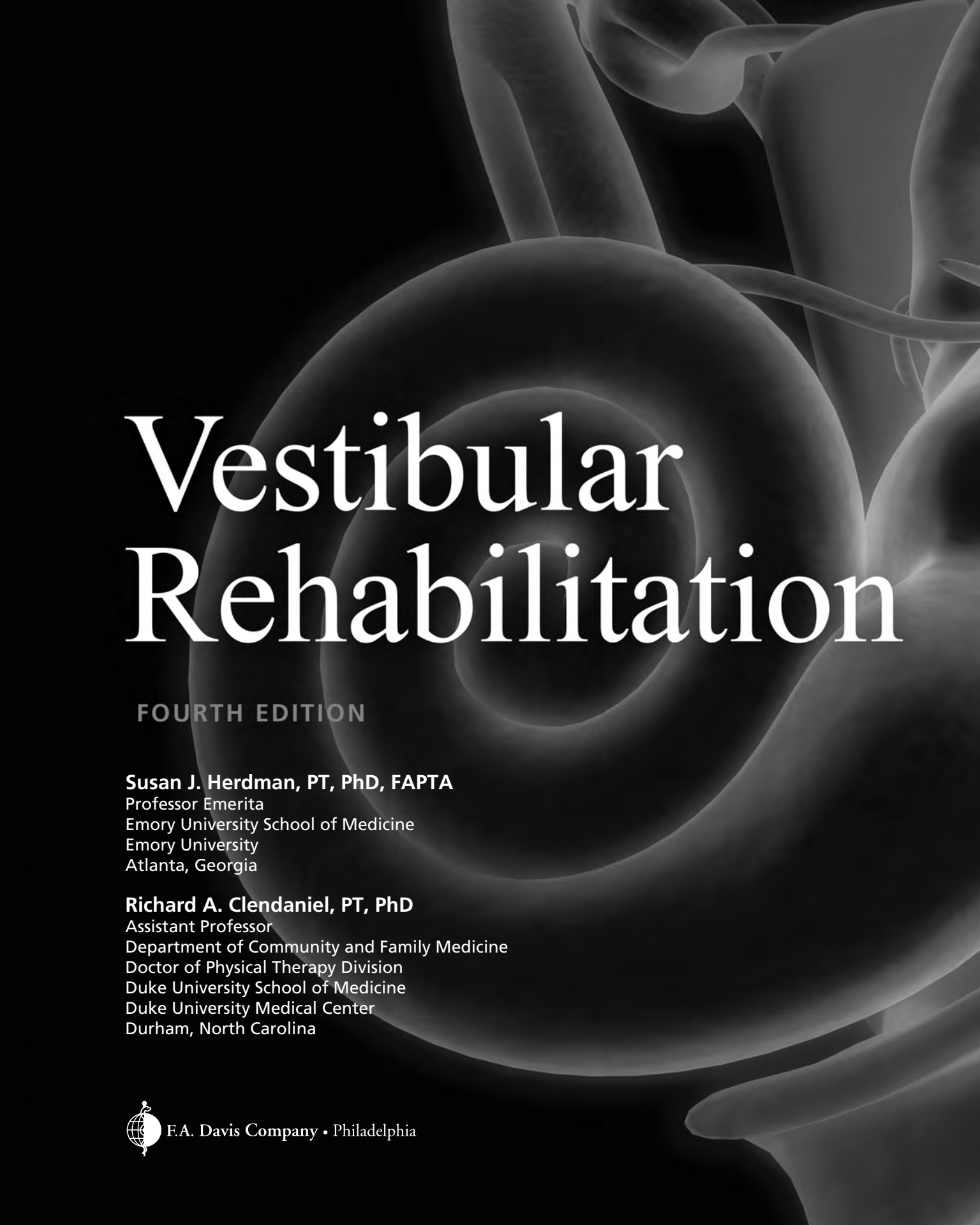

Vestibular Rehabilitation

FOURTH EDITION

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This book is dedicated to "serendipity." The serendipity that led to me walking into a patient's room one weekend and finding there the person who would become my doctoral advisor. The serendipity that led to that surprising day when, after years of saying "no" to treating patients for their dizziness, I saw my first patient with BPPV and was forever "hooked" by that patient's gratitude. The serendipity that led to me working at Hopkins and NIH. The serendipity that led to four colleagues sitting down and planning a competency-based course in Vestibular Rehabilitation that we thought would last maybe 3 or 4 years. The serendipity that put so many wonderful opportunities in my life. The serendipity that put so many incredible people in my life. The grace that led me to take advantage of it all.
SJH

I would like to dedicate this book to my family who has supported me throughout, to my colleagues who have contributed to this book and to my education, and to my friend, colleague, mentor, and coeditor whose knowledge, guidance, and inspiration have been critical to my professional growth.
RAC

Foreword

Benjamin Franklin once said, "Life's tragedy is that we get old too soon and wise too late." While there is substantial wisdom in that statement, I can think of at least one exception that might set old Ben on his rear, or is it ear? It is hard to believe that 20 years have passed since the first edition of *Vestibular Rehabilitation* was published. Yet even at its birth there was considerable wisdom in its words. At that time the responsibility for evaluating and rehabilitating patients presenting with dizziness and vestibular compromise was restricted to a select few, and this area of rehabilitation had not emerged as a specialty. Susan Herdman recognized a need to train rehabilitation clinicians to better understand and treat the postural compromise generated through visual deficits, headache or other maladies that would adversely impact the quality of life of those unfortunate enough to have acquired vestibular pathologies. The extent to which this knowledge has grown over these two decades and with it, the interest of students and clinicians is truly extraordinary. Throughout this time *Vestibular Rehabilitation* has remained the "go to" text for specialists in this area while also serving as a critical reference source for all neurorehabilitationists.

All contributors to the *Contemporary Perspectives in Rehabilitation* series have always prided themselves on maintaining the infrastructural integrity of its foundation…….evidence based referencing, problem solving presentations and the latest and most novel data or treatment techniques. *Vestibular Rehabilitation* continues to adhere to these principles and undoubtedly the result has been a steadfast allegiance to its content from amongst its followers…….teachers, clinicians and students. The fact that the content has been updated consistently at 5 year intervals speaks to the dedication shown by its contributing authors, many of whom have persisted through several editions. Throughout this time, Dr. Herdman has amassed a larger international cohort of knowledgeable clinicians, many of whom have come to assist her in what arguably might be called the most popular (and intense) vestibular rehabilitation course in the United States, if not the world. Many of those once considered novices are now positioned to disseminate information themselves. Undoubtedly, Dr. Herdman has assembled the next generation of contributors. Amongst the 34 individuals whose collective efforts define the 4th edition of *Vestibular Rehabilitation*, 14 are first timers and all possess the skill and knowledge to become persistent contributors to subsequent editions. As one reads Chapters 3 (Laurie King), 6 (Anne Galgon), 7 (Natalia Ricci), 13 (Steve Benton), 14 (Rosalyn Schneider), 17 (Yew Ming Chan), 19 (Jeffrey Staab), 20 (Jeff Hoder), 24 (Jennifer Braswell Christy), 26 (Laura Morris and Kim Gottshall), 28 (Courtney Hall and Dara Meldrum) and 30 (Lisa Heusel-Gillig and Courtney Hall), please know that while these individuals might not be the sole author of those respective contributions, their effort to revise previous content was substantial and the resulting quality of their work is outstanding.

In this edition, all chapters have been updated and new cases permeate the management chapters to illustrate how patients with specific problems are treated. The thought-provoking and problem solving nature of the clinical chapters are supported by the necessity to foster evidence based references, a fundamental tenet of all volumes in the *Contemporary Perspectives in Rehabilitation* series. While the 3rd edition of Vestibular Rehabilitation was accompanied by 68 videos, this edition has over 100. The videos include normal and abnormal eye movements, assessment procedures, demonstrations of some exercises, and gait assessments. Chapters emphasizing tinnitus, novel approaches in the assessment and treatment of anterior and horizontal canal benign paroxysmal positional vertigo (BPPV), and data on outcomes in patients with unilateral and bilateral vestibular hypofunction are distinctly new to the 4th edition. The management of patients with chronic subjective dizziness represents another addition to the text. These are patients who present with chronic dizziness and motion sensitivity that often is accompanied by variable amounts of anxiety and phobic behavior.

The text has been further contemporized through the addition of a chapter on the management of patients with vestibular problems precipitated from head trauma

including etiologies from post-concussion syndrome caused by blast injuries. Regrettably there is ample reason to believe that the incidence of this problem may be profoundly underestimated. If so, then this category of patient will present unique challenges to vestibular rehabilitation clinicians and the treatment components may well incorporate a need to foster compliance because of concomitant behavioral changes. Additional information is contained in information regarding emerging technologies for the treatment of patients with vestibular disorders. Such rehabilitation includes novel biofeedback alternatives, use of virtual reality and gaming, such as the Wii. These advances, while challenging, provoke opportunities to discover and implement new approaches towards enlisting functional plasticity to achieve enhanced quality of life. We can be rest assured that advances in technologies and devices will continue to make their way into the armamentarium of tools to restore optimal balance, reduce dizziness or improve the consequences of migraine, as examples.

The contributions from vestibular neurorehabilitation therapists and specialty physicians are woven along a highly integrated network. Over each edition, the blend of input from these specialists becomes more tightly coupled. This fluidity may go unrecognized by students, but suffice to say, there are very few courses, let alone text books, in which the content amongst different specialists, both physician and non-physician, can be assembled and transmitted so smoothly. Herein rests an opportunity to learn from a panel of experts who would be very difficult to assemble collectively. As I concluded in a previous Foreword, the sum of these parts is truly greater than its whole……

Steven L. Wolf, Ph.D., PT, FAPTA, FAHA
Series Editor

Preface

There are several additions to the fourth edition of *Vestibular Rehabilitation*. One change is the addition of Richard A. Clendaniel as the coeditor. Rick is a clinician, educator, and researcher with many years of experience in vestibular rehabilitation, vestibular function testing, and vestibular physiology. His contributions to the field have been of benefit to many clinicians and researchers as well as to patients. The other changes in this edition of *Vestibular Rehabilitation* are the exploration of new areas of "vestibular" rehabilitation including current evidence that supports the use of the gaze stabilization exercises in patients with non-vestibular dizziness and the use of new technologies in exercise programs.

The practice of vestibular rehabilitation faces numerous challenges in the coming years, as does all health care. We must be more efficient in our assessments and treatments. We must provide evidence that the patient is improving through the application of functional outcome measures. We must be able to defend our choice of treatment based on research establishing the benefits of specific exercises or establishing that a specific exercise is not beneficial. We must be able to support our recommendation on the need for further treatment based on measures that suggest the patient has or has not reached the optimal level of recovery. We need to address the psychological state of the patient as well as the physical problems and their consequences.

As you should hope, the evidence supporting the treatment of patients with vestibular deficits has increased since the last edition 7 years ago. So once again, we have extended the material presented to include several new chapters to reflect and to challenge our understanding of the assessment and treatment of vestibular disorders. One new chapter is on management of patients with chronic subjective dizziness and is a nice complement to the chapter of psychological problems in patients with dizziness. Another chapter tackles a current "hot topic"—management of patients with vestibular dysfunction from concussion. Finally, a third new chapter explores the role of emerging technologies such as virtual reality, sensory substitution devices, and most excitedly, vestibular implants, which will undoubtedly require new treatment approaches. Of course, all chapters contain new material, from management of tinnitus to the newer treatments that have been proposed for anterior and horizontal semicircular canal BPPV. We have added "point and counterpoint" sections in some chapters that highlight the differing opinions about assessment and treatment. The number of videos has been increased to include more examples of eye movement abnormalities and to provide visual examples of some of the newer treatments.

The many chapters in this book are designed to provide you with a foundation for all these challenges, but more importantly, the chapters will hopefully give you the basis from which you can continue to apply new information in your practice for the betterment of all your patients.

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Acknowledgments

I want to share with anyone who finds difficulty in mastering the information in this book and mastering how to treat people with "dizziness" that it's not unusual to start with a very real sense of inadequacy! I will always remember that during my early attempts to learn about "vestibular rehabilitation" I visited Fay Horak and was mystified as to how everyone knew that the patient had a central rather than a peripheral vestibular deficit. If it's any encouragement, I realize that I have come a "long way" since then but I would also add that I am still learning and I hope I always will be. And so, I would like to express my gratitude again to the many people it has been my good fortune to meet, work with, teach, treat, and learn from over my years as a physical therapist. These have included outstanding scientist-clinicians in the field of vestibular rehabilitation (many of whom have authored chapters in this book), teachers and clinicians from all over the world, students, and of course, patients. Thank you all so much.

SJH

I would like to express my sincere gratitude to the authors who shared their knowledge and expertise in this edition of *Vestibular Rehabilitation*; it is their contributions that make this text such a valuable resource for understanding and treating the patient with dizziness. I would like to also thank the patients, clinicians, and students who question and challenge what we do, forcing us to rethink our assumptions, to continue to learn, and continue to investigate the "best" treatments for individuals suffering from vestibular disorders. I have been blessed to learn from some of the very best, and recognizing all these individuals by name would fill many pages. So I would simply like to offer my thanks to all who have taught me over the years, to fortuitous meetings on Nantucket, and to an atypical insight that kept me from asking a really stupid question. Lastly, an immense 'thank you' to my family for their love, support and encouragement over the years.

RAC

Contents in Brief

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ONE

Fundamentals of Function

CHAPTER 1

Anatomy and Physiology of the Normal Vestibular System

Timothy C. Hain, MD ■ *Janet Helminski, PhD, PT*

Purpose of the Vestibular System

The human vestibular system estimates body position and motion. Motion inputs to the vestibular system include the inner ear signals ("vestibular" in Fig. 1.1), as well as position sensation, ("proprioception") visual signals, and intended movement ("motor commands"). These redundant inputs are integrated by the central processor, the "vestibular nuclear complex," which generates motor commands to drive the eyes and body. The system is normally very accurate. To maintain accuracy, the vestibular system is monitored and calibrated by the cerebellum.

The eye and body movement output of the central vestibular system is generally described in terms of three simple reflexes, the vestibulo-ocular reflex (VOR), the vestibulocollic reflex (VCR), and the vestibulospinal reflex (VSR). The VOR generates eye movements that enable clear vision while the head is in motion. The VCR acts on the neck musculature to stabilize the head. The VSR generates compensatory body movement to maintain head and postural stability and thereby prevent falls. Although the nomenclature of these reflexes, for example "vestibular ocular reflex," might make one think that these circuits are only concerned with inner ear input, the vestibular nucleus that drives all these reflexes also processes input from the other sources listed above.

After an acute loss of peripheral vestibular function, as for example after surgical removal of a tumor of the vestibular nerve, postural and oculomotor deficits appear. With the head still, spontaneous jumping of the eyes (nystagmus) and tilting of the body away from upright appear. When the head is moved, vision and balance are further impaired. Associated with these deficits are a reduced propensity to move the head as well as behavioral changes aimed at minimizing the risk of disorientation by avoiding visual input and minimizing the risk of falling by adopting a more cautious and stable stance.

Recovery from vestibular lesions has been studied for over 100 years.[1] Orientation in space and being able to walk upright are critical functions. It is understandable that the vestibular system is supported by multiple vestibular repair mechanisms. The capability for repair and adaptation is remarkable! Plasticity consists of neural adjustments that restore original function. This is supplemented by substitution of other sensory input or internal estimates. Finally, one may change one's behavior to "work around" problems presented by a vestibular lesion.

Given sufficient time, persons with up to approximately 50% loss of vestibular function adapt so well that a casual observer may find them indistinguishable from someone without a vestibular lesion. Nevertheless, such persons can rarely attain the same degree of performance as normal, and a sophisticated clinician can nearly always detect this situation.

In this chapter, we describe the anatomy and neurophysiology of the vestibular system, paying particular attention to aspects relevant to rehabilitation. We proceed

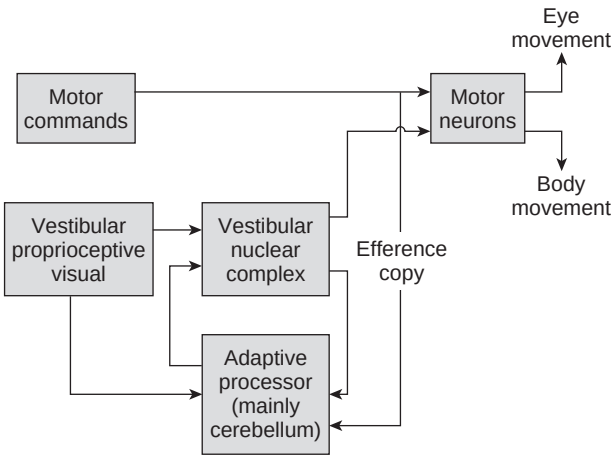

Figure 1.1 Block diagram illustrating the organization of the vestibular system. (Copyright Timothy C. Hain, MD.)

from the peripheral to central structures and conclude with a discussion of "higher-level" problems in vestibular neurophysiology, which are relevant to rehabilitation.

The Peripheral Sensory Apparatus

Figure 1.2 illustrates the peripheral vestibular system, which lies within the labyrinth of the inner ear. The labyrinth, subdivided into the bony and membranous labyrinths, contains the hair cells, which are the motion receptors of the vestibular system. The labyrinth is bordered laterally by the air-filled middle ear and medially by the temporal bone and is posterior to the cochlea.

Bony Labyrinth

The bony labyrinth consists of three semicircular canals (SCCs), the cochlea, and a chamber between the two called the vestibule (Fig. 1.3). The bony labyrinth is filled with perilymphatic fluid, which has chemistry similar to cerebrospinal fluid (high Na:K ratio). Perilymphatic fluid communicates via the cochlear aqueduct with cerebrospinal fluid. Because of this communication, disorders that affect spinal fluid pressure (such as lumbar puncture) can also affect inner ear function.[2]

Membranous Labyrinth

The membranous labyrinth (Fig. 1.3) is suspended within the bony labyrinth by perilymphatic fluid and supportive connective tissue. It contains five sensory organs: the membranous portions of the three semicircular canals and the two otolith organs, the utricle and saccule. Note that one end of each semicircular canal is widened in diameter to form an ampulla (bottom of Fig. 1.3). This widening will be relevant when we later

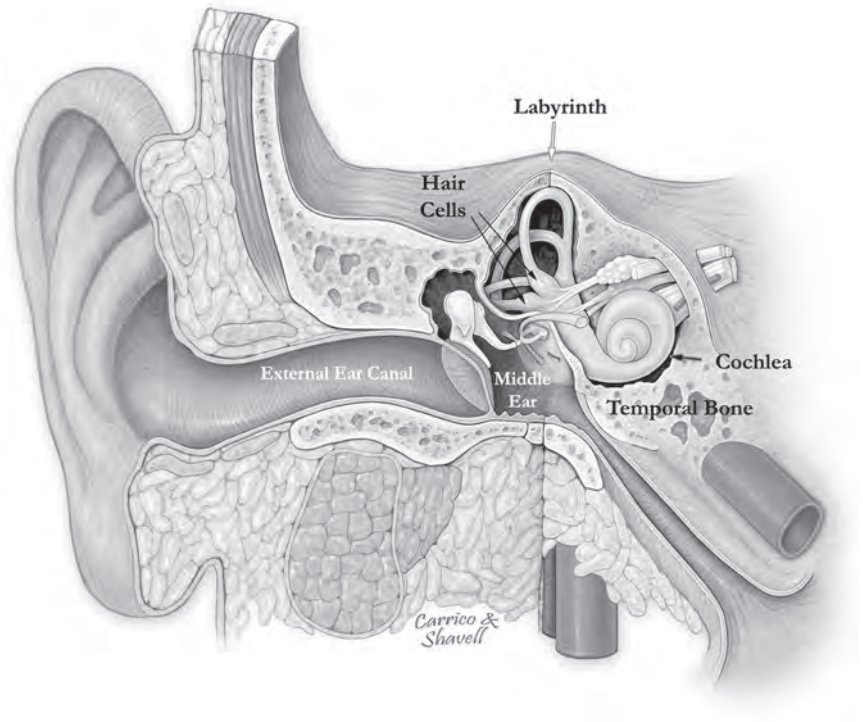

Figure 1.2 Vestibular and auditory apparatus in relation to skull. (Copyright Timothy C. Hain, MD.)

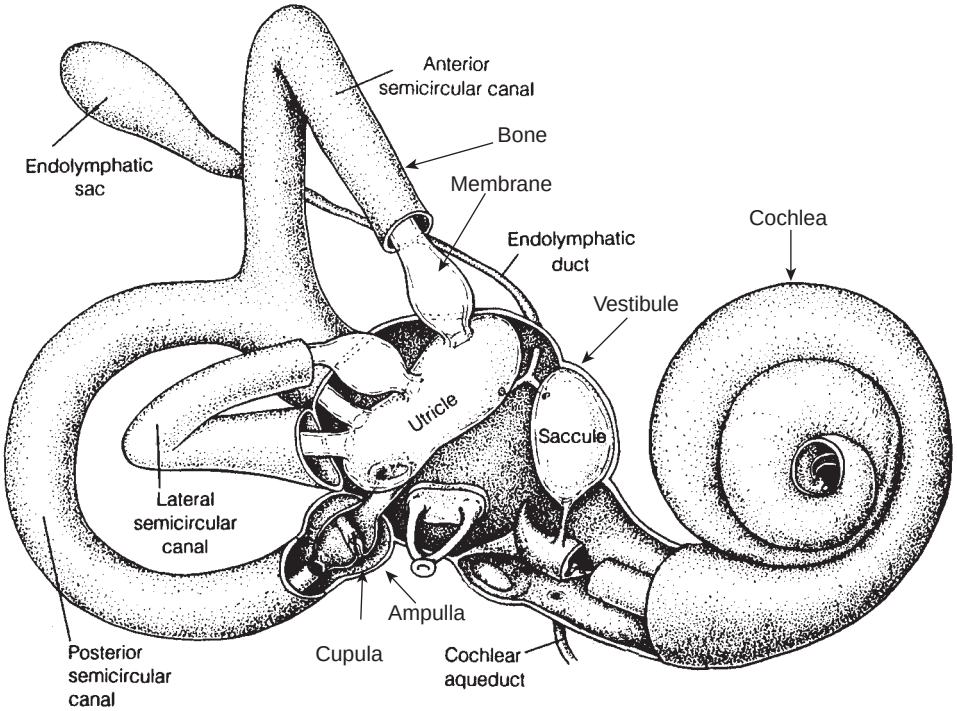

Figure 1.3 The membranous and bony labyrinths. (Adapted from an illustration by Mary Dersch from Pender, 1992.)[2]

discuss a common vestibular condition, benign paroxysmal positional vertigo.

The membranous labyrinth is filled with endolymphatic fluid. In contrast to perilymph, the electrolyte composition of endolymph resembles intracellular fluid (high K:Na ratio). Under normal circumstances, no direct communication exists between the endolymph and perilymph compartments.

Hair Cells

Hair cells contained in each ampulla and otolith organ convert displacement due to head motion into neural firing (Fig. 1.4). The hair cells of the ampullae rest on a tuft of blood vessels, nerve fibers, and supporting tissue called the crista ampullaris. The hair cells of the saccule and utricle, the maculae, are located on the medial wall

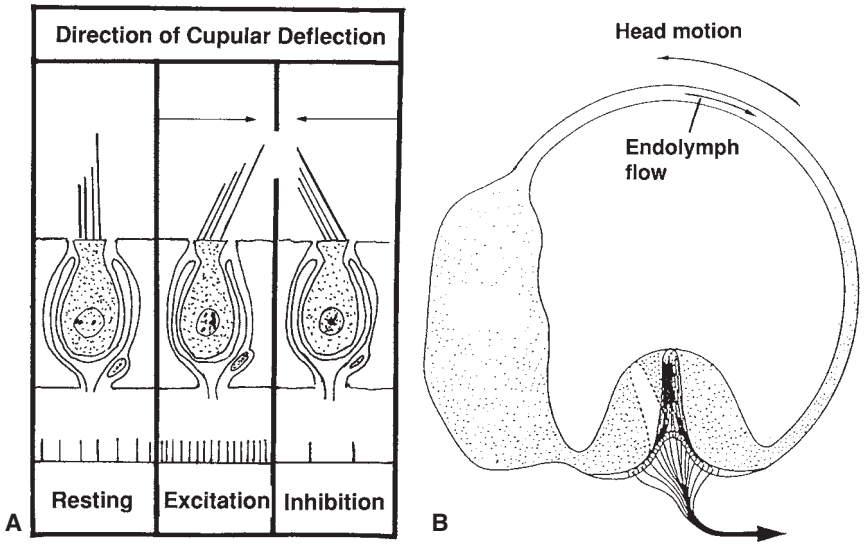

Figure 1.4 Effects of head rotation on the canals. **(A)** The direction from which hair cells are deflected determines whether hair-cell discharge frequency increases or decreases.[3] **(B)** Cross section of the membranous labyrinth illustrating endolymph flow and cupular deflection in response to head motion.[4]

of the saccule and the floor of the utricle. Each hair cell is innervated by an afferent neuron whose cell body lies in the vestibular (Scarpa's) ganglion, which is located close to the ampulla. When hairs are bent toward or away from the longest process of the hair cell, firing rate increases or decreases in the vestibular nerve (see Fig. 1.4*A*). A flexible, diaphragmatic membrane called the cupula overlies each crista and completely seals the ampulla from the adjacent vestibule. With angular head motion, endolymphatic pressure differs across the cupula and causes the cupula to bend back and forth, which stimulates the hair cells (see Fig. 1.4*B*).[4]

The otolithic membranes are structures similar to the cupulae but are weighted. They contain calcium carbonate (limestone) crystals called otoconia and have substantially more mass than the cupulae (Fig. 1.5). The mass of the otolithic membrane causes the maculae to be sensitive to gravity and linear acceleration. In contrast, the cupulae normally have the same density as the surrounding endolymphatic fluid and are insensitive to gravity.[3]

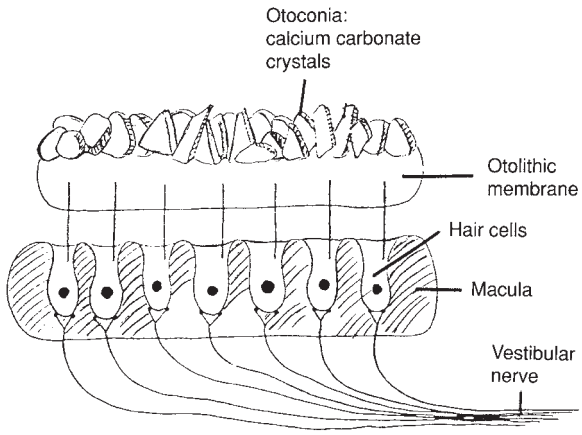

Figure 1.5 The otolithic macula and its overlying membrane.[3]

Vascular Supply

The labyrinthine artery supplies the peripheral vestibular system (Fig. 1.6; see also Fig. 1.11).The labyrinthine artery has a variable origin. Most often it is a branch of the anterior inferior cerebellar artery (AICA—top of 1.6), but occasionally it is a direct branch of the basilar artery. Upon entering the inner ear, the labyrinthine artery divides into the anterior vestibular artery and the common cochlear artery. The anterior vestibular artery supplies the vestibular nerve, most of the utricle, and the ampullae of the lateral and anterior SCCs. The common cochlear artery divides into a main branch, the main cochlear artery, and the vestibulocochlear artery. The main cochlear artery supplies the cochlea. The vestibulocochlear artery supplies part of the cochlea, the ampulla of the posterior semicircular canal, and the inferior part of the saccule.[5]

The labyrinth has no collateral anastomotic network and is highly susceptible to ischemia. Only 15 seconds of

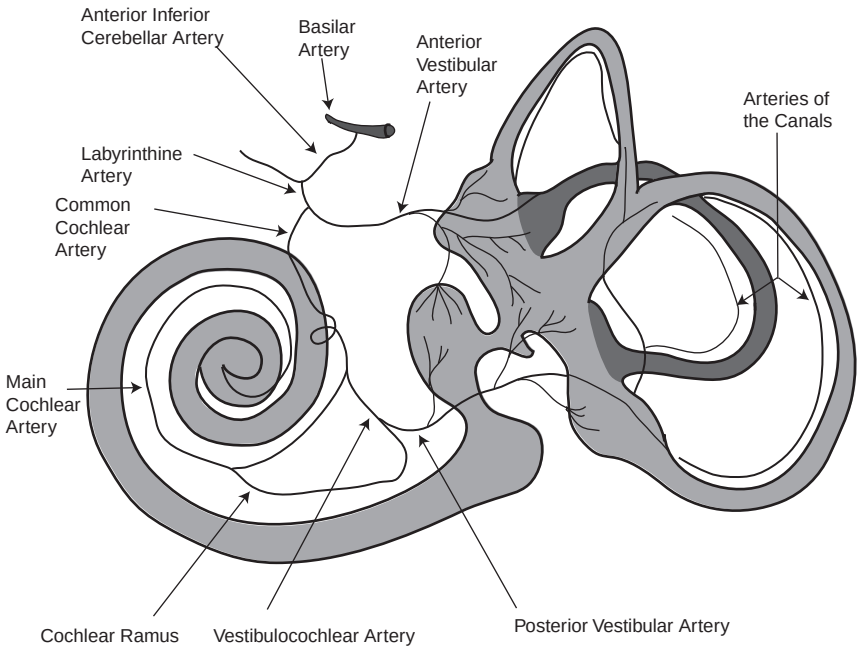

Figure 1.6 The arterial supply of the labyrinth.[5]

selective blood flow cessation is needed to abolish auditory nerve excitability.[6]

Physiology of the Periphery

The hair cells of the canals and otoliths convert the mechanical energy generated by head motion into neural discharges directed to specific areas of the brainstem and the cerebellum. By virtue of their orientation, the canals and otolith organs are able to respond selectively to head motion in particular directions. Because of differences in their fluid mechanics, the canals respond to angular velocity, and the otoliths to linear acceleration.

Semicircular Canals

The SCCs provide sensory input about head velocity, which enables the VOR to generate an eye movement that matches the velocity of the head movement. The desired result is that the eyes remain stationary in space during head motion, enabling clear vision. Neural firing in the vestibular nerve is proportional to head velocity over the range of frequencies in which the head commonly moves (0.5 to 7 Hz). In engineering terms, the canals are "rate sensors."

This fact poses a significant problem: How do the hair cells of the SCCs, which are activated by displacement, produce sensory input proportional to velocity? The labyrinth must have a method of converting head velocity into displacement. Biophysical properties of the semicircular canals' loops accomplish the conversion.[7] The membranous canal loops have very thin walls and a small lumen diameter relative to the radius of the loop curvature. These characteristics make viscous drag on the endolymph very powerful. Viscosity, or fluidic friction, slows down endolymph flow in a way similar to how honey slowly runs down the side of a jar. In a frictionless system with a freely moving cupula, for a step of constant rotational velocity, endolymph displacement would be proportional to velocity times time, or rotational position. The viscosity creates resistance to endolymph movement so that trans-cupular pressure and displacement become more closely proportional to head velocity. Because of these considerations, over the usual frequencies of head movement, endolymph displacement is proportional to angular head velocity, and the SCCs transmit a velocity signal to the brain.

A second important dynamic characteristic of the canals has to do with their response to prolonged rotation at constant velocity. Instead of producing a signal proportional to velocity, the canals respond reasonably well only in the first second or so, because output decays exponentially with a time constant of about 7 seconds. This behavior is due to a springlike action of the cupula that tends to restore it to its resting position.[7]

Three important spatial arrangements characterize the alignment of the SCC's loops. First, each canal plane within each labyrinth is perpendicular to the other canal planes, analogous to the spatial relationship between two walls and the floor of a rectangular room (Fig. 1.7). Second, paired planes of the SCCs between the labyrinths conform very closely to each other. The six individual semicircular canals become three *coplanar pairs*: (1) right and left lateral, (2) left anterior and right posterior, and (3) left posterior and right anterior. Third, the planes of the canals are close to the planes of the extraocular muscles, thus allowing relatively simple connections between sensory neurons (related to individual canals) and motor output neurons (related to individual ocular muscles).

The coplanar pairing of canals is associated with a *push-pull* change in the quantity of SCC output. When angular head motion occurs within their shared plane, the endolymph of the coplanar pair is displaced in opposite directions with respect to their ampullae, and neural firing increases in one vestibular nerve and decreases on the opposite side. For the lateral canals, displacement of the cupula toward the ampulla (ampullopetal flow) is excitatory, whereas for the vertical canals, displacement of the cupula away from the ampulla (ampullofugal flow) is excitatory.

There are three advantages to the push-pull arrangement of coplanar pairing. First, pairing provides sensory

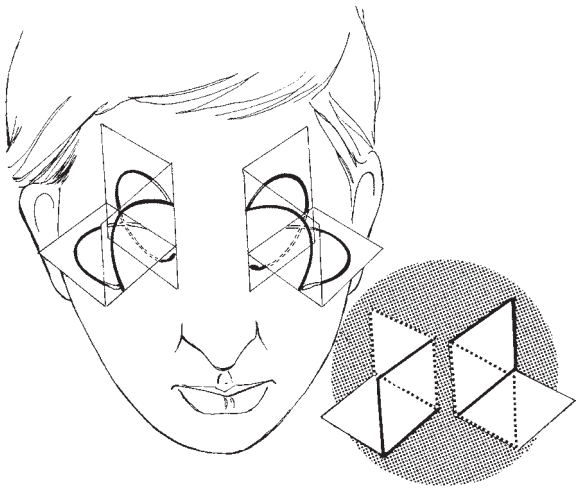

Figure 1.7 The spatial arrangement of the semicircular canals. The canals on each side are mutually perpendicular, are paired with conjugate canals on the opposite side of the head, and also are closely aligned with the optimal pulling directions of the extraocular muscles.

redundancy. If disease affects the SCC input from one member of a pair (e.g., as in vestibular neuritis), the central nervous system will still receive vestibular information about head velocity within that plane from the contralateral member of the coplanar pair.

Second, such a pairing allows the brain to ignore changes in neural firing that occur on both sides simultaneously, such as might occur as a result of changes in body temperature or neurochemistry. These common changes in firing in both nerves are not related to head motion and are called common-mode noise. The engineering term for the desirable feature of ignoring this type of noise is called *common-mode rejection*. Third, as is discussed in a later section, a push-pull configuration assists in compensation for overload.

Otoliths

The otoliths register forces related to linear acceleration (Fig. 1.8). They respond to both linear head motion and static tilt with respect to the gravitational axis. The function of the otoliths is illustrated by the situation of a passenger in a commercial jet. During flight at a constant velocity, we have no sense that we are traveling at 300 miles per hour. However, in the process of taking off and ascending to cruising altitude, we sense the change in velocity (acceleration) as well as the tilt of the plane on ascent. The otoliths therefore differ from the SCC in two basic ways: They respond to linear motion instead of angular motion, and their output is mainly proportional to acceleration rather than velocity.[7]

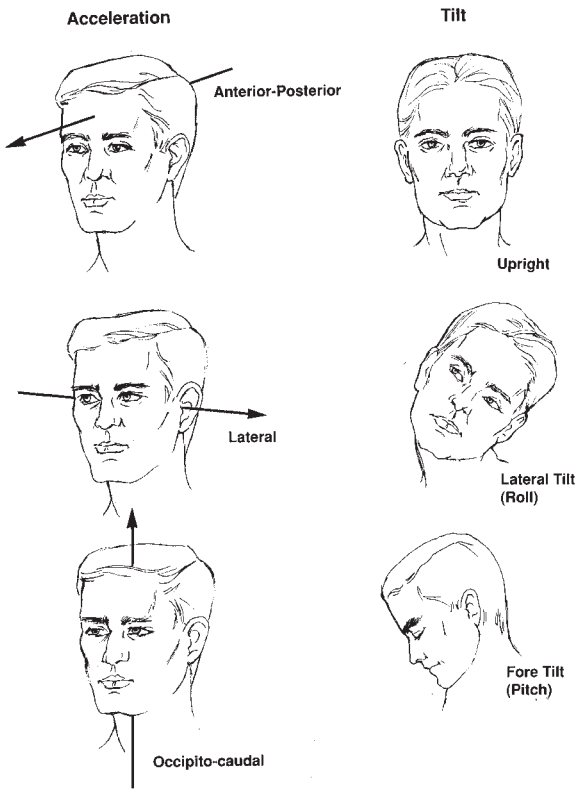

Figure 1.8 The otoliths register linear acceleration and static tilt.

The otoliths have a simpler task to perform than the canals. Unlike the canals, which convert head velocity into cupular displacement through hydrodynamic viscosity, the otoliths need no special hydrodynamic system to do their job. Exquisite sensitivity to gravity and linear acceleration is obtained by incorporating crystals of calcium carbonate (limestone), the otoconia, into the otolithic membrane (see Fig. 1.5). Because force is equal to mass times acceleration, by incorporating a large mass, a given acceleration produces enough shearing force to make the otoliths extremely sensitive. (Shearing force refers to force that is directed perpendicularly to the processes of the hair cells.)

Like the canals, the otoliths respond to motion in all three dimensions (Fig. 1.9). However, unlike the SCC of one ear, which have one sensory organ for each axis of angular motion, there are two sensory organs for three axes of linear motion. In an upright individual, the saccule is vertical (parasagittal), whereas the utricle is horizontally oriented (near the plane of the lateral SCC). The saccule senses linear acceleration in the sagittal plane, such as might be associated with a forward pitch of the head. The utricle senses acceleration in its predominantly horizontal plane, such as might be provoked by a roll (lateral tilt) of the head.[8] The two organs together can encode all possible vectors of linear acceleration.

Because earth's gravitational field is a linear acceleration field, on earth the otoliths register tilt. For example, as the head is tilted laterally (which is also called roll; see Fig. 1.9), shear force is exerted on the utricle, while shear force is lessened on the saccule. Similar changes occur when the head is tilted forward or backward (pitch). Because linear acceleration can come from two sources—earth's gravitational field and linear motion—there is an ambiguity problem. We discuss strategies that the central nervous system might use to solve this problem in our section on higher-level vestibular processing.

For the otoliths, as in the canals, there is redundancy, because there are organs similar in orientation and function located on both sides of the head. Push-pull processing for the otoliths is also incorporated into the geometry of each of the otolithic membranes. Within each otolithic macula, a curving zone, the striola, separates the direction of hair cell polarization on each side. Consequently, head

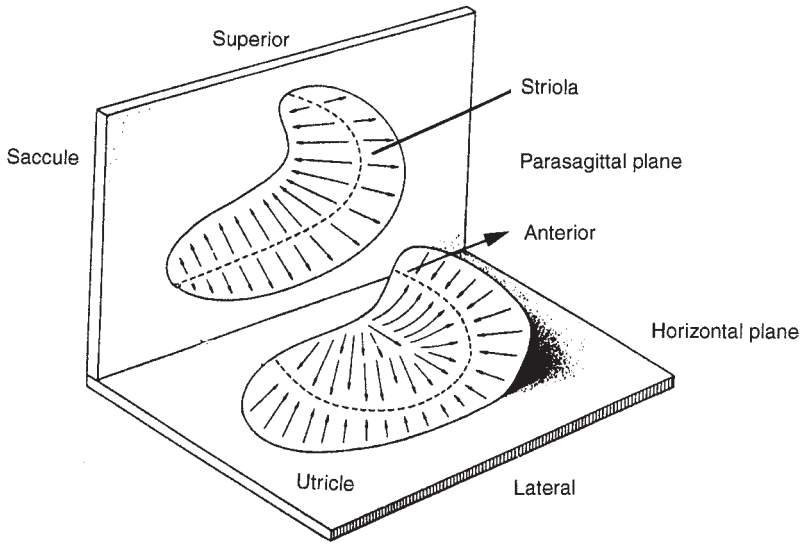

Figure 1.9 Geometry of the otoliths.[8]

tilt results in increased afferent discharge from one part of a macula, while reducing the afferent discharge from another portion of the same macula. This extra redundancy (compared to the SCC) probably makes the otoliths less vulnerable to unilateral vestibular lesions.

The Vestibular Nerve

Vestibular nerve fibers are the afferent projections from the bipolar neurons of Scarpa's (vestibular) ganglion. The vestibular nerve transmits afferent signals from the labyrinths along its course through the internal auditory canal (IAC). In addition to the vestibular nerve, the IAC contains the cochlear nerve (hearing), the facial nerve, the nervus intermedius (a branch of the facial nerve, which carries facial sensation), and the labyrinthine artery. The IAC travels through the dense petrous portion of the temporal bone to open into the posterior fossa at the level of the pons. Note that "petrous" is derived from the Latin word *petrosus*, meaning "stone-like, hard," and thus the inner ear is very well protected! The vestibular nerve enters the brainstem at the pontomedullary junction. Because the vestibular nerve is interposed between the labyrinth and the brainstem, some authorities consider this nerve a peripheral structure, whereas others consider it a central structure. We consider it a peripheral structure.

There are two patterns of firing in vestibular afferent neurons. Regular afferents usually have a tonic rate and little variability in interspike intervals. Irregular afferents often show no firing at rest and when stimulated by head motion develop highly variable interspike intervals.[9] Regular afferents appear to be the most important type for the VOR; irregular afferents can be turned off with electrical stimulation without much change in the VOR of monkeys.[10] Irregular afferents may be important for the VSR and in coordinating responses between the otoliths and canals.

Regular afferents of the monkey have tonic firing rates of about 90 spikes per second and sensitivity to head velocity of about 0.5 spikes per degree per second.[11,12] We can speculate about what happens immediately after a sudden change in head velocity. Humans can easily move their heads at velocities exceeding 300 deg/sec. As noted previously, the SCC are connected in push-pull, so that one side is always being inhibited while the other is being excited. Given the sensitivity and tonic rate noted earlier, the vestibular nerve that is being inhibited should be driven to a firing rate of 0 spikes per second for head velocities of only 180° per second! In other words, head velocities greater than 180° per second may be unquantifiable by half of the vestibular system. This cutoff behavior has been advanced as the explanation for *Ewald's second law*, which says that responses to rotations that excite a canal are greater than responses for rotation that inhibits a canal.[13,14] Cutoff behavior explains why patients with unilateral vestibular loss avoid head motion toward the side of their lesion. More is said about this when we discuss how the central nervous system may compensate for overload.

Central Processing of Vestibular Input

There are two main targets for vestibular input from primary afferents: the vestibular nuclear complex and the cerebellum (see Fig. 1.1). The vestibular nuclear complex

is the primary processor of v estibular input and implements direct, fast connections between incoming afferent information and motor output neurons. The cerebellum is the main adaptive processor—it monitors vestibular performance and readjusts central v estibular processing if necessary. At both locations, vestibular sensory input is processed in association with somatosensory and visual sensory input.

Vestibular Nucleus

The vestibular nuclear complex consists of four major nuclei (superior, medial, lateral, and descending) and at least seven minor nuclei (Fig. 1.10). This large structure, located primarily within the pons, also extends caudally into the medulla. The superior and medial vestibular nuclei are relays for the VOR. The medial vestibular nucleus is also involved in vestibulospinal reflexes and coordinates head and eye movements that occur together . The lateral vestibular nucleus is the principal nucleus for the vestibulospinal reflex. The descending nucleus is connected to all the other nuclei and the cerebellum b ut has no primary outflow of its own. The vestibular nuclei between the two sides of the brainstem are laced together via a system of commissures, which are mutually inhibitory. The commissures allow information to be shared between the two sides of the brainstem and implement the push-pull pairing of canals discussed earlier.[15]

In the vestibular nuclear complex, processing of the vestibular sensory input occurs concurrently with the processing of extravestibular sensory information (proprioceptive, visual, and efferent (see Fig. 1.1). Extensive connections between the vestibular nuclear complex, cerebellum, ocular motor nuclei, and brainstem reticular activating systems are required to formulate appropriately oriented and timed signals to the VOR and VSR effector organs, the extraocular and skeletal muscles.

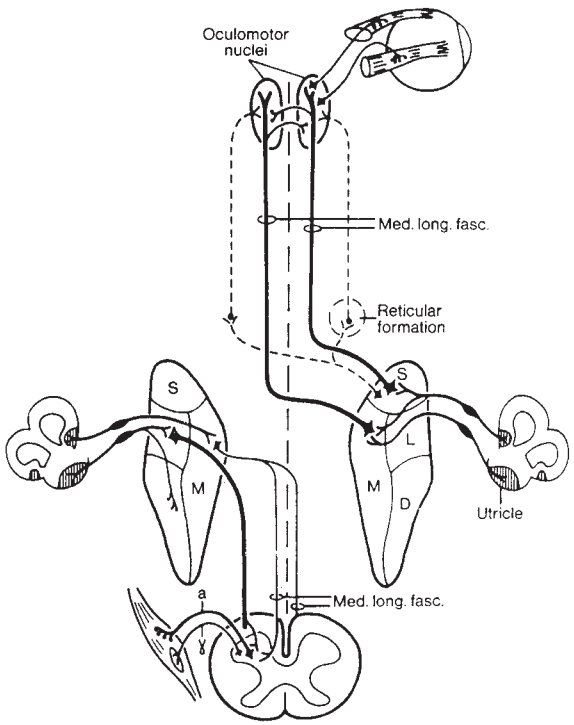

Figure 1.10 The vestibulo-ocular reflex (VOR) and vestibulospinal reflex (VSR) arcs. S, L, M, and D indicate the superior, lateral, medial, and descending vestibular nuclei, respectively. The lateral vestibulospinal and medial vestibulospinal tracts are shown as heavy lines and light lines, beginning in the lateral vestibular nucleus and medial vestibular nucleus, respectively.[15]

Vascular Supply

The vertebral-basilar arterial system supplies blood to the peripheral and central vestibular system (Fig. 1.11). The posterior-inferior cerebellar arteries (PICAs) branch of f the vertebral arteries. The two PICAs are the most important arteries for the central vestibular system. They supply the surface of the inferior portions of the cerebellar hemispheres as well as the dorsolateral medulla, which includes the inferior aspects of the vestibular nuclear complex. The basilar artery is the principal artery of the pons.The basilar artery supplies central vestibular structures via perforator branches (which penetrate the medial pons), short circumferential branches (which supply the anterolateral aspect of the pons), and long circumferential branches (which supply the dorsolateral pons). The AICA is an important branch of the basilar artery because it is the sole blood supply for the peripheral v estibular system via the labyrinthine artery. The AICA also supplies blood to the ventrolateral cerebellum and the lateral te gmentum of the lower two-thirds of the pons.

Recognizable clinical syndromes with v estibular components may appear after occlusions of the basilar artery, labyrinthine artery, AICA, or PICA. PICA territory strokes, also called "lateral medullary syndrome," cause purely central balance symptoms as a result of damage to the vestibular nucleus and inferior cerebellum. AICA territory strokes cause a mixed peripheral/cerebellar pattern because AICA supplies both the labyrinth and part of the cerebellum.

Cerebellum

The cerebellum (Fig 1.12) is a major recipient of outflow from the vestibular nucleus complex and is also a major