Balance Function Assessment and Management

Second Edition

Gary P. Jacobson, PhD
Neil T. Shepard, PhD

PLURAL PUBLISHING INC.

CONTENTS

LIST OF VIDEOS

To access the videos, please visit the companion website.

PREFACE

Why do a second edition? Subsequent editions make it possible to keep a textbook contemporary. This may mean that some topics are removed while others are added. Much has transpired in the area of clinical vestibular sciences and balance since publication of the first edition of this book. For instance, both ocular vestibular-evoked myogenic potentials (oVEMPs), and the video head impulse test (vHIT) have been added to the tools available to the clinical neurophysiologist. To these contributions we have added content describing topics as diverse as the ontogeny of the vestibular system, the effects of age on balance function, compensatory mechanisms following unilateral peripheral vestibular system impairment, and techniques for assessing vestibular system function in children. These chapters have been authored by national and internationally known experts. It is our hope that this updated text will become even more useful than it is currently.

The editors are grateful to the new and returning authors for their outstanding contributions to the 2nd edition of *Balance Function Assessment and Management*.

Last, the editors would like to thank, once again, our families for providing us with the time required to complete this work.

1

Practical Anatomy and Physiology of the Vestibular System

Michael C. Schubert and Neil T. Shepard

INTRODUCTION

The vestibular system is responsible for sensing motion of the head to maintain postural control and stability of images on the fovea of the retina during that motion. When functioning normally, the vestibular receptors in the inner ear provide amazing precision in the representation of head motion in three dimensions. This information is then used by the central vestibular pathways to control reflexes and perceptions that are mediated by the vestibular system. Disorders of vestibular function result in abnormalities in these reflexes and lead to sensations that reflect abnormal information about motion from the vestibular receptors.

Normal activities of daily life (such as running) can have head velocities of up to 550 degrees per second, head accelerations of up to 6000 degrees per square second, and frequency content of head motion from <1 to 20 Hz (Das, Zivotofsky, DiScenna, & Leigh, 1995; Grossman, Leigh, Abel, Lanska, & Thurston, 1988). Only the vestibular system can detect head motion over this range of velocity, acceleration, and frequency (Waespe & Henn, 1987). Additionally, the latency of the vestibulo-ocular reflex (VOR) has been reported to be as short as 5 to 7 ms (Huterer & Cullen 2002; Minor, Lasker, Backous, & Hullar 1999). As a result the vestibular system remains critical not only for detection of head motion, but generation of the appropriate motor command to represent that head motion.

This chapter reviews the anatomy and physiology of the vestibular system and offers examples of how the neurophysiology of the vestibular system can be examined practically.

PERIPHERAL VESTIBULAR ANATOMY

Within the petrous portion of each temporal bone lies the membranous vestibular labyrinth. Each labyrinth contains five neural structures that detect head acceleration: three semicircular canals and two otolith organs (Figure 1–1). Three semicircular canals (SCC) (horizontal, anterior, and posterior) respond to angular acceleration and are approximately orthogonal with respect to each other. Alignment of the SCCs in the temporal bone is such that each canal has a contralateral coplanar mate. The horizontal canals form a coplanar or functional pair, whereas the posterior and contralateral anterior SCC form coplanar or functional pairs. The anterior aspect of the lateral SCC is inclined approximately 20 degrees upward from a plane connecting the boney external auditory canal to the floor of the boney rim of the orbit—Reid's baseline (Della Santina, Potyagaylo, Migliaccio, Minor, & Carey, 2005). The orientation of

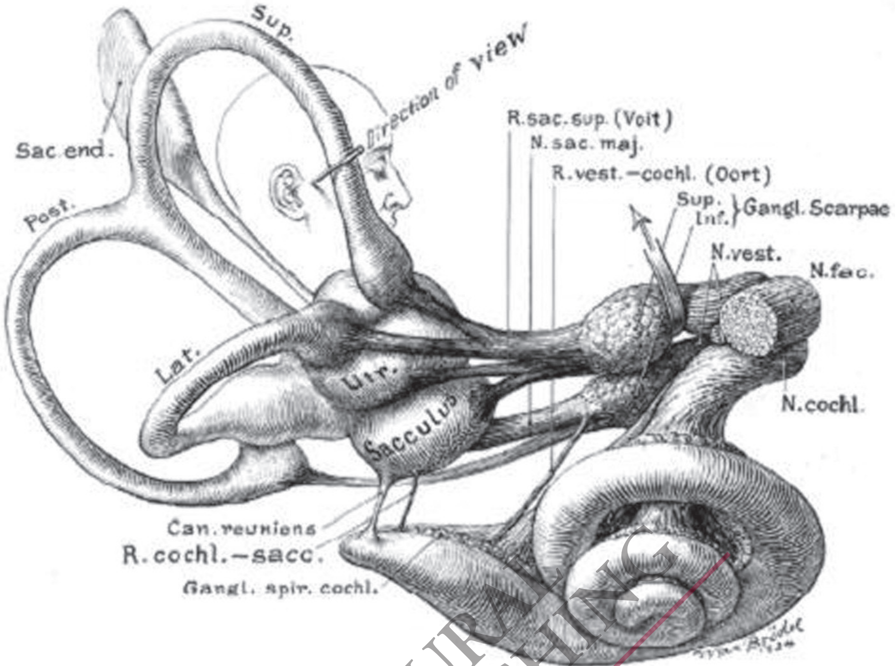

Figure 1–1. Anatomy of the vestibular labyrinth. Structures include the utricle (Utr.), sacculus, anterior (or superior) semicircular canal (Sup.), posterior semicircular canal (Post.), and the lateral (or horizontal) semicircular canal (Lat.). Note the superior vestibular nerve innervating the anterior and lateral semicircular canals as well as the utricle. The inferior vestibular nerve innervates the posterior semicircular canal and the saccule. The cell bodies of the vestibular nerves are located in Scarpa's ganglion (Gangl. Scarpae). Drawing from original art in the Max Brödel Archives (No. 933), Department of Art as Applied to Medicine, The Johns Hopkins University School of Medicine. http://www.hopkinsmedicine.org/medart/history/Archives.html and from M. C. Schubert and L. B. Minor, Vestibulo-Ocular Physiology Underlying Vestibular Hypofunction. *Physical Therapy*, (2004), *84*(4), 373–385, with permission of the American Physical Therapy Association. This material is copyrighted, and any further reproduction or distribution is prohibited.

the vestibular labyrinth with respect to the skull is shown in Figure 1–2.

The posterior and anterior SCCs are inclined about 92 and 90 degrees from the plane of the horizontal SCC (Della Santina et al., 2005). Specifically the range of angles between the planes of the canals is quite large. The angle between the horizontal-posterior planes ranges from 75.8 to 98.0 degrees; the range between the horizontal-anterior planes is 77.0 to 98.4 degrees; and for the posterior-anterior planes the range is 75.8 to 100.1 (Bradshaw et al., 2010). As a result of the anatomical variations the SCCs are not precisely orthogonal with earth vertical or earth horizontal, and angular rotation of the head stimulates each canal to varying degrees (Cremer et al., 1998). There are clinical implications for the treatment of benign paroxysmal positional vertigo (BPPV) as all of the maneuvers are based on the premise that the SCC canals are orthogonal to one another and the vertical canals are at 45-degree angles to the midsagittal plane. When the maneuver say for posterior canal BPPV is not effective on the first or second try, slight reorientation of the head relative to the midsagittal plane and the earth horizontal plane should be considered (i.e., the amount of head rotation may need to be modified) . The SCCs are filled with endolymph that has a density slightly greater than that of water. Endolymph contains a high concentration of potassium, with a lower concentration of sodium (Smith, Lowry, & Wu, 1965).

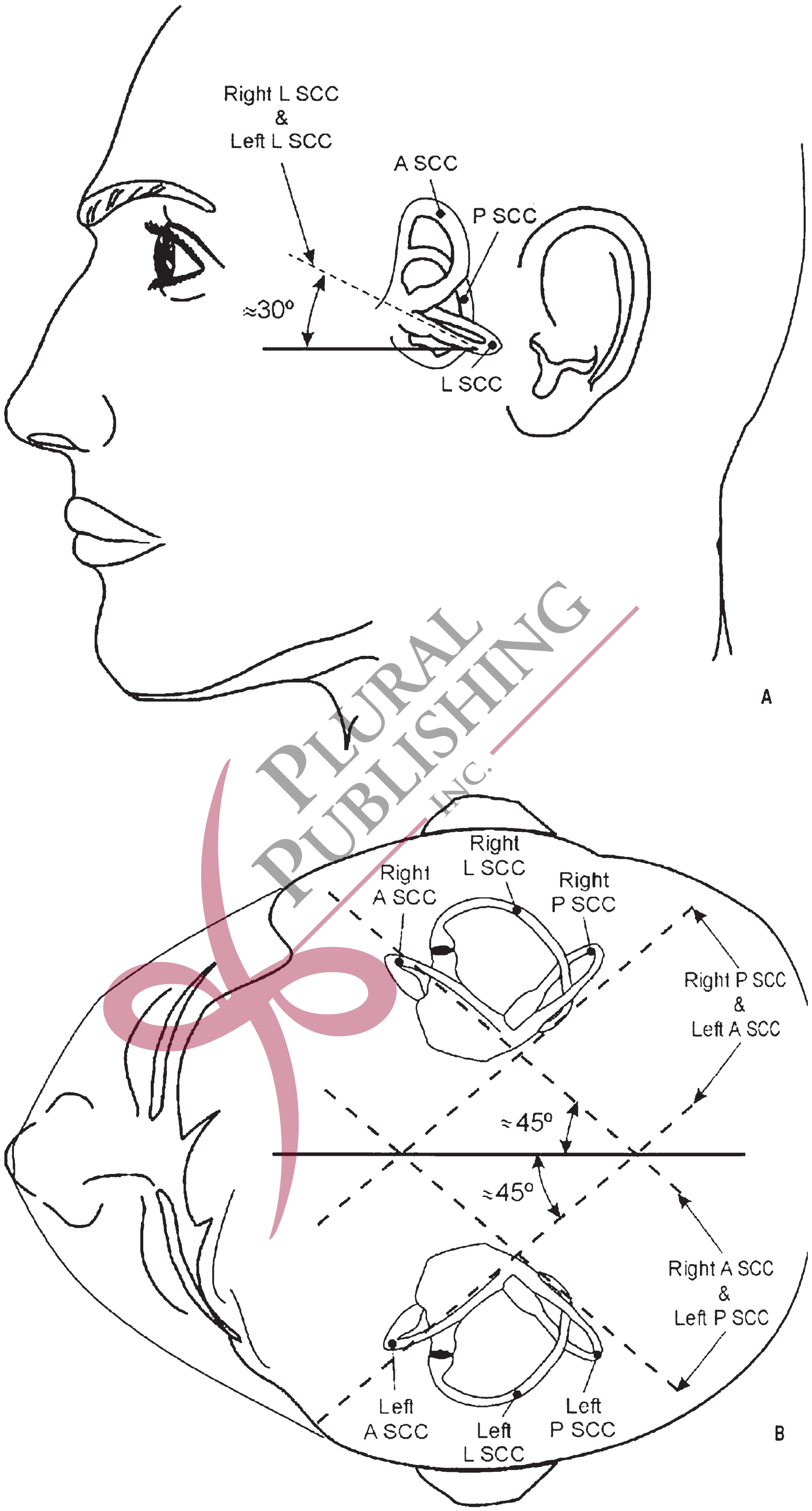

Figure 1–2. **A.** The line drawing shows the sagittal view of the head with the boney vestibular labyrinth ghosted into the position in the skull for the left ear. Reid's baseline, an anatomical landmark used in radiography has been drawn in on the figure for reference. **B.** The line drawing shows both right and left vestibular labyrinths ghosted on a horizontal axial plane view of the skull. The presumed angle orientations are shown. *L SCC* = lateral semicircular canal; *P SCC* = posterior semicircular canal; *A SCC* = anterior semicircular canal. Reprinted with permission.

The SCCs enlarge at one end to form the ampulla. Within the ampulla, a gelatinous goblet-shaped structure called the cupula serves as a barrier separating the semicircular canal from the vestibule. The cupula extends completely across the lumen of the semicircular canal filling the full cross section of the ampulla (Hillman & McLaren, 1979; Scherer, 2001), and data suggest the apex is attached by glycosaminoglycans (Holman, Tran, Arungundram, Balagurumathan, & Rabbitt, 2012; Holman et al., 2013). The cupula has a specific gravity that is equal to that of the endolymph. As a result, the cupula is not responsive to static position changes of the head in the gravitational field. Positioned beneath the cupula is the crista, which extends perpendicularly across the canal in a saddle shape. The crista contains the supporting cells and the cell bodies, commonly known as hair cells, of the stereocilia and the single kinocillium on each of the hair cells, as well as the vestibular afferents (Figure 1–3).

Kinocilia and stereocilia extend into the cupula and are the physical structures that respond to cupular deformation (Figure 1–4). If you remove a cupula and view the underneath surface you find that it is perforated with openings ranging from 3 to 5 microns in diameter. This is the same size range as that of the hair cell bundles of kinocilia and stereocilia (Harada, 1988). These openings in the base of the cupula represent a channel that runs the length of the cupula to allow for circulation of the endolymph to contact the surface of the hair cells (the cuticular plate) into which the stereocilia extend (Lim & Anniko, 1985). Deformation of the cupula occurs from attempted motion of the endolymph (the cupula occludes the canal so continual endolymph current flow is not possible), which enables a billowing (like a sail) of the central portion of the cupula with minimal movement at base on the crista or at the apex. This billowing occurs from the pressure differential that results from the attempted endolymph flow movement. Due to the maximum cupular deformation occurring in the center, a maximum shear force is created at the surface of the crista (Yamauchi et al., 2002). This results in the movement of the stereocilia bundle that causes opening (or closing) of the transduction channels of hair cells,

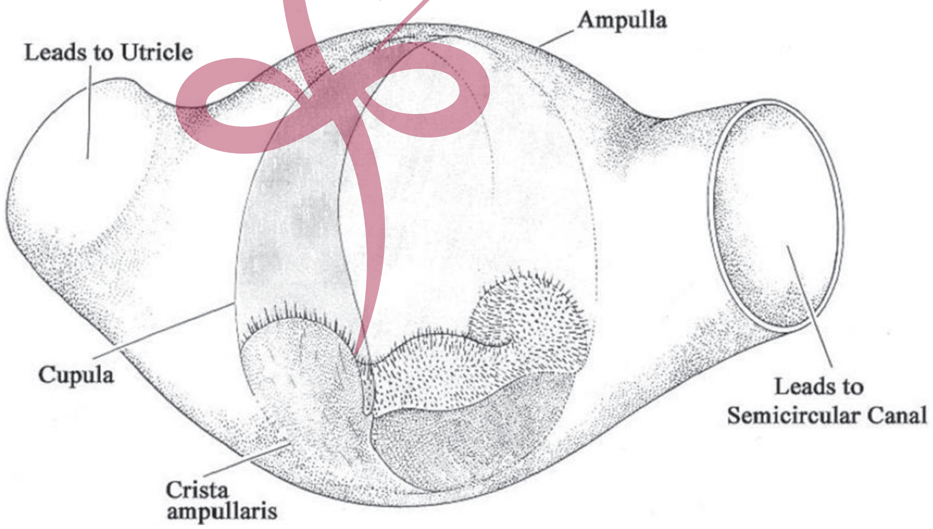

Figure 1–3. The semicircular canals enlarge at one end to form the ampulla. The cupula of the ampulla is a flexible barrier that partitions the canal. The crista ampullaris contains the sensory hair cells. The hair cells generate action potentials in response to cupular deflection. Drawing adapted with permission from Patricia Wynne (patriciawynne.com). Reprinted from M. C. Schubert and L. B. Minor, Vestibulo-Ocular Physiology Underlying Vestibular Hypofunction. *Physical Therapy*, (2004), *84*(4), 373–385, with permission of the American Physical Therapy Association. This material is copyrighted, and any further reproduction or distribution is prohibited.

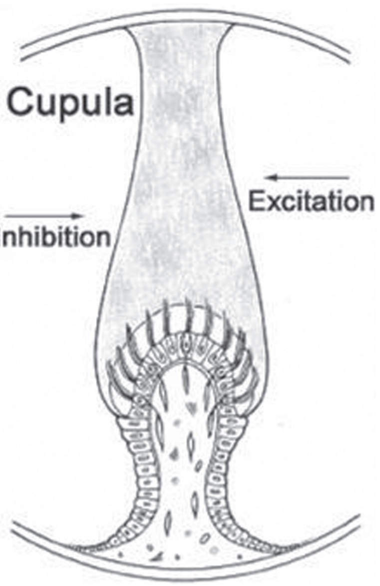

Figure 1–4. Cross section of crista ampullaris showing kinocilia and stereocilia of hair cells projecting into the cupula. Deflection of the stereocilia toward the kinocilia causes excitation; deflection in the opposite direction causes inhibition. Drawing adapted with permission from Patricia Wynne (patriciawynne.com). Reprinted from M. C. Schubert and L. B. Minor, Vestibulo-Ocular Physiology Underlying Vestibular Hypofunction. *Physical Therapy*, (2004), *84*(4), 373–385, with permission of the American Physical Therapy Association. This material is copyrighted, and any further reproduction or distribution is prohibited.

which changes the membrane potential of the hair cells. Deflection of the stereocilia toward the single kinocilia in each hair cell leads to excitation (depolarization), whereas motion of the stereocilia away from the kinocilia causes inhibition (hyperpolarization). Hair cells are oriented in the horizontal SCC so that endolymph motion toward the ampulla (utricle) causes excitation. In contrast, hair cells of the vertical SCCs (posterior and anterior) are oriented so that depolarization occurs when endolymph moves *away* from the ampulla (utricle). Having the hair cells oriented in this manner is referred to as morphologic polarization. Figure 1–5 diagrammatically illustrates this by taking the surface of the crista, flattening it, and looking down on it from above. This is shown for both the horizontal canal in part A and for the two vertical canals in part B. Each of the SCCs responds best to motion in its own plane, with coplanar pairs exhibiting a push-pull dynamic. For example, as the head is turned to the right, the hair cells in the right horizontal SCC are excited, whereas the hair cells in the left horizontal SCC are inhibited (Goldberg & Fernandez, 1971). The brain detects the direction of head movement by comparing input from the coplanar labyrinthine mates.

The saccule and utricle make up the otolith organs of the membranous labyrinth. Sensory hair cells project into a gelatinous material that has calcium carbonate crystals (otoconia) embedded in it, which provide the otolith organs with an inertial mass (Figure 1–6). The presence of the otoconia increases the specific gravity above that of the endolymph. As a result, the maculae (the surfaces of the otolithic organs that contain the hair cells) are responsive to linear acceleration, including the force of gravity as the head is placed in different static positions. The utricle and the saccule have central regions known as the striola, dividing the otolith organs into two parts. This division is used to set up the morphologic polarization for the otolith organs. The kinocilia of the utricular hair cells are oriented toward their striola, whereas the kinocilia of the saccular hair cells are oriented away from their striola. Motion toward the kinocilia causes excitation. Utricular excitation occurs during horizontal linear acceleration or static head tilt, and saccular excitation occurs during vertical linear acceleration. The striola is not a straight line but is a curved region; therefore, as the utricle or saccule is stimulated by a linear movement in any direction there will be a portion of the utricle (saccule) that is excited and a portion that is inhibited. In effect each of the four otolith organs is its own functional pair along with the unit in the other ear (Rabbitt, Damiano, & Grant, 2004).

The cell bodies of vestibular nerve afferents are located in the superior or inferior divisions of Scarpa's ganglia, which lie within the internal auditory canal near the emergence of the vestibular nerve into the cerebellopontine angle (Brodal, 1981). From the vestibular labyrinth, the afferent information travels ipsilateral in one of two branches of the vestibular nerve. The superior vestibular nerve innervates the horizontal and anterior SCC as well as the utricle. The inferior vestibular nerve innervates the posterior SCC and the saccule (Naito, Newman, Lee, Beykirch, & Honrunbia, 1995). The posterior canal has been reported to have a double innervation; therefore, it may have branches from both superior and inferior