Aging of the Human Vestibular System

Christopher K. Zalewski, Ph.D.[1]

ABSTRACT

Aging affects every sensory system in the body, including the vestibular system. Although its impact is often difficult to quantify, the deleterious impact of aging on the vestibular system is serious both medically and economically. The deterioration of the vestibular sensory end organs has been known since the 1970s; however, the measurable impact from these anatomical changes remains elusive. Tests of vestibular function either fall short in their ability to quantify such anatomical deterioration, or they are insensitive to the associated physiologic decline and/or central compensatory mechanisms that accompany the vestibular aging process. When compared with healthy younger individuals, a paucity of subtle differences in test results has been reported in the healthy older population, and those differences are often observed only in response to nontraditional and/or more robust stimuli. In addition, the reported differences are often clinically insignificant insomuch that the recorded physiologic responses from the elderly often fall within the wide normative response ranges identified for normal healthy adults. The damaging economic impact of such vestibular sensory decline manifests itself in an exponential increase in geriatric dizziness and a subsequent higher prevalence of injurious falls. An estimated $10 to $20 billion dollar annual cost has been reported to be associated with falls-related injuries and is the sixth leading cause of death in the elderly population, with a 20% mortality rate. With an estimated 115% increase in the geriatric population over 65 years of age by the year 2050, the number of balanced-disordered patients with a declining vestibular system is certain to reach near epidemic proportions. An understanding of the effects of age on the vestibular system is imperative if clinicians are to better manage elderly patients with balance disorders, dizziness, and vestibular disease.

KEYWORDS: Vestibular, aging, presbystasis, presbyvertigo, presbyequilibrium, geriatric dizziness

[1]Otolaryngology Branch, Audiology Unit, National Institute on Deafness and Other Communication Disorders (NIDCD), National Institutes of Health (NIH), Bethesda, Maryland.

Address for correspondence: Christopher K. Zalewski, Ph.D., 9000 Rockville Pike, Building 10, Room 5C422, Bethesda, MD 20892 (e-mail: zalewski@nidcd.nih.gov).

Cognition and the Aging Auditory System; Guest Editor, Lindsey E. Jorgensen, Au.D., Ph.D.

Semin Hear 2015;36:175–196.
DOI: http://dx.doi.org/10.1055/s-0035-1555120.
ISSN 0734-0451.

Learning Outcomes: As a result of this activity, the participant will be able to (1) describe the epidemiology of dizziness in the elderly, (2) histology of the aging vestibular system, and (3) vestibular test results associated with the aging population.

BALANCE FUNCTION AND THE ROLE OF THE VESTIBULAR SYSTEM

The vestibular system is compromised of a nexus of peripheral sensory end organs and a complex network of central neurons. The peripheral anatomy and physiology are grossly responsible for sensing the degree and direction of acceleration, as well as providing a sense of orientation of the head with respect to gravity. The central connections, including most notably the vestibular nuclei, are grossly responsible for processing the numerous sensory inputs. The accurate and ubiquitous perception of movement and self-orientation occurs, in part, because of a healthy vestibular system. In many ways, this perception is both subconscious and autonomic insomuch that it occurs without intent or self-control. On its most basic level, the vestibular system is both a sensory system as well as a motor system.[1] As a sensory system, the vestibular response not only provides an accurate representation of self motion but also is integral in constructing an "internal map" of one's center of mass in space with respect to gravity. As a motor system, the vestibular response coordinates effective postural and ocular motor reflexes to ensure static and dynamic equilibrium with respect to one's center of gravity as well as maintain visual acuity during head movement. It is a hierarchical system by which a proper motor response is heavily dependent upon accurate sensory perception.[1,2]

Although balance and equilibrium are heavily dependent upon other central nervous system processes, especially the visual and somatosensory systems, the contributions of the vestibular system are undoubtedly critical. This is particularly evident when functioning in vestibular-dependent environments where visual and somatosensory cues are compromised.[3] The importance of an accurate sensory perception of one's environment is most apparent when the vestibular system fails to translate an appropriate signal. An aberrant or absent vestibular response results in significant debilitating balance deficits and often produces an array of symptoms including dizziness, disequilibrium, vertigo, nausea, pallor, diaphoresis, general malaise, and even emesis. Depending on the severity, these symptoms can often lead to physical, mental, and even social isolation.

Although much is known about the vestibular system, there is much that remains unexplained and likely undiscovered regarding its physiology and sophisticated role in postural stability and locomotion. Its complex anatomy and multi-integrational physiology with other sensory systems presents difficulties in evaluating the vestibular system in its entirety. Conversely, evaluating discrete sensory end organs within each vestibular system also presents unique challenges. Secondary to the vestibular system's complex anatomy and physiology, vestibular disease or even subtle changes in vestibular function due to aging are often difficult to differentiate and identify. Moreover, disorders of the vestibular system are not only very prevalent but often nonspecific with regards to the precise site of the lesion. This is particularly true of the elderly for whom nonspecific dizziness ranks as the most frequent health concern reported to primary care physicians at approximately eight million visits each year.[4]

Indisputably, it is difficult to fully appreciate the senescence of human balance without considering the impact aging has on postural reflexes, the visual system, the vestibular system, and the central nervous system. However, a comprehensive discussion conveying the complete effects of how aging impacts every system is beyond the scope of this review. Rather, this review will focus on the prevalence of dizziness in the elderly and the effects of aging on the vestibular system as it relates to anatomical deterioration of the peripheral and central vestibular sensory structures, as well as the physiologic outcomes reported from standard clinical vestibular tests.

EPIDEMIOLOGY OF DIZZINESS

There exists an abundance of literature concerning the prevalence of dizziness, balance dysfunction, and the associated comorbidities related to dizziness in the elderly—namely increased falls, depression, social isolation, fear, and overall functional decline.[5] The data concerning the epidemiology of dizziness and falls in the elderly are staggering. According to Dewane,[6] two of the most frequent problems experienced by the elderly are dizziness and falls. Cutson reported the incidence of dizziness to increase from 22% for adults between 65 and 69 years of age to over 40% for adults between the ages of 80 and 84 years.[7] Hobeika estimated that 65% of individuals older than 60 years of age experience dizziness or loss of balance, often on a daily basis.[8] Data provided by the National Center for Health Statistics (NCHS) cite the prevalence of balance impairment in the United States to be 75.3% over the age of 70 years.[9]

Despite such widespread reports highlighting an overwhelming prevalence of age-related dizziness and balance impairment, the etiology of the balance impairment is often difficult to discern, as the origin of age-related dizziness is frequently multifactorial. In a review of 1194 patients aged 70 years or older, Katsarkas was uncertain of the diagnosis in ~27% of his geriatric population.[10] This is attributed to the fact that dizziness is often an obscure symptom that has been associated with various diseases affecting the central nervous system and other sensory systems, or is comorbid to other system processes such as cardiovascular disease, osteoarthritis, poor visual acuity, polypharmacy, or pharmacokinetics.[5] Baloh describes the term *dizziness* as having no specific definition except that it refers to some kind of altered orientation in space and almost always represents a complex combination of overlapping symptoms.[11] It is likely this obscurity of symptoms and comorbidities in combination with the complexity of the vestibular system's anatomy and physiology that makes it notoriously difficult to assess.

The reported estimates for the prevalence of *vestibular*-related dizziness in the elderly population are often varied. The NIDCD reports that at least half of the U.S. population is affected by a balance or vestibular disorder sometime during their lives.[12] The University of Iowa estimates that, of those individuals presenting to their primary care providers with some form of balance impairment, ~20% are specifically affected by a vestibular disorder and in ~15% of those patients the cause is unknown.[13] However, Marchetti and Whitney report that the prevalence of vestibular disease in elderly patients with a chief complaint of dizziness could be much higher.[4] They reported on the number of patients presenting to otolaryngology clinics with a primary complaint of dizziness and estimated as many as 40 to 50% of patients with dizziness have a vestibular etiology to their symptoms. The clinical link between elderly patients with dizziness and postural instability resulting in falls and vestibular impairments is high. Overall, Jacobson and colleagues argued that peripheral and central vestibular disorders are likely an underappreciated contributor to postural instability and falls in the elderly.[14]

One of the leading deleterious implications to vestibular-related dizziness in the elderly is the significant prevalence of injurious falls. According to the Center of Disease Control and Prevention, more than one-third of adults aged 65 years and older fall each year, many of which are secondary to balance problems and dizziness. When persons 80 years or older are compared with persons aged 70 to 79 years, the incidence of balance impairment increases almost 30-fold from 69.3% to an staggering 88.5% prevalence rate. These data are alarming insomuch that nearly 9 of 10 persons 80 years or older are likely to present with a balance impairment that carries a greater risk for falls and subsequent bone fracture, joint dislocation, or even severe head injury. In fact, falls due to balance impairment represent the most common mechanism of injury in the geriatric population with ~30% of community-dwelling elderly people falling at least once per year.[4] The National Safety Council reports that falls in individuals over 75 years of age are the number one cause of injury-related death.[15,16] Overall, injurious falls rank as the sixth leading cause of death in the elderly population with an estimated cost of caring for fallen elderly to be between $10 and $20 billion annually.[14] Moreover, for those elderly who sustain an injurious fall, the mortality rate is estimated to be ~20%

with an additional 20% receiving long-term center inpatient care.[17]

Epidemiology Forecast for Dizziness and Balance Disorders

The high prevalence of dizziness and falls in the elderly is a growing and immediate medical concern. According to the U.S. Census Bureau, the projected number of U.S. citizens over the age of 65 years will rise sharply from ~40 million in 2010 to nearly 90 million by the year 2050.[5] This represents a 115% projected increase largely due to the aging baby boomer population. If one were to simply project the number of Americans who will be over 85 years of age by the year 2050, the data estimate an overwhelming 20 million Americans.[5] If this population estimate is true, then according to the NCHS, ~18 million Americans are projected to have some form of balance impairment that requires medical attention. The impact of such a large geriatric population becomes even more staggering if one is to factor in the confounding effects of consequential falls and projected medical costs that will inevitably accompany this balance-impaired population. For dizziness and balance impairment to reach such epidemic proportions, the question remains as to whether the medical community will be prepared to effectively handle such an overwhelming number of balanced-impaired patients. The current projections of the number of audiologists, physicians, physical therapists, occupational therapists, and kinesiologists working in clinical vestibular environments would suggest a disproportionate patient-to-provider ratio, with the number of patients far outweighing the number of clinical providers.[18]

OVERVIEW OF THE VESTIBULAR SENSORY HISTOLOGY

As recently as 40 years ago, a great deal of controversy existed regarding whether age-related degeneration existed within the vestibular system.[19] Although this controversy seems implausible, a decided lack of physiologic evidence from clinical testing of older patients failed to support histologic reports showing as much as 40% loss of vestibular sensory cells by the ninth decade of life.[19] In fact, Rosenhall indicated that the correlation between clinical and histologic findings is consistently poor.[20] Nevertheless, marked age-related degeneration has been observed in nearly every type of vestibular-related cell and neuron bundle.[19,21–26] The apparent lack of clinical evidence to support such histologic reports is believed to be secondary to the complex physiology of the central vestibular system and the supporting postural reflex system.[21,22,24,27] Current theory supports effective central vestibular compensation mechanisms that mask the underlying peripheral sensory degeneration.[23,25] Despite more advanced assessment methods and more sensitive clinical equipment, results repeatedly fail to identify any strong correlations between clinical evidence and histologic degeneration.[21–26]

Histology Sensory Degeneration Literature Review

As early as the 1970s, histologic reports have documented age-related sensory degeneration throughout the vestibular system.[25] The term *presbyvertigo* has been used to describe such age-related changes within the vestibular system, similar to that of presbycusis for hearing.[26] Various reports have identified significant degeneration in nearly all sensory components of the vestibular system. Although the age of onset and rate of decline is somewhat disputed across reports, near universal agreement has documented significant degeneration in nearly all types of vestibular cells, including the sensory end organ hair cells, the nerve fibers, Scarpa ganglion cells, vestibular nucleus neurons, and even a significant decline in the number of Purkinje cells within the cerebellum.[21–25,27–30]

AGE-RELATED REPORTS OF VESTIBULAR HAIR CELL LOSS

The majority of the histologic reports investigating vestibular hair cell degeneration confirm a general onset of significant hair cell decline between 65 and 70 years of age. Early microdissections of the vestibular epithelium by Rosenhall and Rubin as early as 1975 concluded that end organ hair cell counts remain relatively stable until 70 years of age, after which a gradual decline is observed.[31] However, degeneration

rates as well as specific age of onset can vary widely with respect to the precise sensory organs, cells, or neurons. Jang and colleagues identified saccular otoconia destruction as early as age 50 years of age that increases in severity with advancing age.[29] Rosenhall identified a large, marked reduction in hair cell counts within the cristae of the semicircular canals as early as 50 to 60 years of age.[20] Richter identified a significant reduction in the number of vestibular ganglion cells as early as 60 years of age,[32] and Bergström identified a significant reduction in vestibular nerve fibers by the fifth decade of life that increased to a 40% loss of fiber counts in individuals 75 to 80 years of age.[33,34] Moreover, Lopez et al reported a 3% neuronal loss per decade within the vestibular nuclei that begins at ~40 years of age.[35] Finally, Alverez and colleagues reported a near 40% loss of neuronal cells within the vestibular nuclei by 89 years of age that mainly impacted the medial vestibular nucleus.[36]

HISTOLOGIC STUDIES OF VESTIBULAR END ORGAN DEGENERATION

In one of the most comprehensive and specific vestibular hair cell histology studies published, Merchant and colleagues utilized Nomarski differential interference contrast microscopy to determine the total number of vestibular hair cells in each of the five vestibular sensory end organs.[37] The authors determined hair cell counts in a series of 67 normal temporal bones ranging in age from birth to 100 years of age. They identified a strong age-related decrease in vestibular hair cell counts for all sensory end organs with a significantly greater loss of hair cell density in the cristae than in the maculae. Merchant and colleagues also determined hair cell densities were greater at the periphery than the central regions of cristae, particularly for type I hair cells. There was no significant difference in hair cell loss between the three semicircular canals, the two maculae, men and women, or right versus left ears.

Rauch and colleagues also reported on decreasing vestibular hair cell counts in the aging human.[27] Similar to Merchant and colleagues, their report cites a highly significant continuous age-related decrease in all vestibular sensory hair cell counts with a propensity for a more rapid loss of sensory hair cells in the cristae than in the maculae that is also hair cell-type dependent. Degeneration rates have also been reported by Velázquez-Villaseñor and colleagues as well as by Rosenthal.[20,38] Velázquez-Villaseñor and colleagues identified a linear decrease in the population of vestibular neurons in Scarpa ganglion from birth to 100 years of age.[38] Rosenhall reported a gradual loss of sensory cells and primary neurons beginning at ~40 years of age, after which a linear degeneration occurs until there is approximately 40% total loss of vestibular sensory cells by 75 years of age.[20]

Anniko[21] and Rosenhall[20] described a greater susceptibility for age-related degeneration that has been identified in the cristae of the semicircular canals when compared with the either the utricle or the saccule. Specifically, they also reported a greater proclivity for hair cell loss in the cristae over the maculae, as much as 40% loss of sensory hair cells in the cristae compared with 20 to 25% loss in the maculae by 70 to 95 years of age.

MACULAR DIFFERENCES IN VESTIBULAR HAIR CELL LOSS

With respect to macular differences, histologic reports have documented distinct differences in degeneration rates of vestibular hair cells between the saccule and the utricle. Rosenhall identified a 25% loss of hair cell counts in the saccule,[20] whereas Engström et al reported a loss of hair cell counts in the utricle by only 20% by 70 to 95 years of age.[39] Igarashi and colleagues later identified a larger disparity between the utricle and saccule macular epithelia.[40] They calculated a utricle-to-saccule ratio of 100:70 in a group of young subjects that degraded to a ratio of 100:35 in a group of seniors. Others have also reported a propensity for an increased rate of age-related saccular degeneration.[41–43] Although the overall reasons for otoconia degeneration are poorly understood,[29] one reason cited for this difference was offered by Johnsson and Hawkins.[43] They suggested that the saccule's vertical orientation leaves it more susceptible to loss of otoconia. One other reason offered by Thalmann and colleagues is secondary to a lack of dark cells within the saccule.[44] In a review of

otoconia in health and disease, Lim discusses the renewal of otoconia through four proposed hypotheses, all of which mitigate the process of calcite seeding from the endolymphatic sac, the dark cells of the vestibule, the maculae supporting cells, and/or from the otoconia membrane itself.[24] A collapse of this process would likely contribute to the degenerative aging process that is distinct to each macular otoconia membrane.[24]

HISTOLOGICAL STUDIES OF VESTIBULAR TYPE I VERSUS TYPE II HAIR CELL LOSS

Degeneration of sensory hair cells has also been reported to be regionally specific across all vestibular epithelia for both type I and type II hair cells. Merchant and colleagues identified a strong age-related decline in type I hair cells in all five sensory end organs.[37] Specifically, Merchant and colleagues described a loss of approximately three cells per 0.01 mm^2 per decade of life in the cristae and only one cell per 0.01 mm^2 per decade of life in the maculae. This observed difference between degeneration rates of type I hair cells between the cristae and the maculae was significantly different. The authors also reported a significant age-related decline in type II sensory hair cells of approximately one cell per 0.01 mm^2 per decade of life; however, no significant differences were identified between the cristae and the maculae.

Rosenhall further identified a general loss of hair cells over the *entire* sensory epithelia with a distinct concentration of sensory hair cell loss in the central region of the cristae and maculae where a preponderance of type I hair cells and large irregular afferent vestibular fibers are located.[20,45–47] This finding was also later confirmed by Engström et al.[39] Anniko also reported a distinct proclivity for age-related degeneration of type I hair cells. Anniko suggested the preferential degeneration of type I hair cells may be explained by phylogenesis, insomuch that type I hair cells are younger and have reached a higher degree of specialization and differentiation.[21] Anniko attributes this declination to morphologic changes that appear to be confined to type I hair cells, particularly in the cuticular plate and the apical part of the cell. He hypothesized that these changes may be secondary to a proliferation of rodlike inclusion bodies that were identified near the cuticular plate of type I hair cells, indicating a weak point within the cell that may be attributable to an actin-based metabolic dysfunction not present in type II hair cells. Anniko also identified pathologic changes that occurred selectively in calyx nerve endings. In his report, he detailed a considerable increase and disintegration of cristae mitochondrion. Such changes to the type I synapse could also help to explain higher selective type I irregular afferent degeneration rates.

Finally, Rauch and colleagues examined vestibular hair cell counts from 67 temporal bones across a wide age range.[27] In their report, Rauch and colleagues investigated whether a difference in degeneration rates exists between the cristae of the semicircular canals and the otoconial layer of the maculae. They also investigated whether an overall difference exists between the degeneration rates of type I versus type II hair cells. They not only reported a faster degeneration rate for type I hair cells over type II hair cells that was independent of all vestibular sensory end organs, but also identified a more rapid degeneration rate of type I hair cells within the cristae than the maculae. Regarding type II hair cells, Rauch and colleagues noted a slower degeneration rate that was independent of any sensory epithelium.

SUMMARY OF VESTIBULAR HAIR CELL HISTOLOGY AND CLINICAL RELEVANCE

There is much agreement for a significant histologic age-related deterioration in the number of type I and type II vestibular peripheral sensory hair cells, as well as for prominent vestibular sensory neurons within the vestibular nuclei and the cerebellum. This loss is highlighted by a more rapid degeneration rate of type I vestibular hair cells with a concomitant higher rate of hair cell loss for the cristae than the maculae; which tends to be greater in the central epithelial regions of the sensory end organs than the periphery. Most reports agree that the loss is most prominent toward the seventh to eighth decade of life, which is also around the time when many sexagenarians and septuagenarians begin reporting the onset of unrelenting dizziness and functional balance problems.

Independent of the difference in the rates for losses of type I and type II hair cells, the specific loss of type I vestibular hair cells, and particularly those in the center region of the epithelium, may have significant clinical relevance. This finding is significant as it relates to one plausible explanation to a lack of correlative clinical evidence failing to demonstrate a significant age-related loss of the angular-vestibular ocular reflex (VOR).[25] Although Minor and Goldberg have reported a near inconsequential impact on the VOR following ablation of type I hair cells and associated irregular afferent nerve fibers,[48] Tang and Woollacott describe a possible association between the distinct age-related loss of type I hair cells and vague complaints of postural instability in older patients.[49] It is not implausible to conceive a subsequent deleterious impact on the delicate phase relationship between functional and dysfunction vestibular hair cell responses and an intact VOR. Tang and Woollacott postulated that such associations might be secondary to "age-related declines in the ability to correctly detect head position and motion in space, to elicit vestibular spinal reflexes, or to solve sensory conflicts."[49(p. 390)] Similar subtle balance complaints have also been reported in patients for whom the only abnormality is a loss of low-frequency VOR phase lead.[50]

Despite such evidence detailing the overall degeneration of the various vestibular sensory end organs, the confirmation of such histologic changes during routine clinical assessment lacks any strong correlation. Many vestibular studies on aging have failed to identify any significant physiologic evidence that is temporally linked to such histologic declines. In fact, some clinical measures have paradoxically reported an *increase* in VOR response rate at a time when histologic reports convey the contrary.[51] The results of objective vestibular studies investigating the clinical outcomes in the aging population will now be reviewed.

TESTS OF VESTIBULAR FUNCTION

Assessment of the vestibular system, whether of the peripheral structures or central connections, is best accomplished through a series of tests that collectively and individually evaluate the various vestibular structures or, more specifically, indirect physiologic reflexes. Balance function tests can be grossly divided into two primary categories: tests that investigate the VOR and those that investigate the vestibular spinal reflex. Standard clinical tests that investigate the VOR would include videonystagmography, rotational vestibular testing, ocular vestibular evoked myogenic potential (oVEMP) testing, and video head impulse testing (vHIT). Tests that investigate the vestibular spinal reflex would include computerized dynamic platform posturography, and cervical vestibular evoked myogenic potential (cVEMP) testing.

The subtle effects of age on test results are both widespread and highly variable across individuals with either marginal clinical significance or no measurable declines reported on nearly every balance function test.[52] Consideration of the effect of age on each test is pertinent to the clinical understanding of geriatric imbalance. However, on a grander scale, it is imperative to understand that the physiologic decline of the vestibular system as measured by each test is often subtle, small, and highly variable, which is often starkly juxtaposed against the anamnesis surrounding a patient's balance complaint. That is, the geriatric vestibular phenotype is rarely confirmed by frank vestibular test results, and, at times, is even poorly depicted by *comprehensive* vestibular test results. Nevertheless, an understanding of the effect of age, as well as having a better appreciation of each test's limitations, is critical when understanding the relationship between the elderly balance-disordered patient and their clinical findings.

Videonystagmography

Videonystagmography testing is the most common vestibular function test and can be divided into ocular motor, positional, and caloric testing. Age-related changes have been widely reported for tests of ocular motor function, caloric testing, and the Dix-Hallpike test (i.e., identification of benign paroxysmal positional vertigo [BPPV]); however, a dearth of literature exists on the effects of aging on static positional nystagmus.[53] Secondary to the increased deterioration rates of the maculae epithelium as discussed earlier, there exists a well-known

higher incidence of BPPV in the elderly. Although this review will not discuss the significant increase of BPPV in the elderly, the reader is nevertheless alerted to the subsequent higher incidence of abnormal Dix-Hallpike maneuvers in the aged population.

OCULAR MOTOR TESTING

Much of the literature on the effects of aging on ocular motor function highlights changes in saccadic tracking, smooth pursuit tracking and optokinetic nystagmus (OKN) reflex. Kerber and colleagues reported on a 9- to 12-year longitudinal study of ocular motor function in healthy older individuals over 75 years of age.[54] In this study, the authors cited a significant decline in saccadic latency but failed to find evidence for a significant decline in saccadic velocity. Although Kerber and colleagues reported a trend suggesting a decline in peak eye velocity for 30-degree target displacements, the authors indicated these data failed to reach statistical significance. Although they reported a similar trend for declining smooth pursuit gain, they did identify a statistically significant decrease in OKN gain for older individuals. Kerber and his colleagues thought the lack of widespread significance was likely due to the limited follow-up for their cohort and the subsequent lack of significant sensory deterioration during this time. They speculated that measurable physiologic changes are more apt to be observed when comparing data from older patients with those of middle-aged or young adults. Interestingly, the authors did find significant declines in performance for nearly all measures of ocular motor function when they compared the data against the final visit from a subgroup of elderly patients who died prior to their completion in the 12-year study. They speculated this may have been due to a subnormal aging processes in a group of patients with subclinical disease that were less healthy than those able to complete the study.

Irving and colleagues investigated the dynamics of age on the production of saccades across the human life span.[55] They analyzed saccadic performance from 195 individuals aged 3 to 86 years. Overall, Irving and colleagues identified an impact of age on all parameters of saccadic tracking: latency, velocity, and accuracy. Specifically, the authors identified an increase in hypometric saccades as age and size of the saccade increased. They further identified an increase in saccadic peak eye velocity from 3 to 14 years of age (with a significant peak eye velocity occurring between 10 and 15 years of age) followed by a gradual decrease through 86 years of age. The results for saccadic latency were more dynamic, showing a decrease in latency from 3 to 14 years of age, followed by a period of relative stability until 50 years of age, before gradually increasing in latency through 86 years of age.

In a more recent study, Peltsch et al evaluated saccadic function in 81 normal healthy elderly subjects (60 to 85 years) in five different age groups delineated by one-half decade intervals.[56] They identified slower initiation of saccades, more directional errors, and increased reaction times for their oldest group when compared with their two youngest subject groups (60 to 64 years, and 65 to 69 years). They further evaluated the difference between voluntary saccade control (antisaccade paradigm) and automatic saccade control (prosaccade paradigm). They identified a greater impact of aging on the mechanism that requires voluntary saccadic control and considered the possibility that different neural substrates and aging processes are likely responsible for this observed difference.

Tuunainen and others analyzed the ocular motor function, vestibular ocular reflex, and postural deficits in 38 individuals between the ages of 80 and 103 years (mean age 89 years).[57] Although specific deficits were not provided, these authors identified 26 of 34 (76%) participants with abnormal saccades and 17 of 20 (85%) with abnormal smooth pursuit. They did highlight a significant correlation between a reduction in smooth pursuit and prolonged saccadic latency, but they did not identify any data regarding reduction in pursuit gain or saccadic velocity.

Sharpe and Sylvester compared the smooth pursuit function between young (19 to 32 years) and old (65 to 77 years) healthy participants.[58] They concluded that smooth pursuit tracking is an age-dependent system characterized by a reduction in pursuit gain and an increase in pursuit initiation latency for elderly individuals.

Sharpe and Sylvester speculated the decline of pursuit function was likely secondary to cerebral cortical atrophy, loss of cerebellar Purkinje cells, and degeneration of extraocular muscle tissue. Spooner, Sakala, and Baloh later confirmed Sharpe and Sylvester's conclusions for an age-dependent smooth pursuit system.[59] However, Spooner and colleagues also provided evidence to extend Sharpe and Sylvester's conclusions to include an age-dependency for the saccadic and OKN systems. They further added a point of caution including a statement on the increased variability that appears to be present for all ocular motor measures with senescence. They cautioned that such an increase in variance can be problematic when using data from individuals over 60 to 70 years of age when determining normative reference ranges for younger populations, as this can significantly reduce the sensitivity of such measures. Moreover, the possible unintended inclusion of data from elderly subjects with subclinical disease would undoubtedly further distort a reference range that accurately reflects the "normal" aging process, particularly for patients over 70 years of age.[54]

Jagacinski et al evaluated smooth pursuit function in a group of older participants (60 to 69 years) and a group of younger participants (18 to 25 years).[60] Their data also provide evidence to corroborate an age-dependency for smooth pursuit tracking. Additionally, Morrow and Sharpe showed presaccadic pursuit velocity deficits, lower postsaccadic pursuit velocities, and reduced peak pursuit velocities in their older healthy volunteers (60 to 76 years) when compared with their younger healthy group (29 to 35 years).[61] Finally, in a report by Kanayama and colleagues, smooth pursuit gain was compared among four groups of healthy volunteers: young (23 to 33 years), fifties, sixties, and seventies and older.[62] Smooth pursuit was noted to decline with advancing age for all stimulus frequencies and target velocities. An effect of pursuit saturation was also seen in the oldest group at a stimulus frequency of 60 degrees per second that was not observed in any other age group.

Studies investigating the effect of age on OKN reflex nystagmus have also identified significant decreases in response peak eye velocity gain. Kato and colleagues identified a significant decrease in linear stimulus-induced slow-phase OKN velocity between a group of young (30 to 59 years) and older (70 to 79 years) healthy volunteers.[63] In a comprehensive study performed earlier by Simons and Büttner, OKN nystagmus was observed to decrease considerably with advancing age.[64] These authors identified a progressive decline in the maximal OKN response from 114 to 93 to 73 degrees per second across three age groups: 20 to 39 years, 40 to 59 years, and 60 to 82 years, respectively. They further reported a significant decrease in smooth pursuit gains across age groups but noted a more pronounced decrease in maximal OKN velocity with a maximal decrease in OKN velocity by ~1 degree per second every year. Other reports have also documented abnormalities in OKN function (increased latency to circular vection, increased rate of OKN asymmetry, and decreasing torsional OKN) with advancing age.[19,53,65]

Finally, Calder provides a good summary on the effects of aging for various ocular motor tests from numerous published reports, which are summarized in Table 1.[52] Overall, prolonged saccadic latencies and reduced smooth pursuit and OKN gains are observed in elderly patients when compared against younger adults.

In summary, many studies on ocular motor function cite progressive declines in saccade, smooth pursuit, and OKN function, particularly over 60 to 70 years of age. Declines in ocular motor function produce subtle consequences of retinal slip and inability for gaze fixation during head movement. Such ocular motor deficits are often manifested as a functional dizziness and postural instability, which are often exacerbated in patients greater than 70 years of age when compounded by subtle age-related VOR deficits and less effective adaptive central plasticity.[70,71] Finally, many studies further agree that smaller standard deviations are often seen with younger populations (possibly due to the increased presence of subclinical vestibular disease in the older population), and the need for age-appropriate ocular motor normative reference ranges is critical when interpreting clinical studies, particularly in the oldest of the old.[72]

Table 1 Age-Related Ocular Motor Findings (adapted from Calder[52])

Ocular Motor Function	Clinical Findings	Literature
Smooth pursuit	Significantly reduced pursuit gain in elderly (66–87 y) compared with middle-aged (35–60 y)	Zackon and Sharpe[66]
	Lower vertical tracking gain in elderly (70 ± 8 y) with increased phase lags for higher frequencies compared with younger (30 ± 6 y) subjects	Demer[67]
	Lower tracking gain for predictable targets in the elderly (70 ± 8 y) with increased phase lags with increasing target stimuli Hz compared with younger (30 ± 6 y) subjects	Demer[67]
Saccadic tracking	Peak eye velocities reduced in the elderly (66–87 y) to unpredictable targets	Sharpe and Zackon[68]
	Latency and accuracy reduced for predictable and unpredictable targets in the elderly (66–87 y)	Sharpe and Zackon[68]
	Frequency hypometric saccades in the elderly (66–87 y)	Sharpe and Zackon[68]
	Increased latency and decreased peak eye velocity with advancing age (20–68 y)	Pitt and Rawles[69]
Optokinetic	Reduced optokinetic gain for elderly (70 ± 8 years) with greater phase lag than younger (30 ± 6 y) group	Demer[67]

CALORIC TESTING

Caloric responses have also received a broad investigation over the past few decades. Calder provided a nice summary of eight studies between 1971 and 1990, which is summarized in Table 2.[52]

Since the 1990s, studies investigating aging effects on the human caloric response have been sparse. Mallinson and Longridge provided one of two studies investigating this topic.[80] They examined the mean of the warm and cool caloric response from the better-responding ear from 185 patients ranging from 9 to 89 years of age who did not have any prior history of frank vestibular disease. The authors were in agreement with Peterka et al and found no significant correlation for the cross-sectional maximum age-related mean caloric response.[79] Although their study only examined the average maximum caloric response from the stronger ear, they argued this likely reflects a more accurate representation of the aging caloric response as it minimizes any deleterious impact on the data from previous subtle or significant vestibular disease that is possible with advancing age. Finally, Davidson and colleagues examined the reproducibility of the caloric response in 15 younger subjects (20 to 30 years) and in 14 older subjects (65 to 75 years).[81] They reported that the intersubject variability of the total caloric response was poor, citing a statistically significant intersubject variability in caloric responses in both young and older groups, with older groups exhibiting a greater magnitude of variability.

Rotational Vestibular Testing

Rotational testing offers clinicians the opportunity to examine the peripheral and, to a lesser extent, the central vestibular response using highly controlled stimuli. Specifically, tests of sinusoidal horizontal acceleration and velocity step testing evaluate the VOR response of the horizontal semicircular canal. Other rotational test paradigms exist, like off-axis vertical rotation, off-axis horizontal eccentric rotation, vestibular-visual interaction testing, and VOR fixation suppression testing; however, these tests are less frequently performed and have

Table 2 Age-related caloric irrigation findings (adapted from Calder[52])

Study	Study Sample	Findings
Bruner and Norris[73]	293 clinic patients with symptoms of dizziness and "normal" vestibular testing	Increase in SPEV caloric response up until 60–70 y with a subsequent decline in response (greater for warm irrigations)
Van der Laan and Ooseterveld[74]	334 normal adult subjects	Younger subjects (3rd decade) lower nystagmus frequency and higher nystagmus amplitude than subjects > 50 y of age
Clement et al[75]		Decreased SPEV with advancing age
Mulch and Petermann[51]	102 health adults from 11–70 y of age divided into 6 age groups	Most intense SPEV response obtained from middle to late middle-aged adults with a decrease in SPEV only observed after 60 y of age
Karlsen et al[76]	75 subjects aged 18–81 y	Declining SPEV, duration, amplitude, and frequency for warm irrigations beginning at age 65–70 y
Ghosh[77]	78 subjects divided into 7 age groups from 10–70 y	Overall decreasing SPEV as a function of age during serial vestibulometry
Jacobson and Henry[78]		Significant declines in VOR fixation suppression with advancing age
Peterka et al[79]	216 normal subjects from 7–81 y of age	No obvious changes in caloric response with advancing age with a high degree of response variability

Abbreviations: SPEV, slow-phase eye velocity; VOR, vestibular ocular reflex.

been less frequently examined for age-related effects.

The effects of aging on rotational testing have primarily been examined using sinusoidal harmonic acceleration and velocity step testing. As early as 1974, Van der Laan and Oosterveld examined the effects of aging on 774 healthy individuals using a torsional swing test.[74] They reported age-dependent changes identifying a significant decrease in slow-phase eye velocity after 70 years of age. Subsequent studies using sinusoidal harmonic acceleration testing further confirmed diminished slow-phase eye velocity with increasing age.[79,82,83] In a well-controlled study examining the effect of age on the horizontal VOR using rotational testing and a sclera search-coil technique, Paige identified distinct effects of aging.[70] Paige analyzed his patients using three different age groups; young (18 to 44 years), middle-aged (45 to 69 years), and elderly (70 to 89 years). He found VOR gain and phase to be remarkably similar across age groups with one notable exception. Paige identified lower VOR gain in the elderly for only the lowest rotational frequency tested with a concomitant increase in VOR phase for the two lowest frequencies tested when compared with the young group. As for the higher rotational frequencies, Paige reported no discernible differences in VOR gain or phase between any age group. When analyzing VOR linearity in response to an increasing velocity stimulus from 50 to 300 degrees per second stimulus, Paige identified a highly significant departure in VOR gain in response to a 300 degrees per second stimulus with the middle-aged and elderly group exhibiting significantly lower peak eye responses when compared with the young group. Nonlinearity in VOR phase was also highly significant and more robust than the disparities seen with VOR gain. Although all three groups were significantly different from one another at 300 degrees per second, the elderly group displayed the greatest departure

from linearity. Paige highlighted a similarity between the changes seen in the elderly response to those seen in younger patients with bilateral vestibular lesions, suggesting the notion that aging of the vestibular system may mirror similar processes.

Enrietto et al investigated aging effects on vestibular responses in a group of 57 healthy volunteers aged 75 years and older (mean age of 82 years) who were able to complete an annual evaluation over a 5-year time period.[84] This study represented the first efforts at identifying VOR changes with a longitudinal study design. Their results confirmed earlier reports by Paige,[70] detecting a decrease in rotational VOR gain and a concomitant increase in VOR phase lead for low-frequency, high-velocity stimuli. They reported a highly significant correlation in individual vestibular test results from the first to the fifth year, suggesting a high degree of reliability and stability to their data.

Furman and Redfern also investigated age-related VOR changes in the elderly.[23] They reported on 90 healthy subjects: 20 individuals between the ages of 20 to 29 years, 30 individuals between the ages of 60 to 69 years, and 30 individuals between the ages of 70 to 80 years. Furman and Redfern identified a significantly smaller low-frequency VOR phase lead in the youngest group when compared with the middle-aged and oldest group. There were no significant differences in VOR phase between the two older groups. The authors further determined a significant difference in the VOR time decay constants that were estimated form the sinusoidal data, citing significantly longer VOR time decay constants between the youngest and oldest group but not between the two older groups. Also in this study, Furman and Redfern offer one of few investigations of otolith functional decline with advancing age. Through off-vertical axis rotation, they identified a significant increase in the modulation component with a concomitant decrease in the bias component in older individuals (60 to 79 years) when compared with younger subjects (20 to 29 years). There were no significant differences in either component between the two older groups; however, the authors cited a trend for a progressively smaller bias component from 60 to 79 years of age. The authors suggested these results reflect an age-related decline of central vestibular processing likely localizing to the nodulus and uvula of the vestibule-cerebellum or its connections with the vestibular nuclei. This hypothesis agrees with previous reports alluded to earlier suggesting age-related declines in velocity storage, reduced OKN reflex nystagmus, and reduced smooth pursuit. Such central vestibular processing declines may also reflect the subtle manifestations of the anatomical deterioration of the peripheral sensory structures.[70] Such deterioration would undoubtedly lead to a consequential loss of velocity storage due to the slow adaptation of central compensation mechanisms.

Tian et al also investigated age-related changes in otolith function but employed a nontraditional stimulus of interaural, whole-body linear heave acceleration using a pneumatically driven servo-controlled chair.[85] The authors compared the linear VOR (LVOR) from a group of younger participants (18 to 31 years) against a group of older participants (56 to 75 years). They identified significant age-related differences in the heave LVOR consisting of increased LVOR latency, decreased early LVOR sensitivity, and decreased vestibular catch-up saccades in darkness. Collectively, the authors suggested that older individuals might have deficient afferent input from their otoliths, deficient central processing of otolith input, or both. They hypothesized such conclusions are likely secondary to well-documented changes in sensory and neural elements of the VOR pathways discussed earlier.

Serrador and colleagues also investigated loss of otolith function with age in 151 healthy participants ranging in age from 21 to 93 years.[86] They identified a significant age-related linear decline in ocular counter roll (OCR) during ± 20-degree roll tilt at 0.005 Hz. Their data further identified a novel gender-related rate of decline in OCR, with females demonstrating twice the decrease in OCR per decade than males. Serrador and colleagues also reported a significant correlation between OCR decline and an increase in mediolateral postural sway, which has previously

been shown to be a good predictor of falls, particularly under somatosensory-dependent conditions.[87] In light of this relationship, the authors postulated that the significant decline in OCR (otolith function) might be a significant predictor of falls in the elderly.

Finally, Chang and colleagues compared VOR response characteristics between a group of young (20 to 26 years) and old (63 to 84 years) individuals.[88] In contrast to other reports, these authors found no significant group differences in VOR gain or VOR phase for rotational frequencies between 0.025 to 0.5 Hz (using a standard 60 degrees per second peak velocity stimulus). Additionally, Chang and colleagues investigated whether psychophysical differences in the detection and discrimination thresholds for earth-vertical axis rotations existed between young and old individuals. They found no significant differences in discrimination thresholds between age groups. Furthermore, the authors found no significant differences in detection thresholds between young and older participants. These data agree with Roditi and Crane who employed similar stimuli but used a slightly different forced-choice paradigm.[89,90] Roditi and Crane suggested a possible trend for higher detection thresholds with advancing age when the stimulus frequency is increased above 0.5 Hz. However, due to a limited number of successful trials in study subjects over the age of 50 years of age ($n = 1$), the authors indicated that no direct conclusions could be made for higher frequency stimuli.

Cervical Vestibular Evoked Myogenic Potential Testing

Investigations have documented age-related changes in the cVEMP response. Researchers investigating the effects of increased age have suggested that a decrease in overall cVEMP amplitude and an increase in cVEMP threshold occurs with advancing age beginning around the sixth to seventh decade of life.[91–96] Janky and Shepard investigated the effects of age on various cVEMP parameters using click, 250-, 500-, 750-, and 100-Hz stimuli while controlling for sternocleidomastoid muscle activation using a blood pressure cuff monitor.[97] Janky and Shepard indicated that previous reports may be misleading or noncomparable secondary to the lack of monitoring of sternocleidomastoid muscle activation throughout these studies. They identified significant mean differences between age groups for P1 latency at 250, 500, 750 and 1,000 Hz as well as threshold at 500 and 1,000 Hz. Specifically, Janky and Shepard identified a significant difference in P1 latency that was dependent upon age for 250, 750, and 1,000 Hz with subjects aged 20 to 29 years exhibiting significantly longer P1 latencies than essentially all other age groups for each frequency. Furthermore, Janky and Shepard identified a significant positive correlation between cVEMP threshold and age and a significant negative correlation between cVEMP amplitude and age at 500 Hz. In short, Janky and Shepard concluded that "age should be taken into account when interpreting cVEMP threshold and that a comparison of threshold response curves may not be appropriate for individuals over 60 years of age."[97(p. 521)]

In a recent report by Akin and colleagues, cVEMP amplitude was also found to have a significant age effect, with the older age group (61 to 86 years of age) having significantly smaller cVEMP amplitudes than the younger cohort (22 to 31 years of age).[91] Aside from an age effect on cVEMP amplitude, Akin and colleagues also documented greater mean electromyography amplitude for the younger group with a concomitant smaller cVEMP electromyography amplitude variability. They concluded that the decrement seen in cVEMP amplitude is, therefore, associated with age-related changes occurring in both the physiology of the vestibular system as well as the sternocleidomastoid muscle. Finally, Agrawal and colleagues also identified significantly smaller cVEMP amplitudes in a group of individuals 70 years of age and older when compared with a group 50 years and younger.[98]

Ocular Vestibular Evoked Myogenic Potential Testing

A few reports have investigated the effect of age on the oVEMP. Iwasaki et al were the first to report on the effects of age on the amplitude and latency of oVEMP.[99] In a brief statement, Iwasaki and colleagues reported a significant

decrease in average oVEMP amplitude with age with a concomitant significant increase in oVEMP N1 latency with age for a cohort of 67 normal healthy subjects ranging in age from 28 to 83 years (mean age 47 years). In a later report from Rosengren et al, similar data were presented from a group of 61 healthy normal individuals from 20 to 80 years (10 subjects in each decade).[100] Specifically, they reported a significant decrease in oVEMP amplitude with increasing age, however, this effect was only present for oVEMPs generated in response to click stimuli. They failed to report any similar effect on amplitude with tone-burst stimuli. Independent of stimuli, Rosengren and colleagues showed a significant increase in oVEMP threshold and slight prolongation of N1 latency with increasing age.

Although both Iwasaki and colleagues and Rosengren and colleagues showed significant effects of age on oVEMP amplitude, neither report performed post hoc analyses to determine if there was a specific age group that was responsible for such age effects.[99,100] It was not until Piker et al investigated the normal characteristics of the oVEMP that a post hoc analysis revealed a clear effect of age on oVEMP amplitude.[101] They analyzed oVEMP response data from three groups of normal subjects: less than 18 years of age, between 18 to 49 years of age, and over 50 years of age. Post hoc pairwise comparisons showed a significant N1-P1 amplitude decrease and threshold increase in the oldest group ($\geq$50 years of age) when compared with both younger groups.

Nguyen and colleagues also identified a significant decrease in oVEMP (and cVEMP) amplitudes for subjects greater than 50 years of age; however, they failed to find any significant differences in latencies or asymmetry ratios.[102] Agrawal and colleagues further confirmed a significantly smaller N1 oVEMP amplitude in a group of individuals 70 years of age and older when compared with a group 50 years and younger.[98]

In a recent cross-sectional age study, Tseng et al investigated the effects of age on oVEMP for 70 healthy individuals (26 to 76 years) spanning six decades.[103] Tseng and colleagues were able to successfully record a bone-conducted oVEMP in 100% of patients through the sixth decade of life. However, responses were obtained from only 22% and 8% of ears in individuals within the seventh and eighth decade of life, respectively. Thus, the mere absence or presence of an oVEMP response was significantly different between those above and below 60 years of age. Furthermore, Tseng and colleagues identified a significant prolongation in P1 and N1 latencies in individuals over 60 years of age, as well as significantly reduced P1-N1 absolute amplitudes in individuals over 40 years of age.

Finally, Piker et al investigated the effect of age on the tuning of oVEMP (and cVEMP) responses recorded via air-conducted stimuli at octave and interoctave frequencies from 125 to 2,000 Hz.[104] They analyzed responses from 39 healthy participants (22 to 78 years) equally divided into three groups: young adult (18 to 39 years), middle age (40 to 59 years), and old adult (60 years or older). They identified a significant main effect for age and frequency on the amplitude of both the oVEMP and the cVEMP response with the older group exhibiting smaller overall response amplitudes, particularly for 125, 250, 1,500, and 2,000 Hz. Piker and colleagues also identified a significant loss of cVEMP tuning for the oldest group with an overall potential higher shift in tuning frequency for the older adult group.

One possible reason for the significant decline in the amplitude (or the mere presence or absence of an oVEMP response) in elderly individuals may be unrelated to utricular damage. Because the absolute displacement of visual gaze is shown to have such a potent effect on oVEMP amplitude, it is possible that the degree of upward gaze may have a relevant and confounding impact secondary to advancing age. This is due to the fact that the degree of maximal gaze is noted to decrease with age or may be restricted due to certain pathologies or physical limitations.[105] Although the degree to which ocular limitations contribute to an abnormal oVEMP response is unknown, other factors are clearly possible. Age-related changes in the mechanical properties of the utricle and the saccule and/or changes in the neuroelectrical responses of the maculae hair

cells could account for some of the age-related differences seen in individuals over 50 to 60 years of age.[104]

Video Head Impulse Testing and Vestibular Autorotation Testing

Studies investigating the effects of aging on vHIT and vestibular autorotation testing (VAT) have been limited. Since 2009, less than a few dozen reports have been published on these topics and most have emerged during the past few years. Most of these reports have concentrated on the use of vHIT and specifically its use in pathologic populations and in the development of normative response ranges in healthy, young, and middle-aged individuals.

Only a handful of reports have investigated the use of vHIT in the aging population. Agrawal and colleagues investigated the validity of using vHIT in the elderly.[106] They reported significant positive correlations between horizontal VOR gain responses from six community-dwelling elderly individuals (71 to 80 years of age) computed using sclera search-coil methods and video-oculography vHIT methods. From these data, Agrawal and colleagues concluded the use of video-oculography during vHIT is a reasonable and valid method for recording VOR gain and identifying corrective eye movements in elderly patients. Although the use of video-oculography vHIT methods were not employed during an investigation of normal results of 50 healthy older adults (70 to 95 years of age), Davalos-Bichara and Agrawal argued for the use of vHIT secondary to their findings of 64% prevalence of horizontal semicircular canal impairment in patients 80 years and older identified by clinical head thrust testing.[107] More recently, Matino-Soler and colleagues reported on normative vHIT data from 10 distinct decades of life using 212 subjects aged 5 to 95 years (110 women and 102 men).[108] They identified stable VOR gain, regardless of head impulse velocity, through 70 years of age. For individuals over 70 years of age, the authors identified a progressive decrease in VOR gain when compared with younger age groups, but only for higher head impulse velocities. However, a significant decrease in VOR gain was observed for all head impulse velocities for individuals greater than 90 years of age. Overall, when controlling for age, sex, and head velocity, Matino-Soler and colleagues identified a significant decrease in VOR gain as both age and head impulse velocity increased, with a greater number of individuals exhibiting refixation saccades over 70 years of age. Finally, Li and colleagues also published on the effects of aging on vHIT.[109] They evaluated 109 elderly subjects with vHIT ranging in age from 26 to 92 years. Li and colleagues reported a nonlinear relationship between vHIT VOR gain and age. They identified relatively stable gain between the ages of 26 to 79 years, after which a significant decline was observed. Specifically, Li and colleagues identified a nearly eightfold decrease in obtaining a vHIT VOR gain greater than 0.80 relative to those less than 80 years of age. No significant associations other than age were identified with a reduction in vHIT VOR gain, including cardiovascular risk factors.

The use of vHIT as a new and potentially effective measure in identifying age-related VOR changes in the elderly remains unclear. However, these data continue to repudiate any clear relationship between a decline in vestibular physiologic evidence and geriatric dizziness. In fact, McCaslin and colleagues recently provided evidence highlighting the absence of any predictive relationship between vHIT measures and self-reported dizziness handicap.[110]

A limited number of studies have investigated the effects of aging on VAT. Hirvonen and colleagues measured VOR gain and phase in 14 healthy elderly individuals (74 to 91 years) during autorotation in the yaw plane from 0.5 to 5 Hz.[111] When compared against 125 healthy subjects of working age, the authors found an increase in VOR gain and VOR phase lead above 2 Hz. They also observed a substantial loss of data from the elderly individuals above 2 Hz that was believed to be due to physical restrictions (e.g., neck stiffness) or psychological restrictions that self-limited higher frequency head shaking (which was hypothesized to increase dizziness secondary to significant

retinal slip). More recently, Hsieh et al evaluated 53 healthy participants between the ages of 25 and 75 years using autorotations between 1 and 3 Hz.[112] In contrast to the data presented by Hirvonen and colleagues, these authors reported a significant decline in mean eye velocity with respect to head velocity beginning at 55 years of age. Although the number of reports on VAT and aging is limited, it is not surprising the two reports differ in their conclusions given the large test variance and poor test–retest reliability that is known to exist with VAT.[113,114]

Comprehensive Vestibular Study

Maes and colleagues conducted one of the most comprehensive clinical studies to investigate age-related physiologic degeneration.[25] The authors provide a brief summary of the literature, which has been summarized in Table 3. These authors cited a fundamental problem in many age-related clinical studies, which may account for the contradictory conclusions across studies. Maes and colleagues concluded that few researchers have ever "explored age-dependent effects by means of *multiple* tests in the *same* population, making it difficult to compare

Table 3 Summary of Age-Related Clinical Findings Adapted from Maes et al[25]

Clinical Measure	Clinical Findings	Literature
Rotational vestibular testing	Reduced VOR gain for the low frequencies	Wall et al[83]; Peturka et al[79]; Paige[71]; Furman and Redfern[23]
	Reduced VOR for the high frequencies	Li et al[115]
	Reduced VOR gain to higher stimulus velocities	Paige[70]; Baloh et al[28]
	Increased VOR phase lead for low frequencies	Li et al[115]; Paige et al[71]
	Increased VOR phase lead for high frequencies	Baloh et al[28]; Peterka et al[79]; Furman and Redfern[23]
	VOR asymmetry	None reported
	Decreasing time constant	Baloh et al[28]
	Larger time constant asymmetry	Stefansson and Imoto[116]; Dizio and Lackner[117]; Furman and Redfern[23]
Videonystagmography	Decreasing max slow-phase velocity	Stefansson and Imoto[116]
	Increasing directional preponderance	Stefansson and Imoto[116]
	Increasing max slow-phase velocity to maximum age followed by a slight decline	Bruner and Norris[73] (60–70 y); Karlsen et al[76] (60–70 y); Mulch and Petermann (50 y)[51]
	No change reported	Peterka et al[79]; Mallinson and Longridge[80]; Zapala et al[118]
Vestibular evoked myogenic potential	Decreasing absolute amplitude	Welgampola and Colebatch[95]; Ochi and Ohashi[119]; Su et al[94]; Zapala and Brey[96]; Basta et al[120,121]; Brantberg et al[92]; Lee et al[93]
	Increasing threshold	Welgampola and Colebatch[95]; Su et al[94]
	Prolonged P1–N1 latencies	Zapala and Brey[96]; Brantberg et al[92]; Lee et al[93]

Abbreviation: VOR, vestibular ocular reflex.

age trends between different function tests." To address this problem, they conducted a within-subject design of 80 healthy participants from 18 through 80 years of age, all receiving rotational testing, VEMP testing, and caloric testing. Regarding rotational testing, the authors concluded that age-related trends could only be elicited when stimulus frequencies exceeded 100 degrees per second. In fact, they indicated that stimulus velocities of 180 degrees per second (or more) are needed to reveal age-related trends, but even then are only subtle VOR gain differences apparent for the oldest group (>66 years of age). These criteria quickly become clinically prohibitive as such stimuli are often inappropriate for and/or not tolerated well by older patients. Regarding caloric testing, Maes and colleagues noted an increase in the slow-phase velocity of the induced nystagmus; particularly in response to warm irrigations. Although Maes and colleagues admit that this finding is not novel, their findings fail to confirm a reported decline in nystagmus slow-phase eye velocity with advancing age. They hypothesized that this may be due to a lack of study patients above 80 years of age, where declines in caloric slow-phase eye velocity have been reported. With respect to labyrinthine symmetry and directional preponderance during caloric testing, Maes et al reported no main effects with advanced age, although a poorer fixation suppression index was observed in their older participants. They attributed this to possible age-related deterioration of the cerebellar flocculus, pons, or mesencephalic visual centers. Maes and colleagues are quick to highlight the discrepancy within their own results; that is, having age-related trends for a low-frequency caloric stimulus while rotational stimuli of less than 100 degrees per second were insufficient to elicit age-related trends. They reported that inconsistencies with the stability of the caloric stimulus were likely to account for such discrepancies. Finally, with respect to VEMP testing, Maes and colleagues reported the largest age-related trends. Results of their study show a significant decline in absolute P1-N1 amplitudes, increasing thresholds, and decreasing N1 latencies. These data show strong agreement with the reported literature. Finally, the authors call attention to the fact that any within-group comparison of VEMP data to the caloric or rotational data are inappropriate given the fact that two distinct reflex pathways are evaluated. Maes and colleagues summarized their salient findings, noting that although age-related declines in clinical data are often subtle or nonsignificant, age-related trends are more likely to be observed using more robust stimuli. Unfortunately, this target population is less inclined to tolerate such stimuli.

CONCLUSION

It is clear that current clinical methods for documenting age-related declines in vestibular function remain elusive. The majority of clinical studies investigating the age-related decline in vestibular physiology have been unable to demonstrate any strong correlation to the histologic reports identifying an obvious decline in sensory hair cell counts, particularly prior to 60 to 70 years of age.[20,25] When abnormalities are present, age-related differences are usually subtle, often fall within the lower ranges of normal limits, and/or exhibit larger variances making statistically discernable differences from younger or middle-aged groups difficult. Although significant central compensation mechanisms are likely responsible for these observations, it is inevitable that "at some critical point both peripheral and central changes reach a level that can no longer be compensated for and vestibular function deteriorates."[28(p. 514)] Paige offers yet one additional explanation for such poor correlations between clinical and histologic data.[70,71] He suggests that the mild stimuli used for most clinical evaluations of the vestibular system may be insufficient to overcome the compensatory and adaptive mechanisms provided by the central nervous system during the slow aging process. In light of this, Paige suggested more challenging and robust stimuli be used to expose the elusive age-related changes present in many older patients. Furman and Redfern also promoted the use of alternative and more robust clinical measures to increase sensitivity to age-related changes.[23] However, conducting more robust measures like off-vertical axis rotation testing or high-velocity step testing of at least 300 degrees per second would likely be impractical and not well

tolerated by elderly patients during routine clinical testing. As it currently stands, the stimuli employed during most routine clinical vestibular assessments have proven to be largely ineffective in segregating any age-related differences in vestibular function. The sensitivity of vestibular testing for identifying age-related physiologic changes is, therefore, questionable given the current use of such impuissant stimuli. As a result, most clinical data highlighting age-related vestibular loss are widely varied in their results and many conclusions are weak with only subtle corollaries prior to 60 years of age.

Dizziness in the elderly is a significant problem that has the potential to reach near epidemic proportions over the next 20 to 30 years. The question still remains as to whether or not the medical community is prepared to handle such an impending challenge. Correctly differentiating a vestibular etiology will undoubtedly be a critical component to managing dizziness in the elderly. However, this can be significantly more challenging in the geriatric population insomuch that dizziness is often a multifocal and nebulous symptom. Some have argued that aging is not the cause of dizziness. Rather, dizziness is secondary to comorbid conditions for which older adults are more susceptible. Such conditions would include metabolic, cardiovascular, central nervous system, gastrointestinal, hormonal, polypharmacy, pharmacokinetics, and pharmacodynamics.[5] Comorbidities in the elderly population, like a higher incidence of polypharmacy, significantly complicate a difficult clinical canvas by which etiologies of a vestibular origin can correctly be identified. As medical diagnoses broaden to include dizziness as its own geriatric syndrome,[122] our approach to discovering the underlying etiology must also broaden. Cross-disciplinary medical care will be imperative to effectively diagnose and treat such a multifactorial and obscure condition as geriatric dizziness. Furthermore, it is clear that advancements and alternatives to our current battery of vestibular tests and stimuli are critically needed to better segregate and identify age-related vestibular changes. As we stand on the brink of a rapidly aging population, it is abundantly clear that a tremendous challenge lies ahead.

REFERENCES

1. Horak F. Role of the Vestibular System in Postural Control. 3rd ed. Philadelphia, PA: F.A. Davis Company; 2008
2. Keshner E. Postural Abnormalities in vestibular Disorders. 3rd ed. Philadelphia, PA: F.A. Davis Company; 2007
3. Peterka RJ. Sensorimotor integration in human postural control. J Neurophysiol 2002;88(3): 1097–1118
4. Marchetti GF, Whitney SL. Older adults and balance dysfunction. Neurol Clin 2005;23(3): 785–805, vii
5. Shoair OA, Nyandege AN, Slattum PW. Medication-related dizziness in the older adult. Otolaryngol Clin North Am 2011;44(2): 455–471, x
6. Dewane JA. Dealing with dizziness and disequilibrium in older patients: a clinical approach. Topics in Geriatric Rehabilitation 1995;11(1): 30–38
7. Cutson TM. Falls in the elderly. Am Fam Physician 1994;49(1):149–156
8. Hobeika CP. Equilibrium and balance in the elderly. Ear Nose Throat J 1999;78(8):558–562, 565–566
9. Dillon CF, Gu Q, Hoffman HJ, Ko CW. Vision, hearing, balance, and sensory impairment in Americans aged 70 years and over: United States, 1999–2006. NCHS Data Brief 2010;31:1–8
10. Katsarkas A. Dizziness in aging: a retrospective study of 1194 cases. Otolaryngol Head Neck Surg 1994;110(3):296–301
11. Baloh RW. Vestibular and auditory disorders. Curr Opin Neurol 1996;9(1):32–36
12. NIH National Institutes of Deafness and Other Communication Disorders (NIDCD); 1997:122
13. University of Iowa Health Care. Comprehensive management of vestibular disorders. Currents 2002;3(2)
14. Jacobson GP, McCaslin DL, Grantham SL, Piker EG. Significant vestibular system impairment is common in a cohort of elderly patients referred for assessment of falls risk. J Am Acad Audiol 2008; 19(10):799–807
15. Kippenbrock T, Soja ME. Preventing falls in the elderly: Interviewing patients who have fallen. Researchers attempt to identify fall-risk factors from the patients' point of view. Geriatr Nurs (Minneap) 1993;14(4):205–209
16. Ambrose AF, Paul G, Hausdorff JM. Risk factors for falls among older adults: A review of the literature. Maturitas 2013;75(1):51–61
17. Todd CJ, Freeman CJ, Camilleri-Ferrante C, et al. Differences in mortality after fracture of hip: The east anglian audit. BMJ 1995;310(6984): 904–908

18. Windmill IM, Freeman BA. Demand for audiology services: 30-yr projections and impact on academic programs. J Am Acad Audiol 2013; 24(5):407–416
19. Matheson AJ, Darlington CL, Smith PF. Dizziness in the elderly and age-related degeneration of the vestibular system. N Z J Psychol 1999;28(1): 10–16
20. Rosenhall U. Degenerative patterns in the aging human vestibular neuro-epithelia. Acta Otolaryngol 1973;76(2):208–220
21. Anniko M. The aging vestibular hair cell. Am J Otolaryngol 1983;4(3):151–160
22. Campos A, Canizares FJ, Sanchez-Quevedo MC, Romero PJ. Otoconial degeneration in the aged utricle and saccule. Adv Otorhinolaryngol 1990; 45:143–153
23. Furman JM, Redfern MS. Effect of aging on the otolith-ocular reflex. J Vestib Resl 2001;11(2): 91–103
24. Lim DJ. Otoconia in health and disease. A review. Ann Otol Rhinol Laryngol Suppl 1984;112:17–24
25. Maes L, Dhooge I, D'Haenens W, et al. The effect of age on the sinusoidal harmonic acceleration test, pseudorandom rotation test, velocity step test, caloric test, and vestibular-evoked myogenic potential test. Ear Hear 2010;31(1):84–94
26. Walther LE, Westhofen M. Presbyvertigo-aging of otoconia and vestibular sensory cells. Journal of vestibular research: equilibrium & orientation 2007;17(2–3):89–92
27. Rauch SD, Velazquez-Villasenor L, Dimitri PS, Merchant SN. Decreasing hair cell counts in aging humans. Ann N Y Acad Sci 2001; 942:220–227
28. Baloh RW, Jacobson KM, Socotch TM. The effect of aging on visual-vestibuloocular responses. Exp Brain Res 1993;95(3):509–516
29. Jang YS, Hwang CH, Shin JY, Bae WY, Kim LS. Age-related changes on the morphology of the otoconia. Laryngoscope 2006;116(6):996–1001
30. Matheson AJ, Darlington CL, Smith PF. Further evidence for age-related deficits in human postural function. J Vestib Res 1999;9(4):261–264
31. Rosenhall U, Rubin W. Degenerative changes in the human vestibular sensory epithelia. Acta Otolaryngol 1975;79(1–2):67–80
32. Richter E. Quantitative study of human scarpa's ganglion and vestibular sensory epithelia. Acta Otolaryngol 1980;90(3–4):199–208
33. Bergstrom B. Morphology of the vestibular nerve. I. Anatomical studies of the vestibular nerve in man. Acta Otolaryngol 1973;76(2):162–172
34. Bergstrom B. Morphology of the vestibular nerve. Ii. The number of myelinated vestibular nerve fibers in man at various ages. Acta Otolaryngol 1973;76(2):173–179
35. Lopez I, Honrubia V, Baloh RW. Aging and the human vestibular nucleus. J Vestib Res 1997;7(1): 77–85
36. Alvarez JC, Diaz C, Suarez C, et al. Neuronal loss in human medial vestibular nucleus. Anat Rec 1998;251(4):431–438
37. Merchant SN, Velazquez-Villasenor L, Tsuji K, Glynn RJ, Wall C III, Rauch SD. Temporal bone studies of the human peripheral vestibular system. Normative vestibular hair cell data. Ann Otol Rhinol Laryngol Suppl 2000;181:3–13
38. Velazquez-Villasenor L, Merchant SN, Tsuji K, Glynn RJ, Wall C III, Rauch SD. Temporal bone studies of the human peripheral vestibular system. Normative scarpa's ganglion cell data. Ann Otol Rhinol Laryngol Suppl 2000;181:14–19
39. Engstrom H, Bergstrom B, Rosenhall U. Vestibular sensory epithelia. Arch Otolaryngol 1974; 100(6):411–418
40. Igarashi M, Saito R, Mizukoshi K, Alford BR. Otoconia in young and elderly persons: A temporal bone study. Acta Otolaryngol Suppl 1993; 504:26–29
41. Johnsson LG. Degenerative changes and anomalies of the vestibular system in man. Laryngoscope 1971;81(10):1682–1694
42. Johnsson LG, Hawkins JE Jr. Otolithic membranes of the saccule and utricle in man. Science 1967;157(3795):1454–1456
43. Johnsson LG, Hawkins JE Jr. Vascular changes in the human inner ear associated with aging. Ann Otol Rhinol Laryngol 1972;81(3):364–376
44. Thalmann R, Ignatova E, Kachar B, Ornitz DM, Thalmann I. Development and maintenance of otoconia: Biochemical considerations. Ann N Y Acad Sci 2001;942:162–178
45. Baloh RW, Honrubia V. Clinical neurophysiology of the vestibular system. 3rd ed. Philadelphia, PA: F.A. Davis Company; 2001
46. Gretsy MA, Lempert T. Pathophysiology and clinical testing of otolith dysfunction. New York, NY: Karger; 2001
47. Leigh RJ, Zee DS. The neurology of eye movements. 4th ed. New York: Oxford University Press 2006
48. Minor LB, Goldberg JM. Vestibular-nerve inputs to the vestibulo-ocular reflex: A functional-ablation study in the squirrel monkey. J Neurosci 1991; 11(6):1636–1648
49. Tang PF, Woollacott M. Balance control in older adults. In: Bronstein AM, Brandt TW, M., Nutt J, eds. Clinical disorders of balance, posture and gait. 2nd ed. London: Arnold; 2004:385–403
50. Jacobson GP, McCaslin DL, Patel S, Barin K, Ramadan NM. Functional and anatomical correlates of impaired velocity storage. J Am Acad Audiol 2004;15(4):324–333

51. Mulch G, Petermann W. Influence of age on results of vestibular function tests. Review of literature and presentation of caloric test results. Ann Otol Rhinol Laryngol Suppl 1979;88(2 Pt 2 Suppl 56)1–17
52. Calder JH. Aging and the balance control systems. In: Weinstein BE, ed. Geriatric audiology. New York, NY: Thieme; 2000:141–167
53. Lynch SJ, Nayak US, Isaacs B. Positional and optokinetic nystagmus in healthy old people. Age Ageing 1985;14(2):122–124
54. Kerber KA, Ishiyama GP, Baloh RW. A longitudinal study of oculomotor function in normal older people. Neurobiol Aging 2006;27(9): 1346–1353
55. Irving EL, Steinbach MJ, Lillakas L, Babu RJ, Hutchings N. Horizontal saccade dynamics across the human life span. Invest Ophthalmol Vis Sci 2006;47(6):2478–2484
56. Peltsch A, Hemraj A, Garcia A, Munoz DP. Age-related trends in saccade characteristics among the elderly. Neurobiol Aging 2011;32(4):669–679
57. Tuunainen E, Poe D, Jantti P, et al. Presbyequilibrium in the oldest old, a combination of vestibular, oculomotor and postural deficits. Aging Clin Exp Res 2011;23(5–6):364–371
58. Sharpe JA, Sylvester TO. Effect of aging on horizontal smooth pursuit. Invest Ophthalmol Vis Sci 1978;17(5):465–468
59. Spooner JW, Sakala SM, Baloh RW. Effect of aging on eye tracking. Arch Neurol 1980;37(9): 575–576
60. Jagacinski RJ, Liao MJ, Fayyad EA. Generalized slowing in sinusoidal tracking by older adults. Psychol Aging 1995;10(1):8–19
61. Morrow MJ, Sharpe JA. Smooth pursuit initiation in young and elderly subjects. Vision Res 1993; 33(2):203–210
62. Kanayama R, Nakamura T, Sano R, et al. Effect of aging on smooth pursuit eye movement. Acta Otolaryngol Suppl 1994;511:131–134
63. Kato I, Sakuma A, Ogino S, Takahashi K, Okada T. Aging effects upon pursuit eye movements. Acta Otolaryngol Suppl 1995;520((Pt 2):):293–294
64. Simons B, Buttner U. The influence of age on optokinetic nystagmus. Eur Arch Psychiatry Neurol Sci 1985;234(6):369–373
65. Farooq SJ, Gottlob I, Benskin S, Proudlock FA. The effect of aging on torsional optokinetic nystagmus. Invest Ophthalmol Vis Sci 2008; 49(2):589–593
66. Zackon DH, Sharpe JA. Smooth pursuit in senescence. Effects of target acceleration and velocity. Acta Otolaryngol 1987;104(3–4):290–297
67. Demer JL. Effect of aging on vertical visual tracking and visual-vestibular interaction. J Vestib Res 1994;4(5):355–370
68. Sharpe JA, Zackon DH. Senescent saccades. Effects of aging on their accuracy, latency and velocity. Acta Otolaryngol 1987;104(5–6): 422–428
69. Pitt MC, Rawles JM. The value of measuring saccadic eye movement in the investigation of non-compressive myelopathy. J Neurol Neurosurg Psychiatry 1989;52(10):1157–1161
70. Paige GD. The aging vestibulo-ocular reflex (vor) and adaptive plasticity. Acta Otolaryngol Suppl 1991;481:297–300
71. Paige GD. Senescence of human visual-vestibular interactions. 1. Vestibulo-ocular reflex and adaptive plasticity with aging. J Vestib Res 1992;2(2): 133–151
72. Hajioff D, Barr-Hamilton RM, Colledge NR, Lewis SJ, Wilson JA. Re-evaluation of normative electronystagmography data in healthy ageing. Clin Otolaryngol Allied Sci 2000;25(4):249–252
73. Bruner A, Norris TW. Age-related changes in caloric nystagmus. Acta Otolaryngol Suppl 1971; 282:1–24
74. van der Laan FL, Oosterveld WJ. Age and vestibular function. Aerosp Med 1974;45(5): 540–547
75. Clement PA, Van der Laan FL, Oosterveld WJ. The influence of age on vestibular function. Acta Otorhinolaryngol Belg 1975;29(1):163–172
76. Karlsen EA, Hassanein RM, Goetzinger CP. The effects of age, sex, hearing loss and water temperature on caloric nystagmus. Laryngoscope 1981; 91(4):620–627
77. Ghosh P. Aging and auditory vestibular response. Ear Nose Throat 1985;64(5):264–266
78. Jacobson GP, Henry KG. Effect of temperature on fixation suppression ability in normal subjects: The need for temperature- and age-dependent normal values. Ann Otol Rhinol Laryngol 1989; 98(5 Pt 1):369–372
79. Peterka RJ, Black FO, Schoenhoff MB. Age-related changes in human vestibulo-ocular reflexes: Sinusoidal rotation and caloric tests. J Vestib Res 1990;1(1):49–59
80. Mallinson AI, Longridge NS. Caloric response does not decline with age. J Vestib Res 2004;14(5): 393–396
81. Davidson J, Wright G, McIlmoyl L, Canter RJ, Barber HO. The reproducibility of caloric tests of vestibular function in young and old subjects. Acta Otolaryngol 1988;106(3–4):264–268
82. Wall C III, Black FO. Intersubject variability in vor responses to 0.005–1.0 hz sinusoidal rotations. Acta Otolaryngol Suppl 1984;406:194–198
83. Wall C III, Black FO, Hunt AE. Effects of age, sex and stimulus parameters upon vestibulo-ocular responses to sinusoidal rotation. Acta Otolaryngol 1984;98(3–4):270–278

84. Enrietto JA, Jacobson KM, Baloh RW. Aging effects on auditory and vestibular responses: A longitudinal study. Am J Otolaryngol 1999;20(6): 371–378
85. Tian JR, Crane BT, Wiest G, Demer JL. Effect of aging on the human initial interaural linear vestibulo-ocular reflex. Exp Brain Res 2002;145(2): 142–149
86. Serrador JM, Lipsitz LA, Gopalakrishnan GS, Black FO, Wood SJ. Loss of otolith function with age is associated with increased postural sway measures. Neurosci Lett 2009;465(1):10–15
87. Maki BE, Holliday PJ, Topper AK. A prospective study of postural balance and risk of falling in an ambulatory and independent elderly population. J Gerontol 1994;49(2):M72–M84
88. Chang NY, Hiss MM, Sanders MC, et al. Vestibular perception and the vestibulo-ocular reflex in young and older adults. Ear Hear 2014;35(5): 565–570
89. Roditi RE, Crane BT. Suprathreshold asymmetries in human motion perception. Exp Brain Res 2012;219(3):369–379
90. Roditi RE, Crane BT. Directional asymmetries and age effects in human self-motion perception. Journal of the Association for Research in Otolaryngology: JARO. 2012;13(3):381–401
91. Akin FW, Murnane OD, Tampas JW, Clinard CG. The effect of age on the vestibular evoked myogenic potential and sternocleidomastoid muscle tonic electromyogram level. Ear Hear 2011; 32(5):617–622
92. Brantberg K, Granath K, Schart N. Age-related changes in vestibular evoked myogenic potentials. Audiol Neurootol 2007;12(4):247–253
93. Lee SK, Cha CI, Jung TS, Park DC, Yeo SG. Age-related differences in parameters of vestibular evoked myogenic potentials. Acta Otolaryngol 2008;128(1):66–72
94. Su HC, Huang TW, Young YH, Cheng PW. Aging effect on vestibular evoked myogenic potential. Otology & neurotology: official publication of the American Otological Society, American Neurotology Society. [and] European Academy of Otology and Neurotology 2004; 25(6):977–980
95. Welgampola MS, Colebatch JG. Vestibulocollic reflexes: Normal values and the effect of age. Clinical neurophysiology: official journal of the International Federation of Clinical Neurophysiology. 2001;112(11):1971–1979
96. Zapala DA, Brey RH. Clinical experience with the vestibular evoked myogenic potential. J Am Acad Audiol 2004;15(3):198–215
97. Janky KL, Shepard N. Vestibular evoked myogenic potential (vemp) testing: Normative threshold response curves and effects of age. J Am Acad Audiol 2009;20(8):514–522
98. Agrawal Y, Zuniga MG, Davalos-Bichara M, et al. Decline in semicircular canal and otolith function with age. Otology & neurotology: official publication of the American Otological Society, American Neurotology Society. [and] European Academy of Otology and Neurotology 2012; 33(5):832–839
99. Iwasaki S, Smulders YE, Burgess AM, et al. Ocular vestibular evoked myogenic potentials to bone conducted vibration of the midline forehead at fz in healthy subjects. Clinical neurophysiology: official journal of the International Federation of Clinical Neurophysiology. 2008; 119(9):2135–2147
100. Rosengren SM, Govender S, Colebatch JG. Ocular and cervical vestibular evoked myogenic potentials produced by air- and bone-conducted stimuli: Comparative properties and effects of age. Clinical neurophysiology: official journal of the International Federation of Clinical Neurophysiology. 2011;122(11):2282–2289
101. Piker EG, Jacobson GP, McCaslin DL, Hood LJ. Normal characteristics of the ocular vestibular evoked myogenic potential. J Am Acad Audiol 2011;22(4):222–230
102. Nguyen KD, Welgampola MS, Carey JP. Test-retest reliability and age-related characteristics of the ocular and cervical vestibular evoked myogenic potential tests. Otol Neurotol 2010;31(5): 793–802
103. Tseng CL, Chou CH, Young YH. Aging effect on the ocular vestibular-evoked myogenic potentials. Otol Neurotol 2010;31(6):959–963
104. Piker EG, Jacobson GP, Burkard RF, McCaslin DL, Hood LJ. Effects of age on the tuning of the cvemp and ovemp. Ear Hear 2013;34(6):e65–e73
105. Chamberlain W. Restriction in upward gaze with advancing age. Am J Ophthalmol 1971;71(1 Pt 2):341–346
106. Agrawal Y, Schubert MC, Migliaccio AA, et al. Evaluation of quantitative head impulse testing using search coils versus video-oculography in older individuals. Otol Neurotol 2014;35(2): 283–288
107. Davalos-Bichara M, Agrawal Y. Normative results of healthy older adults on standard clinical vestibular tests. Otol Neurotol 2014;35(2): 297–300
108. Matino-Soler E, Esteller-More E, Martin-Sanchez JC, Martinez-Sanchez JM, Perez-Fernandez N. Normative data on angular vestibulo-ocular responses in the yaw axis measured using the video head impulse test. Otology & neurotology: official publication of the American Otological Society, American Neurotology Society [and] European Academy of Otology and Neurotology. 2014
109. Li C, Layman AJ, Geary R, et al. Epidemiology of vestibulo-ocular reflex function: Data from the

baltimore longitudinal study of aging. Otology & neurotology: official publication of the American Otological Society, American Neurotology Society [and] European Academy of Otology and Neurotology. 2014
110. McCaslin DL, Jacobson GP, Bennett ML, Gruenwald JM, Green AP. Predictive properties of the video head impulse test: Measures of caloric symmetry and self-report dizziness handicap. Ear Hear 2014;35(5):e185–e191
111. Hirvonen TP, Aalto H, Pyykko I, Juhola M, Jantti P. Changes in vestibulo-ocular reflex of elderly people. Acta Otolaryngol Suppl 1997;529:108–110
112. Hsieh LC, Lin HC, Lee GS. Aging of vestibular function evaluated using correlational vestibular autorotation test. Clin Interv Aging 2014; 9:1463–1469
113. Guyot JP, Psillas G. Test-retest reliability of vestibular autorotation testing in healthy subjects. Otolaryngology–head and neck surgery: official journal of American Academy of Otolaryngology-. Head Neck Surg 1997;117(6):704–707
114. Blatt PJ, Schubert MC, Roach KE, Tusa RJ. The reliability of the vestibular autorotation test (vat) in patients with dizziness. J Neurol Phys Ther 2008;32(2):70–79
115. Li CW, Hooper RE, Cousins VC. Sinusoidal harmonic acceleration testing in normal humans. Laryngoscope 1991;101(2):192–196
116. Stefansson S, Imoto T. Age-related changes in optokinetic and rotational tests. Am J Otol 1986; 7(3):193–196
117. DiZio P, Lackner JR. Age differences in oculomotor responses to step changes in body velocity and visual surround velocity. J Gerontol 1990; 45(3):M89–M94
118. Zapala DA, Olsholt KF, Lundy LB. A comparison of water and air caloric responses and their ability to distinguish between patients with normal and impaired ears. Ear Hear 2008;29(4): 585–600
119. Ochi K, Ohashi T. Age-related changes in the vestibular-evoked myogenic potentials. Otolaryngology–head and neck surgery: official journal of American Academy of Otolaryngology-. Head Neck Surg 2003;129(6):655–659
120. Basta D, Todt I, Ernst A. Normative data for p1/n1-latencies of vestibular evoked myogenic potentials induced by air- or bone-conducted tone bursts. Clinical neurophysiology: official journal of the International Federation of Clinical Neurophysiology 2005;116(9):2216–2219
121. Basta D, Todt I, Ernst A. Characterization of age-related changes in vestibular evoked myogenic potentials. J Vestib Res 2007;17(2–3):93–98
122. Tinetti ME, Williams CS, Gill TM. Dizziness among older adults: A possible geriatric syndrome. Ann Intern Med 2000;132(5):337–344