The vestibular system encodes self motion and spatial orientation. Semicircular canals sense rotation, while the otoliths sense translation and the force of gravity (Fernandez and Goldberg 1976). Displacement of the cupula in the semicircular canal and the otolith membrane transduces the rotational or translational forces into a vestibular nerve action potential. Artificial ways to stimulate the vestibular system, namely galvanic and caloric stimulation, were developed to probe the physiology of the vestibular system and to diagnose disorders of the labyrinth as well as the parts of the brain to where it projects.

Galvanic vestibular stimulation has been known for almost two centuries (Purkyne 1819). The galvanic currents, applied on the skin over the mastoid process, evoke nystagmus and a percept of motion (Hitzig 1974; Volta 1923). Suzuki and colleagues used electrical stimulation as a scientific tool to study the effects of selective modulation of vestibular afferents innervating a specific semicircular canal (Cohen and Suzuki 1963; Cohen et al. 1964; Suzuki et al. 1964, 1969). This concept then inspired the development of the vestibular protheses to selectively modulate the activity of the vestibular afferents with locally implanted electrodes (Della Santina et al. 2005; Gong and Merfeld 2002; Phillips et al. 2011). This approach of artificial vestibular stimulation has the potential to restore impaired balance due to vestibular end-organ failure.

Caloric stimulation, first described by Robert Barany in 1906, is another way to stimulate the vestibular end-organs. Otologists routinely flushed ear canals to remove cerumen. It was noticed that when the external auditory canal was irrigated with cold water, the patient would experience vertigo. The symptoms were minimal if the process was done with the patient sitting upright. Barany proposed that the temperature changes in the external auditory canal during irrigation would transiently change the temperature of the endolymph in the portion of the semicircular canal closest to the external auditory canal. The cold water would decrease the temperature and increase the specific gravity of the endolymph. When the patient was supine, the “heavy” endolymph would begin to settle away from the horizontal-canal ampulla causing horizontal nystagmus with slow phases away from the irrigated ear. The warm water irrigation would cause the opposite phenomenon. When the subject was upright, there would be minimal effect of gravity, thus minimal movement of the endolymph to cause caloric nystagmus and vertigo. By comparing the duration of nystagmus after each caloric stimulus, Barany had described ways to localize malfunctioning semicircular canals. Robert Barany was awarded the Nobel Prize for these pivotal findings (Balah 2002).

For more than a century, we only knew of the electrical and thermal irrigation techniques to artificially stimulate the human vestibular system. One may ask whether we could use magnets to stimulate the vestibular system? Indeed, we know that large field strengths of the magnetic resonance imaging (MRI) scanners induce vertigo (Glover et al. 2007). Altered locomotion patterns, head posturing, conditioned taste aversion, and avoidance learning were reported in rodents exposed to high magnetic field (Houpt et al. 2011; Nolte et al. 1998). It was proposed that movement through the magnetic field induces an electric current that stimulates the end-organ in the semicircular canals causing subjects to experience vertigo (Glover et al. 2007). Movement through the magnetic field was thought to be the fundamental requirement to induce vertigo, but Marcelli and colleagues (2009) showed that nystagmus was present in humans who even remained still in the MRI scanner.

Roberts and colleagues (2011) proposed that magnetic vestibular stimulation (MVS) is due to the Lorentz force. Labyrinthine sensors are unique because they are bony cavities lined with neuroepithelium and filled with potassium rich endolymph. This feature of the endolymph normally facilitates ion current transduction through the hair cells even in the absence of endolymph movement. When the vestibular apparatus is exposed to the magnetic field, even in the absence of head (and endolymph) movement, the Lorentz force is generated in the direction perpendicular to the magnetic field and the ion current. The Lorentz force causes the deviation of the cupula in the semicircular canals. The Lorentz force follows equation 1.

\[ F = J \times B \]

In Eq. 1, F is the vector of Lorentz force, J is the vector of ion current across the hair cells, and B depicts the electromagnetic field. This equation is a simplification, but the computational details of the Lorentz force and its dependence is outlined in Roberts et al. (2011). The amplitude and direction of the Lorentz force rely on the amplitude and the direction of J as well as on the strength and direction of B. At least two
Fig. 1. A: simplified computational model of the vestibulo-ocular reflex (VOR) pathway (Leigh et al. 1981). The box titled “semicircular canal” generates the desired rotational stimuli. The yellow box represents the indirect pathway with 2 components, adaptation and velocity storage. The direct pathway represents 3-neuron-arc. B: constant velocity stimulation paradigm; each color of the trace depicts stimulus velocity. In this graph, stimulus velocity is plotted on the y-axis and time on the x-axis. C: the cupular deflection was simulated by recording the output of cupular transfer function. The values are plotted on the y-axis and corresponding time on the x-axis. Each color of the trace matches the color-coded head velocity plotted in B. D: the slow-phase eye velocity of the rotational nystagmus. Slow-phase eye velocity is plotted on the y-axis and the time is plotted on the x-axis. Each color of the trace matches the color-coded head velocity and cupular deflection traces. The slow-phase eye velocity during rotational phase abruptly increases and then decays slower than the decay of simulated cupular deflection. There is a reversal in the direction of the slow-phase eye velocity after its value reaches zero (i.e., alteration in the direction of the rotational nystagmus) as expected due to adaptation mechanism (Leigh et al. 1981). The velocity storage and the adaptive mechanisms reside in the brainstem and are activated by the persistent vestibular stimulation. E: constant acceleration stimulation paradigm; each color of the trace depicts stimulus acceleration. In this graph, stimulus velocity is plotted on the y-axis and time on the x-axis. F: the cupular deflection was simulated by recording the output of cupular transfer function. The values are plotted on the y-axis and corresponding time on the x-axis. Each color of the trace matches the color-coded head velocity plotted in E. G: slow-phase eye velocity is plotted on the y-axis and time is on the x-axis. Each color of the trace matches the color-coded acceleration and cupular output traces.
sources determine the amplitude and direction of J, the ion currents through the hair cells in the utricle (J\text{U}) and in the ampulla (J\text{A}). The change in the steady-state otolith discharge during static head tilts (Fernandez and Goldberg 1976) may determine the amplitude of J\text{U}, hence, the amplitude and direction of the Lorentz force. Reversing the polarity of the magnetic field (direction of B) also changes the direction of the Lorentz force. The Lorentz force should be zero in the absence of magnetic field or ion currents (for example, in patients with labyrinthectomy).

It was predicted that if MVS is due to the Lorentz force, the direction and amplitude of the resultant nystagmus should be proportional to the amplitude and direction of the Lorentz force. In support of these predictions, Roberts and colleagues (2011) found: 1) the nystagmus was present without head movements in the magnetic field; 2) head orientation with respect to the magnetic field and gravity changed the amplitude and direction of the nystagmus; 3) increasing the magnet strength increased the slow-phase eye velocity of the nystagmus without affecting its direction; 4) nystagmus rapidly decayed when the healthy subjects were brought outside the MRI scanner; and 5) MRI-induced nystagmus was not present in patients who had no labyrinthine function.

**Unique stimulus profile of MVS.** MVS has a unique profile compared with rotational head movements (e.g., transients, sinusoids, or constant velocity rotation). Constant velocity rotation causes transient cupular deflection followed by recovery at the rate of cupular elastic time constant. The slow-phase eye velocity of compensatory vestibulo-ocular reflex (VOR) decays with relatively prolonged time constant due to the involvement of central velocity storage mechanism (Raphan et al. 1979). Figure 1, A–D, depicts simulation of a constant velocity rotation using the simplified model of VOR (Leigh et al. 1981).

Evidence to date suggest that constant cupular deflection due to the Lorentz force is analogous to the constant acceleration (i.e., velocity increasing) rotations (Fig. 1E). Simulated velocity increasing head rotations, resultant cupular movement, and slow-phase velocity of VOR are illustrated in Fig. 1, E–G. The onset and decay of slow-phase eye velocity of nystagmus during MVS resembles that during constant acceleration (velocity increasing) rotational stimulus (Fig. 1G) (Roberts et al. 2011; Trillenberg et al. 2002; Wilson and Melvill Jones 1976).

**Implications of MVS-induced nystagmus.** MVS has many implications for clinical and basic neuroscience. By changing the static position of the head in the magnetic field, one can stimulate different pairs of semicircular canals so that the pattern of nystagmus could be a clue to any deficits in a particular semicircular canal. The utricle appears to make an important contribution to the Lorentz force, so MVS might become a test for the function of the utricle. The ionic composition of the endolymph and the number of hair cells in the utricle and ampulla are important determinants of the amplitude of the Lorentz force (Eq. 1). Thus MVS can be used to monitor the effects of pharmacotherapy that affect the ionic concentration of the endolymph (e.g., treatment of Ménière’s disease with diuretics). The limitation of MVS as a diagnostic tool is that it is not portable, whereas caloric and galvanic stimulation can be practiced in office-based settings. Since MVS causes constant cupular deflection, it might also be a “functional model” for benign paroxysmal positional vertigo, which sometimes is due to adherence of displaced otocochlea on the cupula (cupulalithiasis) (Leigh and Zee 2006). MVS may facilitate rehabilitation in patients with vestibular dysfunction, providing the equivalent of a virtual reality vestibular stimulation. A potential key implication of MVS is in the interpretation of functional MRI (fMRI). MVS may alter the fMRI activation patterns of central vestibular pathways. The retinal image motion (due to nystagmus) may affect the fMRI activation pattern of the visual pathways (Roberts et al. 2011). Other parts of the brain may be activated related to adaptation to the constant vestibular stimulus, for example, the cerebellum.

To summarize, MVS is a unique way to stimulate the vestibular system. It has broad implications in future basic and clinical research.

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**DISCLOSURES**

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**AUTHOR CONTRIBUTIONS**

A.G.S. conception and design of research; A.G.S. analyzed data; A.G.S. interpreted results of experiments; A.G.S. prepared figures; A.G.S. drafted manuscript; A.G.S. edited and revised manuscript; A.G.S. approved final version of manuscript.

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