Dispatches

Neurophysiology: Vertigo in MRI Machines

Subjects of brain-imaging studies often report experiencing vertigo while in MRI machines; a new study shows that the magnetic field stimulates the vestibular sensors in the inner ear by a Lorentz force.

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People frequently feel vertiginous or dizzy in and around magnetic resonance imaging (MRI) machines [1]. A degree of anxiety is not uncommon when one's own organs are being scanned, so this sensation might be interpreted as psychogenic; the vertigo becomes more prominent at scanners with stronger magnetic fields, however, so the perception of vertigo is clearly related to a magnetic field mechanism. Where in the sensory nervous system does the magnetic field act to elicit vertigo? The most obvious candidate is the vestibular part of the inner ear [2], which contains sensors that detect angular and linear movements of the head.

Head rotations cause endolymph flow in the vestibular semi-circular canals, which bends the cupular membrane and deflects hair cells [3] (Figure 1). This mechanism, in turn, changes neural firing in the vestibular nerve. There are six canals, three in each ear, which act as three push-pull pairs; each pair detects rotations about a specific axis, and because the three pairs are spatially orthogonal, they can detect any head rotation. Acting via the fastest reflex of the body, the vestibulo-ocular reflex (VOR), stimulations of the vestibular organs lead to eye movements. The VOR acts as an 'image stabilization system' to keep the eyes steady in space when the head is moving to prevent image blur on the retina; but even if the head is not moving, stimulation of the vestibular organs produces eye movements. This fact is used diagnostically by measuring eye velocity after irrigation of the outer ear of supine patients with warm or cold water, which leads to convection currents in the endolymph within the semicircular canals and stimulation of the vestibular organs [4]. Thus, if healthy subjects show ocular drift in the absence of head movements in an MRI scanner, they quite possibly are experiencing non-physiologic vestibular stimulation.

As they report in this issue of *Current Biology*, Roberts *et al.* [5] analysed the eye movements of healthy subjects and patients with vestibular deficits lying in MRI scanners. All subjects with intact vestibular function showed continuous eye drift in a direction that was determined by the polarity of the magnet and the position of the head within the scanner. Normally, as was also the case in these subjects, ocular drift is interrupted by resetting rapid eye movements in the opposite direction. The succession of drift (slow phases) and fast phases is called nystagmus (see Figure 1B of Roberts *et al.* [5]). The velocity of eye drift, and hence the intensity of nystagmus, increased with the strength of the magnetic field. In patients lacking vestibular function, however, the eyes did not move and no vertigo was induced despite the presence of the same magnetic field. Thus, the magnetic field of an MRI scanner must be acting on the brain via the vestibular labyrinth. Importantly, these experiments were done without scans being conducted — the magnetic field was static throughout the measurements. Also, subjects were measured in darkness, and therefore the eye drift could not be suppressed by visual fixation of an object.

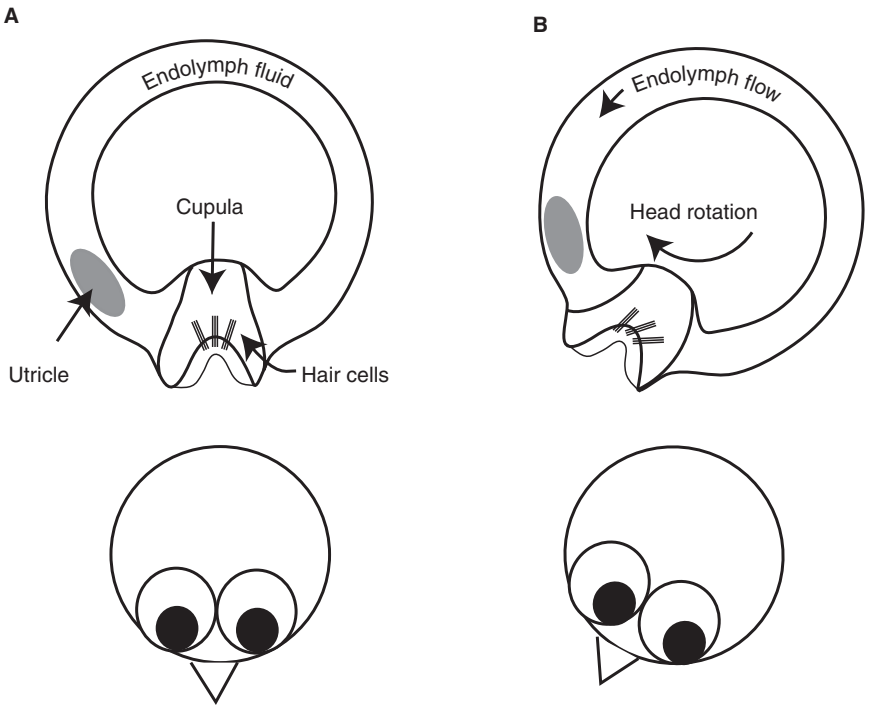

Figure 1. Head rotation is sensed by the semi-circular canals and leads to reflexive eye movements in the opposite direction.

(A) The semi-circular canals are filled with endolymph fluid, with hair cells embedded within the cupula. (B) During head rotations, the endolymph fluid lags behind the canal owing to its high inertia, creating a relative fluid flow that deflects the cupular membrane and hair cells. The change in the discharge firing rate of the hair cells caused by their bending leads to compensatory eye movements (the vestibulo-ocular reflex) that allow the eyes to keep looking at an object of interest and minimize image blur.

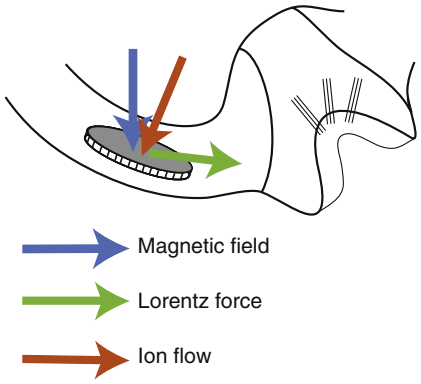

Figure 2. The Lorentz force created in the horizontal semicircular canal arises largely from ionic current flow that is perpendicular to the utricle, and the magnetic field vector. The Lorentz force is given by the cross product of these two vectors and is therefore perpendicular to the plane spanned by the vectors. Depending upon the orientation of the utricle, that is, the position of the head in the scanner, the magnitude of the Lorentz force changes. The Lorentz force deflects the cupular membrane and hair cells of the horizontal canal, producing a vestibular signal of horizontal head rotation.

Roberts *et al.* [5] went one step further to uncover the exact mechanism of MRI-induced eye movements and vertigo in MRI scanners. They tested different hypotheses based on the induced eye drift in different conditions. Dynamic bio-magnetic mechanisms could be discarded, because the ocular drift was permanent as long as the subjects were in the scanner. The only possible static bio-magnetic mechanism that could explain the dependence of the constant vestibulo-ocular response on the polarity and strength of the magnet is a Lorentz force, a force produced by the interaction of an electric current flow and a magnetic field. The endolymph in the vestibular labyrinth has a high concentration of potassium ions, and so carries an ionic current.

From geometric considerations and estimations of force magnitude, Roberts *et al.* [5] localized the MRI-induced Lorentz force mainly at the utricule, a structure that normally detects linear acceleration including gravity (Figure 2). Ions are exchanged between the potassium-rich endolymph and the hair cells [6]; the average net ionic flow at rest enters each utricle perpendicular to its surface, which is pitched upward by about 30 degrees backward with the head in supine position. The interaction between the ionic flow and the magnetic field produces the Lorentz force, which is given by the cross-product between flow and field, and is therefore perpendicular to the plane spanned by the flow and the field. With the head pitched slightly forward from supine, the Lorentz force becomes minimal. The Lorentz force is directed along the horizontal semicircular canal at the level of the cupula. This physiologic pressure sensor is therefore stimulated by the Lorentz force as if the head was rotated with a constant acceleration in the plane of the canal to the left or right [7], depending on the pitch angle and the polarity of the magnet.

The most important ramification of the findings by Roberts *et al.* [5] is that a pure functional MRI resting state usually does not exist, as the magnetic field produces constant vestibular stimulation with ongoing nystagmus and vertigo. As researchers increase the strength of the magnets, this problem becomes more serious. A consequence is that nearly every functional MRI study is contaminated with neural activity related to perception (vertigo) and eye movements (nystagmus) caused by vestibular stimulation. If experiments are performed in the light and subjects have their eyes open, the ocular drift activates the visual system and eye movement areas that suppress the drift. Comparisons of fMRI studies among different laboratories must therefore take into account the strength and polarity of the magnetic field, the position of the head relative to the magnetic field and whether experiments were performed in darkness with ongoing vestibular nystagmus or in the light with visual suppression of nystagmus. Future MRI studies should probably aim to position subject's heads slightly pitched forward, where Roberts *et al.* [5] found the minimal Lorentz force, perhaps even by finding the optimal position for each subject by monitoring nystagmus. This would aid the interpretation of functional MRI studies, as well as increase subject and patient comfort within the scanner.

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Nuclear Architecture: The Cell Biology of a Laminopathy

Lamin mutations cause muscular dystrophies, but the mechanism is unclear. A new study shows that lamin mutant worms display muscle-specific defects linked to altered subnuclear localization of heterochromatin, leading to altered gene expression.

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The nuclear lamina is a fibrous network of proteins associated with the inner nuclear envelope. The primary structural components of this network are the nuclear lamins, intermediate filament proteins that form a very stable