

## Editorial

# Revisiting the Cause of the Attacks of Vertigo During Meniere's Disease

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The mechanism of the attacks of vertigo occurring during the course of Meniere's disease has been a matter of speculation for over a hundred years. An early theory was that vasospasm of intracochlear blood vessels caused the attacks and treatment options included vasodilators and even cervical sympathectomy was undertaken. The most cherished theory [1] has been that endolymphatic hydrops leads to a rupture of Reissner's membrane causing potassium rich endolymph to mix with perilymph. The theory was based on histological findings in Meniere's affected ears which appeared to show healed ruptures of Reissner's membrane. It was proposed that the high potassium concentration within the extracellular fluid surrounding the primary afferent vestibular fibres causes abnormal vestibular activity until the ionic balance is restored.

There are conflicting views regarding precisely how this putative ionic imbalance should play out in regards to vestibular function, and similarly, there is some confusion regarding the time-course of vestibular changes during a vertigo attack in a Meniere's sufferer. Brown et al., (1988) [2] monitored vestibular function in guinea pigs whilst increasing the K<sup>+</sup> content of the perilymph, and demonstrated a systematic change in vestibular function, beginning with an initial irritative phase (an increase in spontaneous neural activity) lasting only a few minutes, followed by a paralytic phase lasting over an hour. In contrast, Klis and Smoorenberg [3] showed in guinea pig, a simultaneous increase in perilymphatic K<sup>+</sup> concentration, together with a reduced Na<sup>+</sup> concentration while keeping the osmolarity constant increased the endolymphatic potential.

However, there are some glaring observations that do not support the rupture theory of vertigo attacks, mostly owing to observations of cochlear function. First, a cochleostomy (a surgery proposed by Schuknect [4] where a surgical hook is introduced through the round window to perforate the basilar membrane) does not induce vertigo, even under local anaesthesia, even though cochlear function is abolished (Gibson 1991) [5]. Second, although a rupture of Reissner's membrane would be expected to alter the endocochlear potential, there appears to be little change in cochlear sensitivity during a vertigo attack. McNeil et al. [6] studied pure tone audiometry undertaken in a number of Meniere's disease sufferers before, during and after attacks of vertigo. No significant changes to the audiogram were

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observed during the attacks. Moreover, Gibson [7] reported the electrocochleogram obtained during attacks of vertigo in three subjects and showed only minimal changes in the waveform.

Recent animal studies also fail to support the rupture theory. Brown and colleagues [8] gradually injected artificial endolymph with a fluorescent marker into scala media of anaesthetised guinea pigs. Functional changes during the injection mirrored those observed clinically, demonstrating a low-frequency hearing loss and an increase in the summing potential in electrocochleography responses. At a particular volume, cochlear function spontaneously improved, followed shortly by a loss of vestibular function. The simplest explanation of these results would be a rupture of the Reissner's membrane, however post-mortem imaging failed to identify any ruptures [8], nor was there any leakage of the fluorescent fluid into the perilymphatic compartments. Moreover, within any given animal, several endolymph injections could be performed in succession, each causing an apparent increase in hydrostatic pressure induced functional changes. Such repeatable changes would not be possible if a rupture was present in the membranous labyrinth. In a more recent study by Brown and colleagues [9] using light sheet fluorescent microscopy, after injection of 25  $\mu$ L endolymph containing fluorescein, a sudden inflow of endolymph into the utricle was observed when this comparatively large volume had been added. However in Meniere's subjects the endolymphatic duct is narrowed and a smaller volume could have the same effect.

Tonndorf [10] speculated in 1983 that the attacks of vertigo might have a mechanical cause rather than a chemical origin to account for the differing timing. Similarly, Gibson [11] has argued for several years that the attack of vertigo is due to endolymph within the pars inferior suddenly entering the utricle causing stretching of the cristae of the semicircular canals.

The osseous endolymphatic duct is narrower in Meniere's subjects compared with a cohort of normal subjects [12] and the membranous endolymphatic duct cannot expand and this may slow the longitudinal flow of endolymph if a sudden surge of endolymph in the cochlear duct occurs. It is difficult to reproduce this in animal studies.

In the past, it was thought that endolymph was draining

continuously into the endolymphatic sac and a blockage of the endolymphatic duct led to the formation of endolymphatic hydrops and hence to the 'ruptures'. This theory has now been disproved and studies now suggest that drainage of endolymph to the endolymphatic sac only occurs when an increase in endolymph volume occurs [13].

Previously it had been considered that the endolymphatic sac was a hollow structure which passively received endolymph. It is now known that the human endolymphatic sac is a series of tubules [14,15] which link the inner ear to the lymphatic system capable of removing debris, viruses, etc. and it is immunologically competent [16]. The human endolymphatic sac lies partly outside the petrous bone on the dura and partly inside the petrous bone where it is part of the endolymphatic duct. This intraosseous section is the most active section. The endolymphatic sac appears to actively attract endolymph into its lumen linked with a secretion of glycoproteins and macrophage activity [17].

Perhaps when debris needs to be cleared from the cochlea to the endolymphatic sac, the cochlea can signal the endolymphatic sac to produce a hormone which increases endolymphatic volume to initiate longitudinal flow. Quotrop and colleagues [18] isolated a substance they named as saccin but there could be other hormones involved. Recently Møller and co-workers [19] have demonstrated the presence of neuronal fibres and neurotransmitter receptors in the human endolymphatic sac. Gibson [20] removed the extraosseous portion of the endolymphatic sac as a treatment for Meniere's disease and noted that the attacks of vertigo ceased or were greatly reduced although there was still endolymphatic hydrops present. Recently Saliba and colleagues [21] have published a further series showing similar outcomes. This would explain why animals who have the endolymphatic sac removed develop endolymphatic hydrops but do not suffer attacks of vertigo.

The time has come to rethink the mechanism of the attacks of vertigo which occur during the course of Meniere's disease. Perhaps this will lead to better ways of controlling or curing the disease.

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