HOW DO MIDDLE EAR MUSCLES PROTECT THE COCHLEA? RECONSIDERATION OF THE INTRALABYRINTHINE PRESSURE THEORY

Andrew Bell

Research School of Biology, College of Medicine, Biology and Environment, The Australian National University, Canberra, ACT 0200, Australia

Abstract

The middle ear muscles are part of a control system for regulating the acoustic input to a supersensitive detector, the cochlea, preventing overload and damage. Yet there is a long-standing paradox. When Békésy measured sound transmission through the middle ear of cadavers, he found that acoustic transmission was not affected when the annular ligament was stretched by pressure. Similarly, reflex activation experiments often show only a few decibels of attenuation, assumed to be due to stiffening of middle ear joints and ligaments. In contrast, psychophysical experiments reveal attenuations of 30 dB or more when the middle ear muscles are voluntarily contracted. How can the difference be explained? This synthesis paper shows how the paradox can be resolved by reconsidering a theory put forward by Gellé in the 19th century. According to Gellé's intralabyrinthine pressure theory, which has long been dismissed, the purpose of the middle ear muscles is to press the stapes inwards and raise the hydraulic pressure in the labyrinthine fluids, thereby regulating cochlear sensitivity. The focus of this review is to revisit the theory and show how it can explain a range of audiological findings. The theory is updated and the hypothesis made that static pressure in the cochlear fluids is sensed by the outer hair cells, which are in continuous hydraulic connection with the stapes. It is this factor which reduces the gain of the cochlear amplifier and provides rapid and effective overload protection. The case is made that the intralabyrinthine pressure theory deserves renewed attention.

КАК МЫШЦЫ СРЕДНЕГО УХА ЗАЩИЩАЮТ УЛИТКУ? ПОВТОРНОЕ РАССМОТРЕНИЕ ТЕОРИИ ВНУТРИ-ЛАБИРИНТНОГО ДАВЛЕНИЯ

Резюме

Мышцы среднего уха – это часть системы управления регулирования акустических даных, поступающих в очень чувствительный датчик, улитку, предотвращая перегрузку и повреждение. Все же существует продолжительный парадокс. Когда Бекеши измерил звуковую передачу через среднее ухо трупов, он открыл, что акустическая передача не была затронута, когда кольцевая связка была растянута давлением. Точно так же эксперименты активации рефлексов часто показывают только несколько децибелов ослабления, предположительно из-за напряжения суставов и связок среднего уха. Напротив, психофизические эксперименты показывают ослабления 30 дБ или более, когда мышцы среднего уха самостоятельно сокращены. Как можно объяснить полученную разницу? Данная синтезированная работа показывает, как парадокс может быть разрешен, пересматривая теорию, сначала выдвинутую Желле в 19-ом веке. Согласно теории внутри-лабиринтного давления Желле, которая долгое время не бралась во внимание, цель мышц среднего уха состоит в том, чтобы нажимать стремя внутрь и поднимать гидравлическое давление во лабиринтной жидкости, таким образом регулируя кохлеарную чувствительность. Целью этой работы является повторное обращение к теории и демонстрация того, как она может объяснить ряд аудиологических открытий. Теория обновлена, и поставлена гипотеза: статическое давление на кохлерную жидкость ощущается волосковыми наружными клетками, которые находятся в непрерывной гидравлической связи со стременем. Именно этот фактор уменьшает увеличение кохлеарного усилителя и обеспечивает быструю и эффективную защиту от перегрузки. Это говорит о том, что теория внутри-лабиринтного давления заслуживает возобновленного внимания.

¿CÓMO PROTEGEN LOS MÚSCULOS DE OÍDO MEDIO LA CÓCLEA? RECONSIDERACIÓN DE LA TEORÍA DE PRESIÓN DENTRO DEL LABERINTO

Extracto

Los músculos de oído medio son la parte de un sistema de control para regular los datos acústicos que entran a un detector extraordinariamente sensible, la cóclea, previniendo la sobrecarga y el daño. Aunque hay una paradoja antigua. Cuando Békésy

midió la transmisión del sonido a través del oído medio de los cadáveres, encontró que la transmisión acústica no fue afectada cuando el ligamento anular fue estirado por la presión. De manera similar, los experimentos de activación de reflejos a menudo muestran sólo unos decibeles de la atenuación, que se supone de ser debido al agarrotamiento de las articulaciones y los ligamentos en el oído medio. Por el contrario, los experimentos psicofísicos revelan atenuaciones de 30 dB o más cuando los músculos del oído medio son contratados voluntariamente. ¿Cómo esta diferencia puede ser explicada? Este trabajo de síntesis muestra como la paradoja puede ser resuelta reconsiderando una teoría propuesta por primera vez por Gellé en el siglo 19. Según la teoría de Gelle de presión dentro del laberinto, que ha sido ignorada durante mucho tiempo, el objetivo de los músculos del oído medio es presionar el estribo hacia adentro y elevar la presión hidráulica en los líquidos laberínticos, regulando así la sensibilidad coclear. El foco de esta revisión es analizar de nuevo la teoría y mostrar como ella puede explicar una variedad de conclusiones audiological. La teoría es actualizada y la hipótesis es hecha: la presión estática en los líquidos cocleares es sentida por las células ciliadas externas, que están en la continua conexión hidráulica con el estribo. Este es el factor que reduce la ganancia del amplificador coclear y proporciona la protección contra sobrecarga rápida y eficaz. Es importante que la teoría de la presión dentro del laberinto merece una atención renovada.

Background

The human middle ear is an intricate arrangement of membranes, bones, muscles, and ligaments (Figure 1). The device functions as a mechanical transformer helping to bring the acoustic impedance of air closer to the impedance of the cochlear fluids [1]. The middle ear has been closely studied since audiology began, but even now its functions are not fully understood [2], not least because the ear can respond to acoustic motions of subatomic dimensions. At hearing threshold, the eardrum moves of the order of picometres [3]. Understanding how such minute movements are transmitted through a delicate system of bones and joints stretches experimental apparatus to its limits.

This synthesis paper concerns itself with one particular aspect of the middle ear, and that is the function of the middle ear muscles, the two smallest skeletal muscles in the human body: the tensor tympani attached to the malleus, and the stapedius, only 1 mm long, attached to the stapes (Figure 1). Years of research have made it plain that the muscles are involved in attenuating loud sounds [4–7], so that the delicate sensing elements in the cochlea are not overloaded or damaged. The question addressed here is, how is this achieved?

The standard answer is that when the middle ear muscles contract, they stiffen up the joints and ligaments, particularly the annular ligament surrounding the stapes, causing an increase in mechanical impedance and hence reducing sound transmission to the cochlea [8–11]. But as Békésy noted, there is only about a 5% alteration in impedance when the muscles contract (p. 72) and he could not find any physiologically important change (p. 203). Our knowledge has since expanded [4–7], but the general picture remains the same – the middle ear muscles appear to provide only a minor degree of protection against loud sounds. Most animal-based studies find a change in impedance of around 5–10 dB [12,13], while human studies show an effect of only 1–2 dB over the range 0.06–11 kHz [14,15]. Changes in cochlear potentials are somewhat larger and more variable, and are discussed in the next section. This paper questions the idea that the purpose of the strategically placed middle ear muscles, with their complex anatomy and physiology, is to cause a minor change in sound transmission. Instead, this paper sets out what seems to be a much more effective mechanism: when the muscles contract they create a fast control signal in the cochlear fluids – hydraulic pressure.

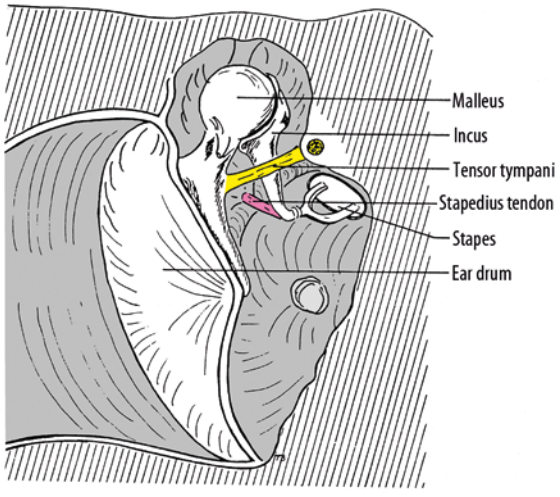

Figure 1. The human middle ear, showing the tensor tympani tendon (yellow) and stapedius tendon (red). The muscles themselves are recessed in bone. According to the intralabyrinthine pressure theory, activation of the tensor tympani muscle pulls the malleus and ear drum inwards and pushes the stapes into the oval window, protecting the cochlea by raising the pressure of fluids inside. It is suggested that the pressure controls the gain of the cochlear amplifier. Modified from [80] and used with permission.

The intralabyrinthine pressure (ILP) theory of middle ear muscle action dates from the 19th century, and, although simple and elegant, it was never widely accepted. By the middle of the 20th century it was totally dismissed. The theory proposes that contraction of the middle ear muscles causes the stapes to press inwards on the cochlea's fluid contents, raising their pressure. In this paper, the pressure is taken to be a key parameter which controls the gain of the cochlear amplifier via its action on outer hair cells – sensing cells which, importantly, are in continuous hydraulic connection with the cochlear fluids. It is this action which rapidly, silently, and with minimum observable movement, protects the cochlea's supersensitive detectors.

In the 1880s, Gellé developed a theory of why action of the stapes should produce lower hearing acuity [16,17]. He had observed that pressure applied to the ear canal led

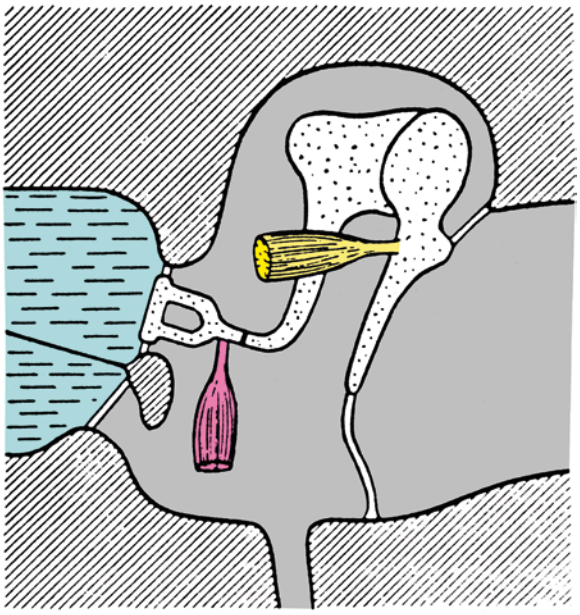

Figure 2. When the tensor tympani muscle (yellow) contracts, it pulls on the malleus and forces the stapes into the oval window, raising the pressure of the incompressible cochlear fluids (blue) and distending the round window. The diagram shows how contraction of the middle ear muscles produces a similar effect to positive pressure in the ear canal or negative pressure in the middle ear cavity – they all cause inward stapes motion and a rise in intracochlear pressure. Adapted from [99] and used with permission.

to a loss in hearing sensitivity, and he proposed that the stapes in a similar way produced a "pressions centripètes" in the labyrinth which caused a reduction in cochlear sensitivity. Figure 2 illustrates the mechanism: contraction of the tensor tympani draws the whole middle ear system inwards and presses the stapes into the oval window. According to Borg [11], the labyrinthine pressure-regulation theory can be traced back to Politzer in 1861, who noted that electrical stimulation of middle ear muscles led to changes in middle-ear pressure and, presumably, to changes in labyrinthine pressure. Similarly, Borg also notes that Lucae in 1866 proposed that contraction of the tensor tympani affected low frequency hearing via labyrinthine pressure, and this was taken up and promoted by Zimmerman in the early 1900s who claimed that pressure somehow controlled the vibration of the basilar membrane fibres.

What happened to Gellé's theory? Borg mentions [11] that Kato in 1913 was the first to discredit the hypothesis by observing no displacement of the round window membrane during tensor tympani contractions. Of course, the displacements involved are minute (micrometres or less), and with the instruments available at the time Kato failed to see an effect. Today, that motion has indeed been seen [18]. However, it was probably Békésy's traveling wave theory [8] that eventually caused the ILP theory to be discarded, for the mechanics of the passive traveling wave do not depend on static pressure. According to the traveling wave theory, the pressure *difference* across the partition is the effective stimulus, and static pressure is not important. For most of the 20th century, the cochlea was considered a passive transducer, and there was no conception of active mechanics that might be sensitive to pressure.

Nowadays, the situation is different, and the cochlea is seen as an active transducer [19]. Although the traveling wave theory remains at the core of cochlear mechanics, there is now room for additional active processes. It is now possible to consider that the outer hair cells, responsible for the activity, could be affected by static pressure. This proposed sensitivity to static pressure is a logical counterpart to a recent speculation that outer hair cells are pressure sensors and respond to the fast pressure wave signal [20,21]. Extending the idea, the proposal is that outer hair cells respond to static (d.c.) pressure as well as alternating (a.c.) pressure.

In the version of the ILP theory put forward here, the d.c. pressure is the factor controlling the gain of the cochlear amplifier, which is part of a positive feedback loop involving the outer hair cells, which themselves are responsive to a.c. pressure. Simply put, pressure acts to squeeze the compressible outer hair cells. In the following, the ILP theory is reexamined and the arguments for it are assessed. The conclusion is that the arguments commonly raised against the ILP theory are not decisive. The ILP theory has the potential to unify much audiological understanding and deserves renewed attention.

A paradox

There is a paradox surrounding middle ear sound conduction, and it begins with Békésy. In his monumental work [8] he took a freshly excised ear from a cadaver and covered its round window with a hollow rubber tube that led to the ear of a living observer (himself). When sound was applied to the ear drum of the preparation, it traveled through the middle ear and cochlea and oscillated the round window membrane. The observer could therefore hear this oscillation – or, more sensitively, hear the null this sound produced when it interfered with another sound source, of appropriate amplitude and phase, also connected to the tubing. Békésy raised the pressure in the cochlea and discovered, based on multiple preparations, that even when the pressure in the cochlea was raised to 4 atmospheres – a point at which the round window or blood vessels burst – there was *no change* in the sound level at the round window (p. 433 of [8]). He concluded that sound transmission through the middle ear, including the stapes and annular ligament, was immune to pressure effects. The inference is that stretching of the annular ligament – from force exerted on one side or other of the oval window – has no appreciable effect on sound transmission.

This decisive experiment has been duplicated [22] with the same result: nothing happens acoustically until the round window bursts. In other experiments, pressure was replaced by force artificially applied to the middle ear muscles [23], but it gave similarly minute effects: an attenuation of less than 1 dB when a 1 g force was applied (see also p. 23 of [5]). Likewise, the generally small impedance changes

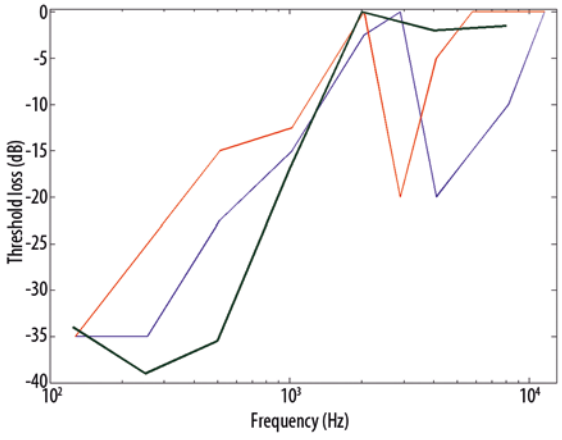

Figure 3. Effect of voluntary contraction of middle ear muscles on hearing threshold. The black line is the average of 8 ears studied by Reger [30]; the red and blue lines are from Smith [29] and show the loss in the right and left ears of a single subject. Data replotted from [29,30].

observed under middle ear manipulations [8,24–27] support the idea that the annular ligament has only a minor effect on sound transmission. Based on otoadmittance measurements, one acoustic model of the middle ear reflex [28] calculated that the corresponding sound attenuation would be about 2 dB(A), a figure that would provide "negligible" protection.

At the same time, there are psychophysical experiments using living subjects which give totally different results. A key piece of early work was that of Smith [29], who measured hearing thresholds before and after a subject contracted his middle ear muscles (the subject was one of those individuals who could voluntarily perform this manoeuvre[1]). The effect of the contraction was large (Figure 3), causing a diminution in threshold of about 30 dB at 128 Hz and 20 dB between 3 and 4 kHz. These findings were corroborated in 1960 by Reger [30] on a group of 4 subjects (8 ears) and the average attenuations are also shown in Figure 3.

What are we to make of these findings? Békésy's work says that, acoustically, pressure difference across the oval window (leading to stretching of the annular ligament) has little, if any, effect; on the other hand, psychophysical experiments suggest that the muscles' exertions are creating major attenuations. In Békésy's time it was not reasonable to consider that raised intracochlear pressure was causing the attenuation. First, the traveling wave theory leads to the natural inference that static pressure has no effect on cochlear mechanics – in a passive system it is all a matter of mass and stiffness of the basilar membrane. Second, the results of more technically advanced experiments, involving simultaneous measurement of cochlear input impedance and of cochlear microphonics, were enough to suggest that changes in sound transmission due to annular ligament stiffness were happening after all. The most influential of these is now critically assessed.

In 1982, Lynch and colleagues [25] studied the input impedance of the live cat cochlea using the Mössbauer technique. The middle ear was removed, leaving only the stapes on which a radioactive source was placed. They found (their Figure 10) that there was about a 5 dB change (below 200 Hz) in input impedance of the cochlea and stapes under a static pressure of 7 cm of water (0.7 kPa). This 5 dB might well be due to stretching of the annular ligament, they thought, given other evidence (their Figure 7) that drying of the ligament also changed the impedance. However, it is worth remembering that this 5 dB change might relate to the cochlea and round window as well, since both of these are in series with the annular ligament. Attributing the change to additional sources is supported by the observation that the authors found considerably larger concurrent changes (10 dB) in cochlear potentials as static pressure was varied by 7 cm of water.

At any rate, these observations opened the way to attributing changes in cochlear impedance to changes in annular ligament stiffness. But there is a problem here. In order to infer annular ligament stiffness, *the authors used changes in cochlear potentials as a gauge of cochlear sound attenuation*. This assumption confounds the effects of middle ear impedance and the effects of intracochlear pressure. In other words, it is a mistake to assume that when a cochlear potential shows a decrease under the action of the middle ear muscles, that this must be due solely to an increase in cochlear impedance (and in particular, that of the annular ligament).

Lynch and colleagues were not the first to interpret cochlear potentials in this way [5,6,12,31], but the underlying logic needs to be made explicit. If it is assumed that reductions in cochlear potentials are due to the middle ear (specifically the annular ligament, although other components may contribute in lesser measure), this automatically discounts the possibility that the observed effects are due to intracochlear pressure acting directly on the hair cells, which is what the ILP theory supposes.

Significantly, there appears to be no work conclusively disproving the ILP theory. On the contrary, results in the literature that purport to show that the cochlear response has been attenuated by middle ear muscle contraction can actually be interpreted as being due to the direct effect of hydraulic pressure on the cochlear receptors, not the effect on middle ear sound transmission.

Moreover, while in general there are parallel changes in admittance and cochlear potentials, notably at frequencies below 1 kHz, there are also puzzling anomalies, particularly at higher frequencies. As an example, at a frequency of 2 kHz, Møller (Figure 15 of [12]) saw an *increase* in admittance at the same time as the cochlear microphonic *decreased*. Similar anomalies can be seen at particular frequencies in a number of his other figures when changes in cochlear microphonic are compared to changes in admittance. This and related work indicate that the cochlear microphonic may not be a reliable measure of sound transmission. Møller expressed it succinctly when he said, speaking of the effect of ear pressure changes in the ear

1. About half the population can contract the muscles by forceful closure of the eyelids [4], when a characteristic fluttering can be heard.

canal (Figure 18 of [6]), that there was a *qualitative* difference between sound transmission and middle ear pressure, meaning that something essential has been missed. This paper suggests that the crucial missing factor is the direct effect of pressure on the cochlea itself.

So while the standard annular ligament theory can work below 1 kHz, it has difficulty accounting for attenuations at higher frequencies, like those between 2 and 4 kHz seen in Figure 3, and it has real problems accounting for *increases* in responses at certain frequencies (e.g. [32]).

Nevertheless, it has now become standard to assume that changes in cochlear potentials in living ears are due to sound transmission effects, not pressure. Based on the standard traveling wave model, the natural inference is that sound transmission is attenuated by the same amount in dead and living ears.

In 1986 Pang and Peake [33] cemented that way of thinking. They asked the pertinent question "How do contractions of the stapedius muscle alter the acoustic properties of the ear?" and came to the conclusion that it was stretching of the annular ligament. Their reasoning was based on calculations that showed a direct match between changes in stapes admittance and change in cochlear microphonic (their Figure 5); the authors interpreted this result in terms of sound transmission changes directly caused by changes in stapes impedance. They overlooked the possibility that when the annular ligament is stretched by stapes displacement, it also increases intracochlear pressure. They reasoned that acoustic admittance would vary with stapes displacement, basing their calculations on the curve of Figure 10 of the earlier Lynch work – which was, as described above, based not only on impedance changes but also on cochlear potential changes. In other words, it is possible that the key assumption underlying Pang and Peake's work – that changes in cochlear microphonics are due entirely to changes in physical sound input – could be erroneous.

The general idea to be presented here is that most, if not all, changes in cochlear microphonic potential that are observed when the middle ear muscles are activated are due to increases in intracochlear pressure. Perhaps microphonics do not indicate changes in "sound transmission" but instead reflect the operation of a rapid control signal acting on all the thousands of outer hair cells which together generate the cochlear microphonic.

Having outlined the issues, the rest of this paper looks in more detail at the reasons for and against the ILP theory and, given what we now know about otoacoustic emissions, puts the theory in a contemporary perspective.

Arguments against the ILP theory

Two major reasons have already been mentioned as to why the ILP theory was not positively viewed: a reliance on traveling wave theory and indications that pressure had no physical effect on sound transmission. Both are possibly mistaken, but it is illuminating to go back to the literature and see what reasons were given for rejecting the ILP theory.

In 1960, Huizing [34] gave a summary of the situation (his p. 20). Gellé's *pressions centripètes* theory has proved untenable, he said, for three major reasons, and they are listed below.

(1) A negative change of pressure [in the ear canal] has almost the same influence as a positive

The force of this argument is hard to understand, as it applies equally well to the ILP theory as to the annular ligament theory. Assuming that positive and negative pressure changes in the ear canal lead to corresponding pressure changes in the cochlear fluids, there does not seem to be any fundamental reason to think that the effect of pressure on the cochlea's sensing elements might not be more or less symmetrical. Certainly, contraction of the tensor tympani leads only to an increase in pressure, but the inner ear transducers themselves may only respond to the absolute magnitude of the intracochlear pressure (a negative ear canal pressure is, via the mechanics of the middle ear, likely to give rise to a negative intracochlear pressure). Note that annular ligament stretching also occurs symmetrically, and this is the basis of the standard model of how otoacoustic emissions change in frequency under imposed ear canal pressure [35].

Møller in his acoustic impedance studies [12] found a broad symmetry in the impedance and cochlear microphonics of a cat ear as air pressure in the ear canal was raised or lowered, giving V-shaped curves (his Figures 9–11). On closer inspection, however, there are marked deviations from symmetry, and the effect of negative middle ear pressure – which will lead to increased intracochlear pressure in the same way as a muscle contraction will – are particularly informative. Under negative pressure, the acoustic resistance entirely disappears (his Figure 9), while the acoustic impedance diverges from the cochlear microphonic (his Figures 10, 11). The first observation means that the cochlea's sensing elements, which give the ear its characteristic resistance [12,15], have been effectively disengaged from the system, while the second means that the cochlear microphonics have been reduced much more than the impedance has. For example, Møller found that a negative pressure of 15 cm of water (1.5 kPa) reduced impedance by 12 dB while reducing microphonics by 24 dB. These results are explicable in terms of the ILP theory but are hard to explain otherwise.

(2) No decrease of auditory acuity occurs if the labyrinthine pressure is increased via another method, for example by congestion of the jugular veins

Taken at face value, this is a powerful argument against the ILP theory, if it were true. It is not clear what evidence Huizing was referring to when he wrote this assessment in 1960, but evidence now clearly points the other way. Compression of the neck veins does indeed increase intracranial pressure, and it results in impedance changes observable at the ear [27]. In such cases, the experimental subject usually hears "a clear attenuation" of the 550 Hz probe tone, directly refuting point 2. Another finding of direct relevance to the ILP theory is that before, during, and after this rather uncomfortable procedure, a brief contralateral tone was used to elicit the acoustic reflex, and immediately

the ear under study showed a simultaneous jump in impedance. Notable, however, is the observation (Figure 2 of [27]) that the amplitude of the reflex response steadily diminished (in terms of impedance) as intracranial/intralabyrinthine pressure rose, until it eventually disappeared at the point when the pressure and impedance reached a plateau. This "adaptation" invites the interpretation that the impedance might be a secondary effect of pressure. If so, and if pressure is the controlling factor for cochlear gain, then once pressure has built up to some particular limit, further efforts to increase pressure will be ineffective. Such a model provides some understanding of why reflex effects sometimes appear to be so small: it is a matter of the experimental situation not providing sufficient time for reflex-induced pressure to dissipate.

This raises the issue of long time constants and the patency of the cochlear aqueduct, and these are discussed in the section on page 18. The particular problem here is that the patency of the cochlear aqueduct, by which pressure is dissipated, is limited and variable ([36] and discussed in [37]), and it can take some minutes for pressures to equilibrate [37,38]. Since jugular veins are not normally compressed for minutes at a time, this might provide some basis for Huizing's argument.

The other issue is the magnitude of the maximum effective pressure. Normally, variations in cerebrospinal fluid pressure are likely to be no more than some tens of centimetres of water [39], and Klockhoff's work suggests a functional limit of about 50 cm of water [27]. This limit tallies with the maximum force that the tensor tympani muscle can exert, about 1 gram (p. 194 of [10]). If such muscular effort is transferred through the middle ear chain to the stapes, this creates a force of 1 g over a footplate area of 3 mm^2, which is a pressure of about 3,000 N/m^2 (3 kPa or 30 cm of water).

Further evidence against point 2 is that people suffering from pathologically raised intracranial pressure suffer an average hearing loss of about 30 dB [40]. This loss can be alleviated by surgery, after which pressure reduces and hearing improves; one study found an average improvement of 8 dB below 500 Hz between preoperative and postoperative conditions [41]. Moreover, changes in labyrinthine pressure can be brought about by postural manipulations, and these changes in pressure can also affect hearing thresholds [42].

In addition, with modern equipment it is possible to detect subtle changes in cochlear function. Using otoacoustic emission techniques it can be shown that small pressure changes, brought about by alterations in posture, have measurable effects on the cochlea [37,43,44]. CSF pressure fluctuations from breathing and heartbeat can also be detected as frequency modulation of spontaneous OAEs [37]. These approaches and their integrating potential will be described more fully in the section on page 17, where it is argued that a direct effect of pressure on the outer hair cells provides a more consistent explanation than does stretch of the annular ligament.

Distinguishing these two possibilities is difficult because measurements of intracochlear pressure involve minute volumes of fluid. The fluid contents of the inner ear amount to only 200 µL, and the fluid, being mostly water, is virtually incompressible. This means that any gauging system must be of extremely high impedance to measure this pressure without disturbing the system, and certainly in the early days of auditory research this was not the case. Another way of viewing the problem is to appreciate that deflections of the stapes footplate due to muscle contraction are less than 20 µm [33,45], and these displacements will be matched by an equally small volume displacement (0.02 µL) of the round window membrane (p. 181 of [10]). Hence the middle ear muscles work against the compliance of the round window membrane as well as the annular ligament, and both these factors are important in controlling the input impedance of the cochlea, as Figure 10 of ref. [25] clearly shows. We begin to appreciate how extremely sensitive and finely graded must be the action of the middle ear muscles.

It is possible to infer a relationship between stapes displacement and intracochlear pressure. On the basis of raising perilymph pressure by 10 cm of water, Densert and colleagues [46] measured a tympanic membrane displacement of 2 µm; by reciprocity, a similarly small motion will raise intracochlear pressure by about that amount.[2] As calculated at the start of this section, such a pressure calls for the middle ear muscles to delicately exert a force of 0.3 g, a task for which these muscles are anatomically well suited [48].

(3) The loss in hearing remains the same for constant pressure [in the ear canal], while it may safely be assumed that a rise of liquor pressure, if any, will disappear very soon again

This inference again relies on patency of the cochlear aqueduct, but as already noted this is not often an open pathway, particularly in humans. Studies have shown that the aqueduct is of small diameter and filled with a meshwork of fibres [49]; moreover, it may possess a barrier membrane [50] or one-way valve [51] so that positive pressure takes a different time to die away than negative.

A particularly revealing experiment is one that measured the time course of the middle ear reflex to long-lasting tones [52] and it found that hearing thresholds steadily increased over 30 seconds (their Figure 2, shown here in Figure 4) and, for a 1 kHz tone, for 90 seconds or more (their Figure 3). The authors, Loeb and Riopelle, were surprised to find that after the activating tone in the left ear was switched off, hearing thresholds (at 1 kHz) in the right ear continued to increase, which suggests a time constant of several minutes. Unfortunately, Loeb and Riopelle did not appreciate the significance of this and only followed the course of the threshold for an additional 30 seconds – when it was still increasing. However a time constant of several minutes explains two peculiar findings. First, Loeb and Riopelle found that the threshold shifts were very small, only 3–5 dB. Second, the thresholds tended downwards, seemingly even before the stimulation began. The

2. Similarly, measurements by Ivarsson and Pedersen [47] give a relationship between change in intracochlear pressure and volume displacement of the oval and round windows of 0.12 µL/kPa.

explanation lies in appreciating that the authors conducted two experiments back to back, and in a balanced order of presentation, so that the effects of the 30-second activation (their Figure 2) were compounded with the effects of the 90-second activation (their Figure 3) and vice versa. Persistence of effects means that, half the time, each experiment began from an elevated baseline (so the measured effects were small) and also means that the thresholds were still in recovery mode before the new activating tone was applied.

When Rosowski and colleagues measured the middle-ear impedance of chinchillas [53] they noticed decreases in admittance associated with contraction of the middle ear muscles. Not only did the changes occur in time with the observed contractions, but in most cases the finding was that *the admittance change persisted*. In other words, middle ear impedance cannot be seen as exclusively the direct mechanical result of muscle contractions. Instead, this result points to a long-lasting build-up of pressure within the cochlea.

On the basis of studies of frequency drifts in otoacoustic emissions, which are a convenient, non-invasive measure of intracochlear pressure (see first point in next section), the time constant for pressure relief is at least tens of seconds [44] and often some minutes [54,55]. Békésy found that the effect of pressure in the ear canal on hearing persisted for more than 30 minutes (Figures 9–31 of [8]).

A long time constant offers an explanation of why experimental results have such variable outcomes. In the same way as our eyes require about 20 minutes to adapt to the dark, so too our ears require an appreciable time to adapt to quiet. Other confounding variables which also contribute to producing apparently small protective effects – ipsilateral reflexes and touch stimulation – will be discussed in later sections.

***

Wever and Lawrence in their 1954 text [10] give consideration to the ILP theory as part of a review of experimental findings on tympanic muscle action (their Chapter 10). They conclude that the theory must be set aside (p. 196) on the basis of two experiments. The first was Békésy's work on post mortem specimens, already summarised above, which showed that pressure had no effect on sound transmission – an important result which, when conventionally interpreted, misses the point. Yes, it has no effect on sound transmission, but in a live ear it directly attenuates cochlear amplifier gain. The second was work on a live monkey by Lempert and colleagues in 1949 [56]. The experimenters inserted a needle into scala media and increased the pressure up to 50 mm of mercury with a hypodermic syringe: there was no effect on the cochlear microphonic. As others have noted [57], the experiment involved only one animal, so the report is less than conclusive. The pressure was applied to the semicircular canal, not the cochlea, and the authors go to some length to point out that the semicircular canals are filled with a fibrous trabecular network. Later follow-up work on guinea pigs by McCabe and Wolsk [57] did find that cochlear potentials were affected by pressure, but by that stage it had little impact.

On page 196 of their work [10], Wever and Lawrence reveal what is perhaps the true reason for rejecting the ILP theory: inconsistency with traveling wave theory. They say that "on theoretical grounds" the ILP theory is not supported because the *change in density* of cochlear fluid produced by a pressure of 50 mm of mercury is only 0.03% – meaning of course that, in terms of traveling wave mechanics, the mass of fluid surrounding the basilar membrane would be negligibly affected.

Wever and Bray devote their Chapter 11 to the effect of changes in air pressure in the ear canal and middle ear cavity. Clear and significant effects are produced both in humans (psychophysically) and in experimental animals (cochlear potentials). The difficulty comes from separating the effect on the middle ear apparatus (including the ear drum) from the indirect effect of increased intracochlear pressure. In the final part of the text, Wever and Bray address the question of where the effects were produced, and the possible effect of ILP is considered. They describe specific experiments to test for ILP effects in the cat, and found (p. 210) a significant effect for bone conduction (up to 10 dB of attenuation *and gain*) when the ossicular chain was broken and pressure of 50 mm of mercury applied (breaking the chain was meant to rule out middle ear effects). Nevertheless, Wever and Bray saw these results as insignificant compared to the result of pressure applied to the ear canal when the chain was intact, so they concluded that the main effect of pressure is its effect on the drum membrane. However, what Wever and Bray ignored was the transformer action of the middle ear, which means that force applied to the ear canal is *multiplied* by the chain as it is conducted to the stapes footplate (the area of the ear drum is 17 times the area of the oval window). It could well have been that pressures in the labyrinth produced most of the effects they observed.

There have been other passing mentions of the ILP theory, all of them negative. In 1968, Zemlin's textbook [9] used the same two citations as did Wever and Lawrence to conclude (p. 387) that there was little evidence supporting the ILP theory. He states that Békésy (1936) (no doubt he meant 1942) and Lempert et al. (1949) each found that middle ear contraction increased labyrinthine pressure "but not to the extent that sound transmission is significantly affected" – which again, on the basis of possible effects on the cochlea, begs the question.

In 1984, Borg and colleagues summarised the ILP theory (pp. 71–72 of [11]) and dismissed it on the basis of the same two citations, plus that of Kato in 1913. They concluded that labyrinthine pressure changes during muscle contraction have no effect on hearing.

Evidence for the ILP theory

Given the negative, although largely misdirected, arguments against the ILP theory, it is now worth looking at evidence that tends to support the theory.

(i) Change in pitch

Perhaps the most compelling piece of evidence in favour of the ILP theory is the long-standing observation that

when pressure is applied to the ear, pitch tends to rise [29,58]. Pitch is the psychophysical aspect of a particular oscillation frequency within the cochlea, and the important point is that the frequency of a sound will always remain the same as it traverses the middle ear. This contrasts with amplitude or perceived loudness, which can be affected by the middle ear and the cochlea. A shift in frequency is therefore a clear sign that some aspect of cochlear functioning has changed, and the ILP theory naturally attributes this to static pressure. In modern terms, the interpretation is that some aspect of the cochlear amplifier (that is, the outer hair cells) has changed, but even before the time of Kemp's pivotal discoveries the argument carries the same force.

Békésy's finding was that compressing the veins of the neck (which increases intracranial pressure) caused a 2% reduction in pitch (p. 738 of [58]). Similarly, Corey [59] found that clenching his jaw (which activates the middle ear muscles) caused a rise in pitch of almost a semitone in pure tones below 1 kHz. Fritze (1995) reported an upward pitch shift of around 0.6% at 1 kHz when pressure in the ear canal was either lowered or raised by 40 cm of water[3]. Rowan and colleagues found an average drop of 0.2% in the pitch of a 500 Hz tone among 9 normally hearing subjects [60]. These generally small changes are comparable to the small frequency shift of spontaneous otoacoustic emissions, which were mentioned earlier [14,35] and will be addressed later, and they clearly point to an effect of pressure on the cochlea. Based on observed and inferred shifts in SOAE frequencies and intracranial pressure, Bell calculated a coefficient of about 20 Hz/kPa. In this way, changes in SOAE frequency can be taken as a useful gauge for changes in intracochlear pressure.[4]

Strengthening such an interpretation, Figure 5b of [35] shows that SOAE frequencies are susceptible to pressure below 2 kHz and largely immune to pressure above that frequency – in the same way as the middle ear is considered to attenuate sound below and above that frequency based on cochlear microphonic measurements. Deriving a conversion factor from hertz to kilopascals could be a basis from which to interpret existing work, and it could be a useful starting point for further investigation. Care needs to be taken, however, because both pressure increases and decreases can cause an increase in frequency, depending on where on the V characteristic the starting point lies. This symmetry in frequency is similar to symmetrical changes in loudness.[5]

(ii) Stapes fixation and bone conduction

Gellé's name is associated with a clinical test used to diagnose otosclerosis [62,63]. In people with the condition the stapes is rigidly fixed in the oval window, so when pressure is applied to the ear canal it fails to cause motion of the stapes, and intracochlear pressure remains constant. People with otosclerosis give a *negative* Gellé test, meaning that if a tuning fork is applied to the skull, the bone-conducted sound – which is presumed to largely enter the cochlea and its fluids via the cochlear walls – is *not* reduced in loudness when pressure is applied to the ear canal. By contrast, normal subjects have a *positive* Gellé test, so that bone-conducted sound is reduced in loudness when pressure is applied. The effects are more complicated with air conducted sound because multiple pathways are involved, but Rasmussen [62] relates a clinical investigation which supports the ILP theory.

(iii) Increases in sensitivity

While the conventional theory of middle ear muscle action can account for losses in sound sensitivity when the muscles act, it is rather more difficult to account for why at certain bands of frequencies – typically between 2 and 4 kHz – the sensitivity to sound can increase (e.g., Figure 76 of [10]; [32]). The usual explanation is to assume that muscle contraction has created some sort of resonance, but it is then difficult to account for how improvements are seen over a considerable band of frequencies.

(iv) Problems associated with accepted theory

As early as 1962, anomalies in the standard theory had already come to light. Studies in guinea pigs [64] had shown that, when the animals' middle ear muscles contracted, cochlear microphonics in the third turn behaved differently to those in the first turn, a result that the author pointed out cannot be explained by acoustic transmission changes. Noting escape of fluid whenever the muscles contracted, he suggested that an increase in fluid pressure was responsible.

Taking a similar electrophysiological approach, in 1992 Avan and colleagues [65] did guinea pig experiments that uncovered a related set of anomalous responses. The findings prompted the authors to express a general dissatisfaction with "the most widespread hypotheses concerning the acoustic reflex". Their paper concluded that middle ear muscles seem to have several different functions and that attenuation of loud sounds might not be their primary role. Although the ILP theory was not specifically mentioned, it does make a good candidate for explaining what the authors reported.

For example, the cochlear microphonic was found to *increase* over the entire range of 2 to 5 kHz during spontaneous activation of the tensor tympani (Figure 8 of [65]), an effect highly relevant to point iii above. In another puzzling finding, the effect of muscle contraction on the cochlear microphonic was extremely small: in 10 animals, activation of the acoustic reflex resulted in an average reduction in the cochlear microphonic by only 1 dB, and in 35 other animals no significant effect could be detected. In 30

3. In this study, the state of the Eustachian tube (open or closed) was not ascertained, and this is a major factor in determining whether ear canal pressure is conveyed to the labyrinth [46].

4. This coefficient also probably relates to a phase shift in click-evoked OAEs under intracranial pressure changes [61]. The shift was found to be about 20°/kPa.

5. See point 1 on page 13. Note also that displacement of the tip from zero is a measure of intracochlear pressure (provided middle ear pressure is zero, which is usually the case if the Eustachian tube is opened by swallowing).

guinea pigs, there was no appreciable impedance change during stimulation. Increasing the puzzle, the authors performed the same experiments on rabbits and here the reductions in cochlear microphonics were large.

What lessons can be learnt from these disparate findings? At the very least it can be concluded that there are clear differences between species, perhaps in regard to intracranial pressures and cochlear aqueduct patency, and that understanding of the middle ear muscles is far from complete. It would seem that the accepted theory – attenuation via stretching of the annular ligament – is not satisfactory and closer examination of intracochlear pressure is warranted.

The modern view of an active cochlea

The ILP theory has now disappeared from consideration, but there is one new area of endeavour in which the theory might reestablish a foothold, and that is in the area of otoacoustic emissions.

First, there were the studies of Kemp [35] and Wilson and Sutton [66], already alluded to, which found that increasing (or decreasing) air pressure in the ear canal caused the frequency of spontaneous otoacoustic emissions to systematically rise (see also [67,68]). Kemp attributed the effect to impedance changes at the oval window – stretching of the annular ligament – which altered the phase with which reverse traveling waves were reflected. This is still the standard view, although it has been questioned [69]. An alternative view is that the inward motion of the stapes increases the pressure within the labyrinth, and this pressure affects the outer hair cells and the timing of the positive feedback loop between them [70]. The conjecture is that this loop, operating between the three rows of outer hair cells, is the basis of the cochlear amplifier.[6]

These ideas can be placed within a common framework using insights from a study of the natural frequency variations of spontaneous emissions [37]. This work found that the frequency changes, although small (a fraction of a percent), varied in step with normal diurnal and menstrual cycles. The common denominator for these variations was intracranial pressure, which was presumed to flow through to intracochlear pressure. Instead of supposing that pressure causes stretch of the annular ligament, an alternative explanation is that pressure directly squeezes the outer hair cells, which are in continuous hydraulic connection with the cochlear fluids via the spaces of Nuel. This idea leads on to another: if the outer hair cells could be affected by static (d.c.) pressure, might it be the case that they are also sensitive to oscillating (a.c.) pressure in the cochlear fluids – in other words, sound? A further evolution of the idea is to consider pressure due to the inwards pull of the middle ear muscles, and at this point the ILP theory begins to reemerge.

Significant support for the ILP theory comes from Burns and colleagues and their work on the effect of voluntary contraction of middle ear muscles on spontaneous otoacoustic emissions [14]. When their gifted subject contracted her middle ear muscles, there were small changes in impedance, large but inconsistent changes in SOAE amplitude, and most interestingly, graded shifts in frequency (up to 2%), nearly always upwards. These shifts in frequency fell back towards baseline over some tens of seconds, whereas contractions elicited by contralateral noise saw the shifts maintained. These results are generally consistent with SOAE frequency shifts being substitute measures for intracochlear pressure changes. Before some pertinent literature is examined, it is notable that the changes in magnitude of some of the SOAEs were as much as 25 dB or so, a figure comparable to some of the apparent "transmission changes" seen in hearing threshold shifts and cochlear microphonic changes under the influence of middle ear muscles. To reiterate, inner ear pressure could be the effective controlling variable.

The acoustic reflex has both ipsilateral and contralateral components, so if the ILP theory is true then one expects to see that contralateral acoustic stimulation will lead to pressure effects on the ipsilateral cochlea. It is therefore not surprising that Veuillet and colleagues [67] found considerable similarity, although some differences, between the effect of ear canal pressure and contralateral acoustic stimulation (that is, middle ear contraction) on the magnitude of click-evoked otoacoustic emissions. About 4 cm of water (positive or negative) had nearly the same effect as contralateral stimulation right across the frequency band, and as might be expected, the average effect of positive and negative pressures was even closer. Similarly, Avan and colleagues [71] found a striking similarity between the effect of increased intracranial pressure and the effect of stapedius muscle contraction.

For some time, investigators of otoacoustic emissions have been trying to isolate the effect of middle ear muscle contraction (the MEM reflex) from that of direct efferent action on the outer hair cells via the medial olivocochlear pathway, the MOC reflex [72–76]. Because both reflexes share nerve pathways, disentangling their effects is not easily done, although Guinan and colleagues [74] put forward evidence that the issue can be settled by measuring phase delays of SFOAEs: long group delays of 10 ms point to MOC effects, whereas a figure of less than 1 ms signifies an MEM effect.[7] However, it seems wise to exercise caution in this area because the effects are complex and subtle, and middle ear muscles do not exhibit a clear-cut threshold, as usually thought, but gradually increase their tension as sound levels rise [14]. Certainly, failure to detect reflex effects using impedance probes in the ear canal does not rule out that the muscles have been subtly increasing tension and changing fluid pressure in the cochlea. The putative effects of efferent stimulation on DPOAE fine structure could actually be due to pressure effects from middle ear muscles, even at levels as low as 55 dB SPL [74,75] or perhaps less. Various mechanisms for MOC effects have been suggested [76], and only further research will show whether shifts in SOAE frequency provide an accurate indicator of changes in intracochlear pressure.

6. It follows that if the loop gain at any point along the partition exceeds unity then the result will be a spontaneous otoacoustic emission.

7. Tuning – dependence of effects in one ear on the frequency of stimulation in the other – would also point to MOC rather than MEM effects, as the latter is not tuned.

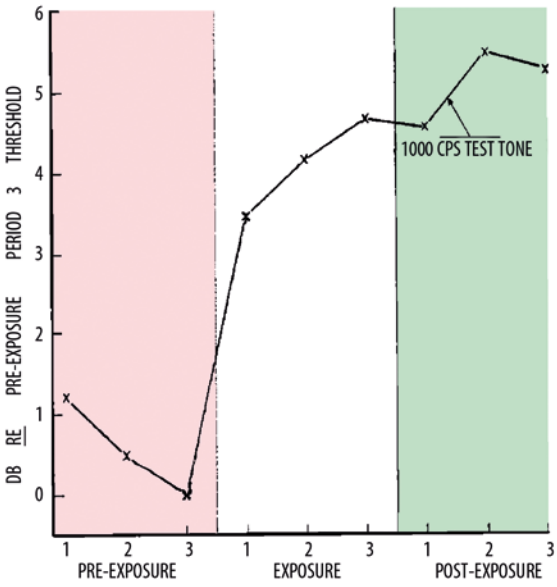

Figure 4. Long-lasting, but apparently small, effects of middle ear muscles. In this study of 12 subjects, acoustic thresholds to a 1 kHz tone were measured in the right ear before, during, and after exposure to a 2 kHz tone at 100 dB SPL in the left ear. Note that the thresholds, measured at 10-second intervals, continue to increase for some 30 seconds after the activating tone has ceased (green). Moreover, because this test was interleaved with another similar test, thresholds were still recovering in the 30 second period *before* exposure began (pink). The long time constant makes the measured threshold shifts smaller than they actually are. Adapted from [52] and used with permission of the Acoustical Society of America.

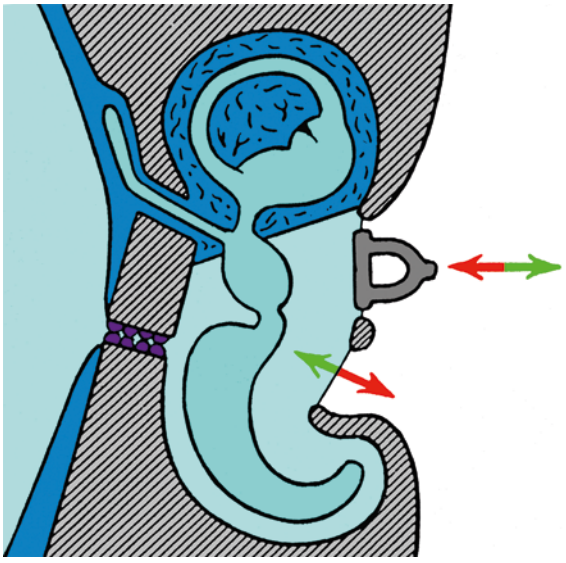

Figure 5. The cochlea is virtually a hydraulically sealed system, as this diagram from Kirikae (after de Burlet) illustrates. When the stapes moves, the round window moves in the opposite direction an equal amount (red/green arrows). The cochlear aqueduct (purple) is a narrow channel filled with fibrous tissue, meaning that pressure can only dissipate through it very slowly. The aqueduct connects perilymph (light blue) with cerebrospinal fluid in the cranium (left). Endolymph is shown in mid blue. Pressure in the cochlea therefore depends strongly on the state of tension in the tensor tympani, which pushes the stapes into the oval window. Evidence suggests that the muscle, once activated, remains in a state of tonic contraction for many minutes. Figure from [99], and used with permission.

A long time constant

As emphasised by Borg in point 3 above, the long time constant of protective action of the middle ear muscles argues against the ILP theory – if the pressure created readily dissipates through the cochlear aqueduct or similar channel. Some evidence countering the force of this point has already been covered, such as that shown in Figure 4, but in this section additional evidence relating to otoacoustic emissions will be set out. The working hypothesis is that shifts in the frequencies of otoacoustic emissions are reasonable gauges of changes in intracochlear pressure. Can this idea be sustained?

Consideration of Figure 5 suggests that the patency of the cochlear aqueduct is a major factor in controlling the time constant. The fibres of the tensor tympani muscle are of the fatigue-resistant type [48], so that once the muscle is activated, the muscle might be expected to sustain its force for a considerable time, limited perhaps by the leakage of fluid through the narrow cochlear aqueduct. The time for which the tensor tympani can exert a force on the incompressible fluids without fatiguing is difficult to judge, as evidence is scant. Some recent clinical work [77] found that responses of the tensor tympani could sometimes habituate after a single stimulation (their Figure 4), suggesting that the muscle's function is to drive up cochlear pressure; the elevated pressure appears to be taking many seconds to dissipate. By way of contrast, the time constant in the guinea pig has been found to be only a few seconds [51]. The complication then is that the patency varies between species and even among individuals. The cochlear aqueduct is not a narrow hard-walled tube with a fixed flow resistance, but is filled by a loose network of connective tissue with a high resistance to flow, as the partial blocking in Figure 5 is meant to convey. Patency is clearly an important issue, and aspects are discussed in [36,37,39,49].

The evidence from otoacoustic emissions is that the time constant can vary from several seconds to several minutes or more. de Kleine and colleagues [78] found that it took about a minute for spontaneous emission frequency to return to normal after a subject laid down (increasing intracochlear pressure) but less than 10 seconds for the reverse manoeuvre. Recently, Voss and colleagues [55] measured otoacoustic emissions from first the left and then the right ears of 12 subjects and found that data from the right ears

had to be discarded because the emissions had not returned to baseline values after the subjects had been tilted to –45° and back, even though more than 5 minutes had elapsed from the initial tilt. This behaviour is similar to that of Büki and colleagues [43] who also found that 5 minutes was not always enough to reestablish pre-tilt conditions. At this point it is worth saying that the latter authors summarise their study by saying that posture changes – which increase intracochlear pressure – have effects closely resembling those induced by stapedius contraction.

These findings support the idea that the cochlear aqueduct appears to function as a one-way valve: fluid can easily flow into the cochlea, but it takes longer for it to leave. A similar effect (and hence one difficult to separate) could derive from tonic contraction of the tensor tympani; the muscle might continue to press on the stapes for extended periods and, like a ratchet, permit motion in one direction but not the other. Relevant here is the observation that the muscle contains proprioceptors [4,48]. Early work on SOAEs showed that frequency changes occurred over a 10–30-minute time frame [38,54] and this is discussed in the next section. Given these long time constants, the 20 minutes required for pressure effects to decay, observed by Békésy, may well have been due to limited patency of the cochlear aqueduct.

Focus on the tensor tympani

This paper has generally treated the middle ear muscles as if they were a common unit, but anatomically and functionally this is not the case. Work is needed to distinguish the effect of the tensor tympani, which seems to be well placed to control intracochlear pressure, from that of the stapedius, whose function is probably more of a circuit breaker, turning a piston movement of the stapes into a rocking motion [8,45].

Compared to the stapedius, the tensor tympani has been little studied, and it has even been proposed that the muscle performs no useful function in mammals [79]. More likely, the situation is that the muscle's effects are fairly subtle under standard tympanometry and hard to separate from those of the stapedius [77,80]. It would seem that a low to moderate stimulus causes this muscle to slowly and steadily increase its resting tension [81,82], which gradually dissipates [83]; the sudden jerks in response to high sound levels, detected by tympanometry, are more like emergency responses. It is possible that the tensor works so quietly and effortlessly that its actions, which must be very close to isometric, are hard to detect by tympanometry. If such a description is accurate, jumps in impedance measurements may seem to show adaptation or habituation [24,27,77,83,84], becoming apparently weaker over time. But, recalling point 2 above, if the tensor tympani's function is to steadily build up pressure, protection may actually continue in the cochlea even though detectable muscular effort – seen most readily in stapedius responses – may look like it is dwindling. In experimental language, temporal integration effects, supplied by the tensor, may be countering adaptation effects, experienced by the stapedius [83,84]. Clearly, the middle ear is part of a tightly regulated[8] servo system [5,83].

It is known that the tensor tympani is most sensitive to ipsilateral stimulation (the uncrossed reflex), with contralateral (crossed) effects coming into play at higher sound levels [5,77,80,86]. This particular feature helps explain why measurements of contralateral reflex effects sometimes return relatively small values of protection [52,87] because the ipsilateral effects are meeting most of the attenuation requirements. Another confounding factor is that the tensor tympani is innervated by the 5th cranial nerve, whereas the stapedius is controlled by the 7th facial nerve; understandably, then, it is possible for patients with Bell's palsy[9], which affects only the 7th [77], to still have some acoustic reflex protection despite what some experimenters [87] have erroneously supposed.

A noteworthy property of the tensor tympani is that it can be activated by touch, vocalisation, swallowing, facial gesture, and startle, as well as sound [80]. It will therefore be activated by putting on headphones or placing a probe microphone in the ear canal, with puzzling consequences if these stimuli are not recognised. Thus, there is the long-standing conundrum that low-frequency sounds (about 500 Hz) appear to be some 10 dB softer when heard through earphones – which touch the external ear and ear canal – than they do when heard over loudspeakers [88]. The "missing 10 dB" is contentious, but descriptions of the procedures used in these conflicting experiments reveal that the common denominator in causing loss of threshold is activation of the tensor tympani by touch. Unwittingly, those who contend there is no gap between the two conditions used a probe tube inside the ear canal during their free-field testing, whereas those who measured a gap avoided this reflex-activating factor. This unrecognised problem continues to have implications today for any threshold experiment involving headphones and probe microphones, and the same touch-elicitation problem will affect tympanometry as well (as alluded to on p. 117 of [24]). Similarly, use of headphones will also tend to confound experiments aimed at gauging the strength of sound-evoked reflexes [52], helping to explain why the observed effects (e.g. Figure 4) are seemingly so small.

The headphone factor directly relates to the finding that when a probe microphone is inserted into the ear canal, SOAEs tend to drift downwards in frequency for 10–30 minutes after insertion [38,54]. But if the probe microphone is repeatedly inserted and withdrawn, the frequency goes up [54]; likewise, placing the probe in the contralateral ear has the same effect as keeping it in the ipsilateral ear [89]. These results can be understood in terms of a bilateral tensor reflex and its effect on intracochlear pressure: sitting in a quiet chamber for an extended period allows the tensor tympani to gradually relax and the

8. It is remarkable that the reflex threshold for tones is only a few dB less in subjects with appreciable hearing losses (of up to 50 dB) compared to normal subjects [85].

9. When Brask [80] used extratympanic manometry to test patients with Bell's palsy, he observed a tensor reflex which could also create a sensation of dizziness, due he thought to pressure in the labyrinth.

hearing threshold to adapt; but stimulating the touch receptors in the ear canal (in either ear) activates the tensor tympani and causes pressure to rise. On the basis of a shift of 10 Hz, it can be estimated, using the earlier figure of 20 Hz/kPa, that the pressure changes were about 0.5 kPa and, on the basis of Table II of [88], caused a threshold shift[10] of 2–3 dB.

Experimentally, some sort of non-contact microphone might be particularly informative in these situations.

A related issue is the Tullio phenomenon, a curious clinical condition in which a sound, or even a touch to the ear or face, induces vertigo or nystagmus [91]. The ILP theory readily explains why activation of the tensor tympani – such as from humming or playing a violin – might cause an abnormal pressure increase in the labyrinth. Of relevance, the condition is rare in cases of otosclerosis.

Pressure and Ménière's disease

There is a connection between Ménière's disease and intracochlear pressure, which has long been suspected but been difficult to prove [92]. Of course, if middle ear muscles can readily affect the hydraulic pressure in the cochlea, it follows that there could be a relationship between faulty activity of these muscles and the disease. The ILP theory brings to the fore such findings as that general tension and aural fullness frequently precede a Ménière's attack. Perhaps inappropriate activity of the middle ear muscles, not hydrops-induced excess pressure, might be the trigger [93]. With the ILP model, it is possible to see how hydrops and excess muscle activity could both lead to the same symptom – an increase in pressure and low frequency hearing loss.

An excess of endolymph (hydrops) is naturally expected to lead to higher intracochlear pressure but, as noted previously, pressures in the cochlea are hard to measure and the association of Ménière's attacks with elevated pressures, and indeed with hydrops itself, remains indirect. Nevertheless, the ILP theory strengthens the relationship to pressure and gives a functional context to the fullness and pressure in the ears. It helps explain the low-frequency hearing loss, why sectioning of the middle ear muscle tendons is often an effective remedy for the condition [93], and why tones in the affected ear have a higher pitch than in the unaffected one [94]. Shifts in OAE phase, similar to those induced by body tilt, are also more common in Ménière's sufferers [95]; such rapid changes – over a matter of seconds – could be caused by failure of the middle ear muscles to exert steady force.

Another related condition, and one that underlines the important role of pressure in hearing, is one where deafness comes on abruptly; in an appreciable number of such cases the precipitating factor is a sudden change in cerebrospinal fluid pressure, such as from a Valsalva manoeuvre [96,97].

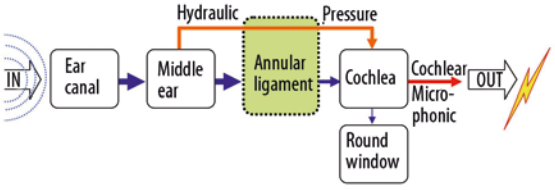

Figure 6. Outline of the essential difference between the ILP theory and the conventional "sound attenuation" theory of the middle ear muscles. Sound transmission is shown in blue, hydraulic pressure in orange, and cochlear microphonic in red. Conventional understanding is that middle ear muscles stretch the annular ligament (green box), changing its input impedance and reducing sound input to the cochlea. Cochlear microphonics are used to measure this impedance change. However, on the ILP theory, the annular ligament is acoustically transparent, independent of pressure (as Békésy found), and the main factor controlling cochlear microphonics is hydraulic pressure (orange) mediated by the middle ear muscles.

Conclusions

This paper has put together a case in favour of the intralabyrinthine pressure theory, and diverse evidence from the literature indicates that fresh consideration of the theory is warranted. In the theory, middle ear muscle contraction causes pressure in the labyrinth to increase, and it is this factor, not annular ligament stretch and reduced sound transmission, which is the main cause of reduced cochlear responses (Figure 6). In the ILP theory, the pressure reduces the gain of the cochlear amplifier via a direct physiological effect on outer hair cells.

A necessary next step is to experimentally separate the two variables that have so far been treated as aspects of just one – sound transmission and cochlear response. Isolating them is not easy because, as Figure 6 illustrates, the annular ligament is intimately connected to ILP and its impedance is in series with that of the cochlea and round window. Moreover, it is not just the annular ligament that is in the mechanical chain: the eardrum and articulating joints also have effects.[11] The suggestion has been made that frequency shifts in spontaneous otoacoustic emissions might serve as an effective gauge of intracochlear pressure (a figure of 20 Hz/kPa is a reasonable starting point),

10. When endeavouring to relate a change in frequency change to a change in pressure, it is important to distinguish between pressure in the cochlea and pressure in the ear canal, which are interrelated. Forces acting on the eardrum are transmitted via the transformer action of the ossicles to the stapes, and vice versa. So Kemp's finding of 10–50 Hz/kPa [35], and Robinson and Haughton's of 150 Hz/kPa [90], are in terms of pressure in the ear canal. The figure of 20 Hz/kPa was calculated on the basis of pressure in the cochlear fluids.

11. The same difficulty arises in establishing the locus of a related multifactorial effect. In extratympanic manometry, deviations of the impedance minimum from zero pressure are often interpreted as indicating "middle ear pressure" whereas more accurately it may be the result of intracochlear pressure [98]. On the basis of a displaced peak in the tympanogram, a common diagnosis of a hearing deficit is a blocked Eustachean tube; however, as the foregoing reference discloses, many such patients have, paradoxically, an open tube. Their hearing problem may therefore relate more to elevated intracochlear pressure.

and this idea also calls for investigation. Interestingly, the sensitivity of SOAEs to pressure increases steeply in the same frequency range – below 2 kHz – where the acoustic reflex comes into play (Figure 5b of [35]), another indication that apparent attenuations through the middle ear might have more to do with the cochlea than the annular ligament.

Patency of the cochlear aqueduct is a major factor in controlling the way ILP affects the cochlea, and present indications are that it is highly variable. Clearly more work, anatomical and audiological, is needed to resolve this issue.

Historically, the ILP theory has never gained wide acceptance. It was rejected last century because of its incompatibility with the passive mechanics of the traveling wave theory. The traveling wave theory has now been given active aspects, and there are other proposals for cochlear responses to the fast pressure wave, but these alternatives to standard theory remain speculative.

This focused review has shown how clinical observations have been especially useful in understanding what the middle ear muscles are doing. Work from the 19th century is still valuable, and Gellé's insights remain a landmark. Studies on subjects with voluntary control of their middle ear muscles have been particularly rewarding.[12]

The aim of this review has been to synthesise key elements of a wide-ranging literature on the function of the middle ear muscles, a system whose finesse has generally not been appreciated. It is hoped that the ideas put forward will prompt further investigation and lead to a better understanding of the auditory system and its refinements. The delicate interplay between the cochlea and the middle ear muscles deserves renewed attention.

Acknowledgements

Collaboration and some funding from the Institute of Physiology and Pathology of Hearing, Poland, assisted in the writing of this paper.

References:

1. Fletcher NH: Acoustic Systems in Biology. New York: Oxford University Press; 1992
2. Rosowski JJ: External and middle ear function. In: Fuchs PA (ed.), The Oxford Handbook of Auditory Science. Oxford: Oxford University Press, 2010; 49–91
3. Kemp DT: Otoacoustic emissions: concepts and origins. In: Manley GA, Fay RR, Popper AN (eds.), Active Processes and Otoacoustic Emissions. New York: Springer, 2008; 1–38
4. Jepsen O: Middle-ear muscle reflexes in man. In: Jerger J (ed.), Modern Developments in Audiology. New York: Academic Press, 1963; 193–239
5. Borg E: Acoustic middle ear reflexes: a sensory-control system. Acta Oto-Laryngol, 1972; Suppl. 304: 1–34
6. Møller AR: Function of the middle ear. In: Keidel WD, Neff WD (eds.), Handbook of Sensory Physiology. Berlin: Springer-Verlag, 1974; 491–548
7. Møller AR: The acoustic middle ear muscle reflex. In: Keidel WD, Neff WD (eds.), Handbook of Sensory Physiology. Berlin: Springer-Verlag, 1974; 519–48
8. Békésy Gv: Experiments in Hearing. Wever EG (ed.), New York: McGraw-Hill, 1960
9. Zemlin WR: Speech and Hearing Science. Englewood Cliffs, N.J.: Prentice-Hall, 1968
10. Wever EG, Lawrence M: Physiological Acoustics. Princeton: Princeton University Press, 1954
11. Borg E, Counter SA, Rösler G: Theories of middle-ear muscle function. In: Silman S (ed.), The Acoustic Reflex: Basic Principles and Clinical Applications. New York: Academic Press, 1984; 63–99
12. Møller AR: An experimental study of the acoustic impedance of the middle ear and its transmission properties. Acta Otolaryngol, 1965; 60: 129–49
13. Nuttall AL: Tympanic muscle effects on middle-ear transfer characteristic. J Acoust Soc Am, 1974; 56: 1239–47
14. Burns EM, Harrison WA, Bulen JC, Keefe DH: Voluntary contraction of middle ear muscles: effects on input impedance, energy reflectance and spontaneous otoacoustic emissions. Hear Res, 1993; 67: 117–27
15. Zwislocki JJ: Auditory Sound Transmission: An Autobiographical Perspective. Mahwah, NJ: Erlbaum, 2002
16. Gellé M: Les lésions nerveuses dans la surdité. Transactions, International Congress of Medicine, 1881; 3: 370–72
17. Gellé M: Valeur de l'épreuve des pressions centripètes [Consideration of the test of centripetal pressure]. Annales des Maladies de l'Oreille, du Larynx, du Nez et du Pharynx, 1885; 11: 63–70 (in French)
18. Khanna SM, Tonndorf J: The vibratory pattern of the round window in cats. J Acoust Soc Am, 1971; 50: 1475–83
19. Ashmore JF, Avan P, Brownell WE et al: The remarkable cochlear amplifier. Hear Res, 2010; 266: 1–17
20. Bell A: Are outer hair cells pressure sensors? Basis of a SAW model of the cochlear amplifier. In: Gummer AW (ed.), Biophysics of the Cochlea: From Molecules to Models. Singapore: World Scientific, 2003; 429–31
21. Ren T, Nuttall AL: Cochlear compression wave: an implication of the Allen-Fahey experiment (L). J Acoust Soc Am, 2006; 119: 1940–42
22. Andersen HC, Hansen CC, Neergaard EB: Experimental studies on sound transmission in the human ear. Acta Otolaryngol, 1962; 54: 511–20
23. Neergaard EB, Andersen HC, Hansen CC, Jepsen O: Experimental studies on sound transmission in the human ear. III. Influence of the stapedius and tensor tympani muscles. Acta Oto-Laryngol, 1963; Suppl. 188: 280–86
24. Metz O: The acoustic impedance measured on normal and pathological ears. Acta Otolaryngol, 1946; Suppl. 63: 1–254
25. Lynch TJ, Nedzelnitsky V, Peake WT: Input impedance of the cochlea in cat. J Acoust Soc Am, 1982; 72: 108–30

12. At the same time the literature describes animal-based experiments that have been seriously misleading. The view of this author is that work using consenting subjects offers ethical and scientific advantages. Discussion of these issues is the focus of the Minding Animals conferences. A broad context is offered by Bekoff at *http://www.mindinganimals.com/index.php?option=com_content&task=view&id=69&Itemid=91*.

26. Tonndorf J, Khanna SM: Some properties of sound transmission in the middle and outer ears of cats. J Acoust Soc Am, 1966; 41: 513–21
27. Klockhoff I, Anggard G, Anggard L: Recording of cranio-labyrinthine pressure transmission in man by acoustic impedance method. Acta Otolaryngol, 1966; 61: 361–70
28. Lutman ME, Martin AM: Development of an electroacoustic analogue model of the middle ear and acoustic reflex. J Sound Vibration, 1979; 64: 133–57
29. Smith HD: Audiometric effects of voluntary contraction of the tensor tympani muscles. Arch Otolaryngol, 1943; 38: 369–72
30. Reger SN: Effect of middle ear muscle action on certain psychophysical measurements. Ann Otol Rhinol Laryngol, 1960; 69: 1179–98
31. Nuttall AL: Measurements of the guinea-pig middle-ear transfer characteristic. J Acoust Soc Am, 1974; 56: 1231–47
32. Rasmussen H: Studies on the effect upon the hearing through air conduction brought about by variations of the pressure in the auditory meatus. Acta Otolaryngol, 1946; 34: 415–24
33. Pang XD, Peake WT: How do contractions of the stapedius muscle alter the acoustic properties of the ear? In: Allen JB, Hall JL, Hubbard AE et al. (eds), Peripheral Auditory Mechanisms. New York: Springer, 1986; 36–43
34. Huizing EH: Bone conduction: the influence of the middle ear. Acta Oto-Laryngol, 1960; Suppl. 155: 1–99
35. Kemp DT: The evoked cochlear mechanical response and the auditory microstructure: evidence for a new element in cochlear mechanics. Scand Audiol, 1979; Suppl. 9: 35–47
36. Włodyka J: Studies on cochlear aqueduct patency. Ann Otology, 1978; 87: 22–28
37. Bell A: Circadian and menstrual rhythms of spontaneous otoacoustic emissions from human ears. Hear Res, 1992; 58: 91–100
38. Whitehead ML: Slow variations of the amplitude and frequency of spontaneous otoacoustic emissions. Hear Res, 1991; 53: 269–80
39. Marchbanks RJ, Reid A: Cochlear and cerebrospinal fluid pressure: their inter-relationship and control mechanisms. Br J Audiol, 1990; 24: 179–87
40. Saxena RK, Tandon PN, Sinha A, Kacker SK: Auditory functions in raised intracranial pressure. Acta Otolaryngol, 1969; 68: 402–10
41. John PY, Kacker SY, Tandon PN: Bekesy audiometry in evaluation of hearing in cases of raised intracranial pressure. Acta Otolaryngol, 1979; 87: 441–44
42. Macrae JH: Effects of body position on the auditory system. J Speech Hear Res, 1972; 15: 330–39
43. Büki B, Chomicki A, Dordain M et al: Middle-ear influence on otoacoustic emissions. II: contributions of posture and intracranial pressure. Hear Res, 2000; 140: 202–11
44. de Kleine E, Wit HP: The behavior of spontaneous otoacoustic emissions during and after postural changes. J Acoust Soc Am, 2000; 107: 3308–16
45. Hüttenbrink KB: The mechanics of the middle-ear at static air pressures. Acta Oto-Laryngol, 1988; Suppl. 451: 1–35
46. Densert O, Ivarsson A, Pedersen K: The influence of perilymphatic pressure on the displacement of the tympanic membrane. Acta Otolaryngol, 1977; 84: 220–26
47. Ivarsson A, Pedersen K: Volume-pressure properties of round and oval windows. Acta Otolaryngol, 1977; 84: 38–43
48. van den Berge H, Wirtz P: Detailed morphology of the tensor tympani muscle of the rat. J Anat, 1989; 164: 215–28
49. Gopen Q, Rosowski JJ, Merchant SN: Anatomy of the normal human cochlear aqueduct with functional implications. Hear Res, 1997; 107: 9–22
50. Waltner JG: Barrier membrane of the cochlear aqueduct. Arch Otolaryngol, 1948; 47: 656–69
51. Wit HP, Thalen EO, Albers FMJ: Dynamics of inner ear pressure release, measured with a double-barreled micropipette in the guinea pig. Hear Res, 1999; 132: 131–39
52. Loeb M, Riopelle AJ: Influence of loud contralateral stimulation on the threshold and perceived loudness of low-frequency tones. J Acoust Soc Am, 1960; 32: 602–10
53. Rosowski JJ, Ravicz ME, Songer JE: Structures that contribute to middle-ear admittance in chinchilla. J Comp Physiol A, 2006; 192: 1287–311
54. Zurek PM: Spontaneous narrowband acoustic signals emitted by human ears. J Acoust Soc Am, 1981; 69: 514–23
55. Voss SE, Adegoke MF, Horton NJ et al: Posture systematically alters ear-canal reflectance and DPOAE properties. Hear Res, 2010; 263: 43–51
56. Lempert J, Wever EG, Lawrence M, Meltzer PE: Perilymph: its relation to the improvement of hearing which follows fenestration of the vestibular labyrinth in clinical otosclerosis. Arch Otolaryngol, 1949; 50: 377–87
57. McCabe BF, Wolsk D: Experimental inner ear pressure changes. Ann Otol Rhinol Laryngol, 1961; 70: 541–55
58. Bekesy Gv: Zur Theorie des Hörens. Physik Zeit, 1929; 30: 721–45 (in German)
59. Corey VB: Change in pitch of sustained musical note by clenching of the jaws of the observer. J Acoust Soc Am, 1950; 22: 640
60. Rowan D, Kapadia S, Lutman ME: Does static ear canal pressure influence pure tone pitch perception? Acta Otolaryngol, 2004; 124: 286–89
61. Büki B, Avan P, Lemaire JJ et al: Otoacoustic emissions: a new tool for monitoring intracranial pressure changes through stapes displacements. Hear Res, 1996; 94: 125–39
62. Rasmussen H: Studies on the effect on the air conduction and bone conduction from changes in the meatal pressure in normal subjects and otosclerotic patients. Acta Oto-Laryngol, 1948; Suppl. 74: 54–64
63. Dankbaar WA: The diagnotistic value of Gellé's test. Acta Otolaryngol, 1970; 69: 266–72
64. Legouix J-P: Changes in the cochlear microphonic of the guinea pig produced by mechanical factors in the inner ear. J Acoust Soc Am, 1962; 34: 1504–8
65. Avan P, Loth D, Menguy C, Teyssou M: Hypothetical roles of middle ear muscles in the guinea-pig. Hear Res, 1992; 59: 59–69
66. Wilson JP, Sutton GJ: Acoustic correlates of tonal tinnitus. In: Evered D, Lawrenson G (eds.), Tinnitus. London: Pitman, 1981; 82–107
67. Veuillet E, Collet L, Morgon A: Differential effects of ear-canal pressure and contralateral acoustic stimulation on evoked otoacoustic emissions in humans. Hear Res, 1992; 61: 47–55
68. van Dijk P: The effect of static ear canal pressure on human spontaneous otoacoustic emissions: spectral width as a measure of the intra-cochlear oscillation amplitude. J Assoc Res Otolaryngol, 2011; 12: 13–28
69. Nobili R, Vetesnik A, Turicchia L, Mammano F: Otoacoustic emissions from residual oscillations of the cochlear basilar membrane in a human ear model. J Assoc Res Otolaryngol, 2003; 4: 478–94
70. Bell JA: The Underwater Piano: A Resonance Theory of Cochlear Mechanics. PhD thesis, Australian National University, Canberra, 2005. *http://hdl.handle.net/1885/49307*

71. Avan P, Büki B, Maat B et al: Middle ear influence on otoacoustic emissions. I. Noninvasive investigation of the human transmission apparatus and comparison with model results. Hear Res, 2000; 140: 189–201
72. Deeter R, Abel R, Calandruccio L, Dhar S: Contralateral acoustic stimulation alters the magnitude and phase of distortion product otoacoustic emissions. J Acoust Soc Am, 2009; 126: 2413–24
73. Rabinowitz WM, Widin GP: Interaction of spontaneous otoacoustic emissions and external sounds. J Acoust Soc Am, 1984; 76: 1713–20
74. Guinan JJ, Backus BC, Lilaonitkul W, Aharonson V: Medial olivocochlear efferent reflex in humans: otoacoustic emission (OAE) measurement issues and the advantages of stimulus frequency OAEs. J Assoc Res Otolaryngol, 2003; 4: 521–40
75. Goodman SS, Keefe DH: Simultaneous measurement of noise-activated middle-ear muscle reflex and stimulus frequency otoacoustic emissions. J Assoc Res Otolaryngol, 2006; 7: 125–39
76. Zhao W, Dhar S: Fast and slow effects of medial olivocochlear efferent activity in humans. PLoS One, 2011; 6: e18725
77. Jones SEM, Mason MJ, Sunkaraneni VS, Baguley DM: The effect of auditory stimulation on the tensor tympani in patients following stapedectomy. Acta Otolaryngol, 2008; 128: 250–54
78. de Kleine E, Wit HP, Avan P, van Dijk P: The behavior of evoked otoacoustic emissions during and after postural changes. J Acoust Soc Am, 2001; 110: 973–80
79. Howell P: Are two muscles needed for the normal functioning of the mammalian middle ear? Acta Otolaryngol, 1984; 98: 204–7
80. Brask T: Extratympanic manometry in man. Scand Audiol, 1978; Suppl 7: 1–199
81. Simmons FB: Middle ear muscle activity at moderate sound levels. Ann Otol Rhinol Laryngol, 1959; 68: 1126–43
82. Sesterhenn G, Breuninger H: The acoustic reflex at low sensation levels. Audiology, 1976; 15: 523–33
83. Møller AR: Neurophysiological basis of the acoustic middle-ear reflex. In: Silman S (ed.), The Acoustic Reflex. New York: Academic, 1984; 1–34
84. Dallos PJ: Dynamics of the acoustic reflex: phenomenological aspects. J Acoust Soc Am, 1964; 36: 2175–83
85. Silman S, Popelka GR, Gelfand SA: Effect of sensorineural hearing loss on acoustic stapedius reflex growth functions. J Acoust Soc Am, 1978; 64: 1406–11
86. Loth D, Avan P, Menguy C, Teyssou M: Crossed acoustic middle-ear muscle reflex and attenuation of low-level pure tones in man. Acta Acustica, 1994; 2: 149–55
87. Ward WD: Further observations on contralateral remote masking and related phenomena. J Acoust Soc Am, 1967; 42: 593–600
88. Keidser G, Katsch R, Dillon H, Grant F: Relative loudness perception of low and high frequency sounds in the open and occluded ear. J Acoust Soc Am, 2000; 107: 3351–57
89. Whitehead ML, Baker RJ, Wilson JP: Drifts in the frequency and amplitude of spontaneous otoacoustic emissions (SOAEs) during recording: an efferent effect? Br J Audiol, 1989; 23: 149–50
90. Robinson PM, Haughton PM: Modification of evoked otoacoustic emissions by changes in pressure in the external ear. Br J Audiol, 1991; 25: 131–33
91. Fenton RS, Smith OD: Self-induced vertigo. J Otolaryngol, 1990; 19: 264–66
92. Horner KC: Functional changes associated with experimentally induced endolymphatic hydrops. Hear Res, 1993; 68: 1–18
93. Franz P, Hamzavi JS, Schneider B, Ehrenberger K: Do middle ear muscles trigger attacks of Ménière's disease? Acta Otolaryngol, 2003; 123: 133–37
94. Tonndorf J: The mechanism of hearing loss in early cases of endolymphatic hydrops. Ann Otol Rhinol Laryngol, 1957; 66: 766–84
95. Avan P, Giraudet F, Chauveau B, Gilain L, Mom T: Unstable distortion-product otoacoustic emission phase in Menière's disease. Hear Res, 2011; 277: 88–95
96. Hallberg OE: Sudden deafness of obscure origin. The Laryngoscope, 1956; 66: 1237–67
97. Simmons FB: Theory of membrane breaks in sudden hearing loss. Arch Otolaryngol, 1968; 88: 41–48
98. Virtanen H: Middle-ear pressure and eustachian tube function. Arch Otolaryngol, 1982; 108: 766–68
99. Kirikae I: An experimental study on the fundamental mechanism of bone conduction. Acta Otolaryngol, 1959; Suppl. 145: 1–111