ISSN: 2083-389X,
eISSN: 2084-3127

J Hear Sci, 2021; 11(2): 25–34
DOI: 10.17430/JHS.2021.11.1.3

THE CURIOUS 'TYPE C' TYMPANOGRAM: CONTRACTION OF THE TENSOR TYMPANI MASQUERADES AS NEGATIVE MIDDLE EAR PRESSURE

Contributions:
A Study design/planning
B Data collection/entry
C Data analysis/statistics
D Data interpretation
E Preparation of manuscript
F Literature analysis/search
G Funds collection

Andrew Bell[A,E-F]

Eccles Institute of Neuroscience, John Curtin School of Medical Research, Australian National University, Australia

Corresponding author: Andrew Bell; Eccles Institute of Neuroscience, John Curtin School of Medical Research, Australian National University, 131 Garran Road, Canberra, Australia; email: andrew.bell@anu.edu.au

Abstract

Negative middle ear pressure presents something of a paradox. The 'Type C' tympanogram, in which the peak of the tympanogram occurs below zero pressure, seems to indicate that the air pressure in the middle ear is actually below atmospheric pressure – that there is a degree of suction – and yet the peak can remain persistently in place even if the subject swallows and opens their Eustachian tube. Negative middle ear pressure can even be measured when a subject has a permanently open (patulous) Eustachian tube, a situation that seems physically impossible. This paper reviews the paradox and concludes that in many cases of "negative middle ear pressure" the actual pressure inside the middle ear is in fact zero, but the tympanometric offset comes about because of the unappreciated action of the tensor tympani: when this muscle contracts, it pulls the eardrum inwards, and this inwards force is registered as negative middle ear pressure during tympanometry. That is, the force exerted by the muscle needs to be countered by a negative pressure in the ear canal in order to bring the eardrum back to its equilibrium position. This interpretation is reinforced by a number of findings in the literature, which are reviewed. A proposal for how tensor tympani effects might be separated from actual middle ear pressure offsets is made.

Key words: tympanometry • tensor tympani • negative pressure

CIEKAWY TYMPANOGRAM TYPU C: SKURCZ MIĘŚNIA NAPINACZA BŁONY BĘBENKOWEJ POZORUJE UJEMNE CIŚNIENIE W UCHU ŚRODKOWYM

Streszczenie

Zjawisko ujemnego ciśnienia w uchu środkowym wiąże się z pewnym paradoksem. Tympanogram typu C, którego szczyt wypada poniżej zerowej wartości ciśnienia, wydaje się wskazywać, że ciśnienie powietrza w uchu środkowym jest niższe od ciśnienia atmosferycznego – że występuje pewien stopień ssania – jednak szczyt często pozostaje niezmiennie w tym samym miejscu, nawet gdy osoba badana przełknie i odblokuje trąbkę słuchową. Ujemne ciśnienie w uchu środkowym można nawet zmierzyć, gdy osoba badana ma stale otwartą (rozwartą) trąbkę słuchową, co wydaje się fizycznie niemożliwe. W niniejszej pracy zbadano ten paradoks, dochodząc do wniosku, że w wielu przypadkach „ujemnego ciśnienia w uchu środkowym", ciśnienie to jest tak naprawdę zerowe, a przesunięcie tympanogramu jest efektem niedocenionego działania mięśnia napinacza błony bębenkowej: skurcz tego mięśnia wciąga błonę bębenkową do środka. Ta skierowana do wewnątrz siła jest w badaniu tympanometrycznym rejestrowana jako ujemne ciśnienie w uchu środkowym. Aby przywrócić błonę bębenkową do pozycji równowagi, działanie mięśnia musi zostać zrównoważone ujemnym ciśnieniem w kanale słuchowym. Ta interpretacja jest zgodna z wieloma opublikowanymi doniesieniami, których przegląd jest zamieszczony. Zaproponowano rozwiązanie, w jaki sposób można rozdzielić efekty działania mięśnia napinacza błony bębenkowej od właściwego efektu wyrównania ciśnienia w uchu środkowym.

Słowa kluczowe: tympanometria • mięsień napinacz błony bębenkowej • ujemne ciśnienie

Introduction

Tympanometry is a useful tool that allows a number of middle ear problems to be diagnosed. The procedure involves placing an acoustic probe in the ear canal and measuring the stiffness (or its inverse, compliance) of the middle ear system as the air pressure in the canal is continuously swept from low pressure (suction) to high pressure (overpressure). Figure 1 shows the main features of the arrangement. The minimum stiffness of the eardrum – its maximum compliance – will occur when the pressures on either side of the drum are equal, and this is the situation found in most subjects where the tympanogram registers a compliance peak at zero ear canal pressure. According to the classification system of Jerger, this is the 'Type A' tympanogram [1].

The focus of this paper is on the 'Type C' tympanogram in which the peak of the tympanometric curve is displaced appreciably towards negative values, taken by Jerger to be more negative than –100 daPa but which is not infrequently recorded at pressures of –200 daPa or more (1 daPa is a pressure of about 1 mm of water). In one report, a middle ear pressure of –380 mm of water is recorded [1a]. Figure 2 illustrates typical Type A and Type C tympanograms. Paradoxically, this "negative middle ear pressure" can persist even when the subject swallows and opens their Eustachian tube, an action which connects the middle ear cavity

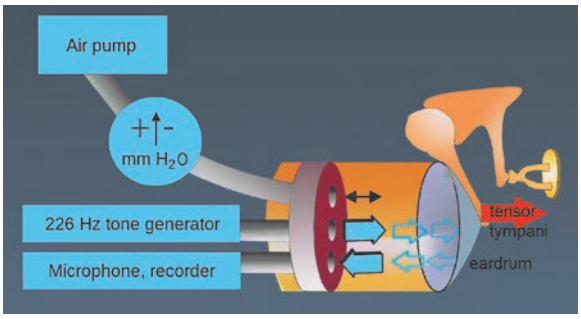

Figure 1. The elements of tympanometry. The impedance of the eardrum is sensed by comparing the original signal from a tone generator with that picked up with a microphone. During a measurement the air pressure in the ear canal is swept from –400 to +200 daPa with an air pump. Note how the tensor tympani is attached to the malleus and eardrum, and its contraction exerts an inwards force that needs to be countered with negative pressure in the ear canal to return the ear drum and ossicular chain back to their neutral position. That is, muscle contraction gives the same outcome as negative middle ear pressure. Figure adapted from Wikipedia Commons, CC BY-SA 3.0

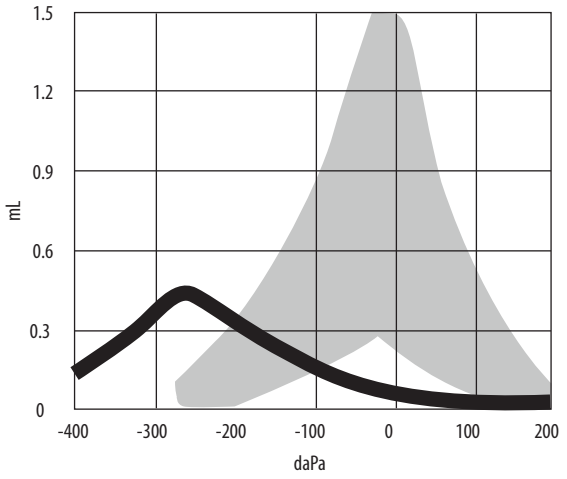

Figure 2. Illustration of Type A (light grey) and Type C (black) tympanograms. The peak of the curves reflects where the eardrum has its maximum compliance (measured as mL) and is taken to be where the pressure in the middle ear matches the pressure in the ear canal. That is, most people will show a peak near zero daPa (Type A), although there is a considerable range. If the peak occurs at negative values, it is called a Type C tympanogram (here registering a pressure of –275 daPa). Figure from audiologyonline.com

to the outside air (their ears 'pop'). The standard interpretation of a negative middle ear pressure is that the subject has a blocked Eustachian tube, usually due to otitis media (glue ear), allowing absorption of gas by the mucous layer lining the middle ear cavity and producing a negative pressure. But strangely, negative pressure can also be measured in subjects with a patulous (open) Eustachian tube. Sadé (2001) notes the "astounding and seemingly paradoxical fact of occasional association of atelectasis [retracted tympanic membrane] with a patent patulous eustachian tube" (p. 136 of [5]).

As has been nicely said, paradox is truth standing on its head in order to attract attention. Recognising the paradox, this paper seeks to resolve it. The conclusion is that true suction in the middle ear is not common (although possible); instead, a negative middle ear pressure reading as recorded by tympanometry is more often a case in which the tensor tympani muscle is under steady contraction, pulling the eardrum inwards. Otoscopically, the eardrum looks very similar to when it's really under suction but in most cases this is illusory. Tympanometry is really indicating that the force exerted by the muscle, tending to pull the eardrum inwards, needs to be countered by a negative pressure in the ear canal in order to restore the eardrum to its neutral position.

This hypothesis explains a number of anomalies in the literature and gives an insight into how the tensor tympani – the larger and stronger of our two middle ear muscles – regulates the operation of the ossicular chain. This paper supports the intralabyrinthine pressure theory of middle ear muscle action – the mechanism whereby the tensor tympani controls the pressure of fluid inside the cochlea, and so, acting like a hydraulic brake, controls hearing sensitivity.

Context for the evidence

There should really be no surprise to learn that contraction of the tensor tympani muscle draws the eardrum – the tympanum – inwards and tenses it, for that is just what its name (in Latin) means. When an audiologist inspects the eardrum with an otoscope, and they cause the tensor tympani to contract by applying a puff of air to the eye or brushing the cheek with a finger, that is just what is seen. A retracted drum will sometimes show 'retraction pockets', where parts of the drum are more yielding than others. You can make the tensor tympani contract yourself – just by yawning or tightly closing the eyes. The low fluttering rumble one hears is the sound of the muscle at work.

Perhaps this temporary reflex action of the tensor tympani has distracted people from appreciating that the muscle is designed for long-term constant contraction. It is a muscle made up of very fine fibres which are designed for sustained, isometric force generation (see Bell [2] for a detailed description of some of this unappreciated muscle's unique properties). According to this author, the main role of the tensor tympani is as a fast and precise acoustic gain controller: by constantly adjusting the force it applies to the ossicular chain, it changes the hydraulic pressure of fluids inside the cochlea and thereby changes the gain of the cochlear amplifier [2-4]. Because the fluids of the cochlea are virtually incompressible, and the round window so small, the range of movement of the muscle is minuscule, perhaps 0.1 mm [4], and so the constant activity of this nearly isometric muscle goes largely unnoticed.

Tympanometry is perhaps the best way of measuring tensor tympani activity directly. The pressure in the ear canal balances the pull of the muscle, and so the offset of the compliance peak (in daPa or mm of water) is a measure of the force the muscle is exerting. Referring to Figure 3, it can be calculated that a negative pressure of 100 daPa (= 1 kPa) acting over the area of the eardrum (about 50 mm^2) generates a force of 50 mN, which counters the contractile force of the muscle. In other words, when tympanometry

records a middle ear pressure of –100 daPa, the tensor tympani is exerting a counteracting force of about 5 grams weight, a figure that matches the likely power of a muscle this size (a body of 25 mm).

It is worth noting at this point that if that same force is transferred to the ossicular chain and finally to the stapes, which has an area of 3 mm^2, then the force will create a hydraulic pressure in the sealed cochlea of about 20 kPa (2000 mm water). This is important in understanding how the middle ear muscles adjust hearing sensitivity, and will be brought up later in connection with Eustachian tube dysfunction, which also relates to middle ear pressure anomalies.

Jerger notes that negative middle ear pressures are a regular finding. He says that "slight" negative pressures are "quite common" in many otherwise normal ears, and even values more negative than –100 daPa are routinely encountered. He presents data for 142 normal ears, and his graph shows that for people aged 6 to 59 about 5% have Type C tympanograms. The figure rises markedly for children aged 2 to 5, where about 30% show Type C tympanograms, although the ears are still classed as "normal". In other words, Type C tympanograms occur regularly, and this raises the question of where the "negative pressure" comes from.

Evidence that actual middle ear pressure is close to zero

Much has been written about negative middle ear pressure but a common problem is that most experimenters almost invariably interpret negative pressures as recorded by a tympanometer as an actual state of suction in the middle ear. In this section we systematically address the measurements that have been made and suggest that, in most situations, the true state of affairs is a middle ear pressure close to zero – but associated with a substantial inwards pull of the tensor tympani, which masquerades as negative pressure inside the middle ear.

1. The effect of swallowing

The middle ear can in large part be considered a sealed cavity, but whenever we swallow the Eustachian tube connecting the middle ear cavity to the back of the throat opens. Figure 4 illustrates the arrangement.

If there is indeed a pressure difference between the middle ear and the atmosphere – such as during an aircraft descent or travelling several floors in a lift – we sense the pressure and automatically swallow, at which point we hear our ears 'pop'.

A number of studies of direct middle ear pressure measurements – made with an invasive technique involving the puncturing of the tympanic membrane with a needle and measuring the pressure via tubing connected to a manometer – confirm that the middle ear pressure returns to zero after swallowing (e.g. Tideholm [6]), at least in normal subjects. Although the invasive technique is difficult to apply for long periods, results tend to confirm that normal subjects usually have middle ear pressures not far from zero. The more complex situation of otitis media is taken up in later discussion.

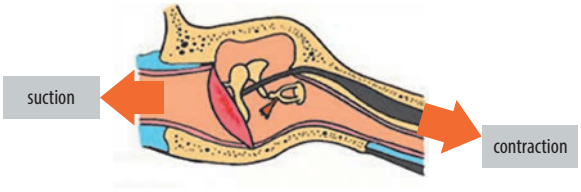

Figure 3. Relation between the eardrum (red) and its attachment to the tensor tympani (dark brown). When the tensor tympani contracts, it pulls on the malleus and the attached eardrum, pulling them inward. Based on the area of the eardrum, a contractile force of 5 grams weight will be needed to counterbalance a suction in the ear canal of about –100 daPa in order to keep the eardrum in a neutral position. Adapted from a figure by MF Dauzvardis, Loyola Medical School

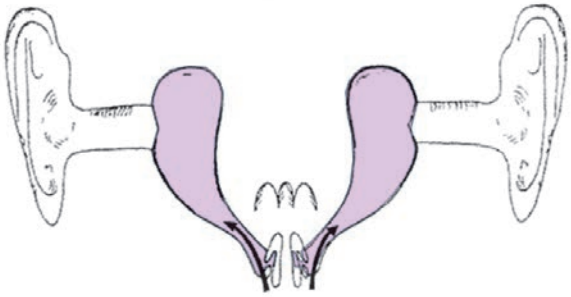

Figure 4. The middle ear opens via the Eustachian tube to the throat whenever we swallow, an action that establishes zero pressure across the eardrums. Under this condition, tympanometry should now register zero middle ear pressure. Adapted from Sadé [5]

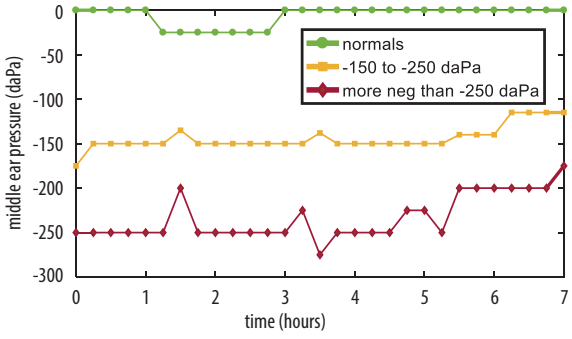

Figure 5. Tympanometry readings taken every 3 minutes for 7 hours in three groups of subjects (plotted as 15 minute averages). The groups were: normals ($n = 5$); patients with tympanometric pressure –150 to –250 daPa ($n = 6$); and patients with pressure more negative than –250 daPa ($n = 7$). Over the whole 7 hours, none of the patients' 3-minute measurements ever reached zero. Replotted from Grøntved [7]

Since we automatically swallow every minute or so, one might expect that it would be very difficult to sustain a middle ear pressure much away from zero. Surprisingly, however, monitoring of 20 patients who complained of hearing loss found that, measured tympanometrically every 3 minutes for 7 hours, their average pressure was –150 mm of water, and that for two-thirds of them (13 or 65%) the pressure never hit zero [7]. For 5 control subjects, the pressure did not deviate far from zero. See Figure 5.

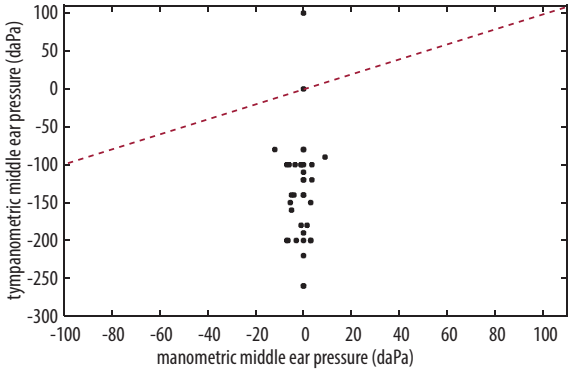

Figure 6. Lack of correlation between tympanometric readings (*y*-axis) and concurrent measurements using a needle and manometer (*x*-axis) in 36 subjects with secretory otitis media. If there were a correlation, it would show as a 1:1 plot (dashed red line). However, in this case the points are far removed and the *r*-squared value is minute (less than 0.0001). Data from Fig. 2 of [34]

Grøntved and colleagues [7] interpreted the fact that the 13 patients who "never equalized their negative middle ear pressure" had Eustachian tubes which did not open during the test period, but there is room for doubt, and here we suggest an alternative explanation. The authors are puzzled that even though the tube never appeared to open during 7 hours of measurement, the recorded tympanometric pressure in any patient never reached –600 daPa, the supposed equilibrium pressure of the middle ear mucosa. They remark that a pressure more negative than –150 mm is considered an indicator of serous otitis media and hence a candidate for placement of a ventilation grommet in the tympanum – the traditional treatment for clearing infection and relieving negative middle ear pressure.

2. Poor correlation between tympanometry and manometry

In order to understand the unusual tympanometric findings, one approach is to use both tympanometry and a manometric needle one after the other on the same set of patients. Figure 6 shows the results in 36 ears of children who were about to have a grommet inserted for treatment of serous otitis media (SOM) [34]. The manometry showed an average pressure of just –1.7 mm water (effectively zero) whereas tympanometry showed an average pressure of –130 mm water, two orders of magnitude larger. These data indicate that something is seriously awry in our interpretation of tympanometry. To add weight to that statement, Sadé and colleagues later made tympanometric measurements after grommets had been inserted [34]. The natural expectation is that the ventilated ear should show zero pressure. Remarkably, Sadé reports that "tympanometric measurements did not change after insertion of the ventilating tube" (p. 61) [34]. He appears unperturbed by these findings and simply concludes that "tympanometry … does not reflect in these cases the real intratympanic pressures." Whether grommets actually help overcome SOM is a separate matter, but we certainly need a better understanding of what negative middle ear pressure really means. Although Sadé's work was done towards the end of last century, there are still recent reports which claim that negative middle ear pressures are indicative of SOM and the need for grommets. It is a concern that most patients with retracted ear drums do not complain of ear pressure [5]. The effect of grommets on otitis media is considered later, but the important point is that negative middle ear pressures may simply reflect different degrees of tensor tympani contraction, and so negative pressures may well be a fairly normal circumstance.

3. Of 'blocked' Eustachian tubes

The major implication of this revised outlook is that there have been many cases of negative middle ear pressure in the past which have been falsely diagnosed as blocked Eustachian tube or Eustachian tube dysfunction – the standard interpretation of negative pressure, based on the observation that a truly blocked Eustachian tube leads to absorption of gas by the mucosa lining the middle ear cavity and creates actual suction (the supposed equilibrium pressure of the middle ear mucosa is presumed to be –600 daPa [7]). This new perspective casts doubt on such a conclusion, and in the course of this paper more evidence is marshalled that measurements of middle ear pressure are often better interpreted as tension from the tensor tympani.

However, returning to address the question of "blocked" Eustachian tubes, it is indeed true that subjects with otitis media have pressures in the negative range (as high as –250 daPa; Tideholm [6]). Some studies in which a needle has been used to penetrate the mastoid or the tympanic membrane have confirmed this result [8], although the results are open to interpretation because of problems with leaks around the puncture site. It is difficult to say, without dedicated experiments, the extent of this misdiagnosis, but there are some useful papers that shed light on the issue, and this section looks closer at them.

Misdiagnosis of a blocked Eustachian tube is not a trivial matter. Gaihede and colleagues [9] report that treatment with ventilation tubes (or grommets) – the standard response – was associated with a 66% rate of tympanic membrane pathology compared to untreated ears (12%).

Magnuson [10] discusses the subject of "atelectatic ears" in children (those showing retraction pockets) and casts doubt on the classical "hydrops ex vacuo" theory in which middle ear effusion (serous otitis media) is presumed to result in negative pressure. He relates how experimental and clinical data are inconsistent in trying to find a causal relationship between tubal obstruction, high negative pressure, effusion, and tympanic membrane retraction. Some prior experiments had shown that negative pressure developed by gas absorption is negligible. In his own work, retraction pockets were visible in large numbers of ears even though they had been repeatedly treated with ventilating tubes. The factors involved were complex, but a consistent observation was an inability to equalize negative intratympanic pressure. This suggests that the inwards pull of the tensor tympani may be able to act on the eardrum in such a way as to create retraction pockets.

A hypothesis consistent with the findings is that infection of the middle ear (otitis media) leads to irritation of the middle ear muscles, and this in turn causes the tensor

tympani to contract and generate an inward force which mimics negative middle ear pressure. There is good evidence supporting this interpretation: histopathology of the muscle in cases of otitis media has shown more inflammatory cells and more hypercontracted fibers [11]. The authors of this study, which involved 105 temporal bones, also found that the muscle fibres displayed contracture knots consistent with hypercontraction. Why otitis media develops so commonly in young children but progressively disappears with age is an area of research that would pay large dividends in terms of child health and hearing preservation. In any case, the immediate message should be that a negative middle ear pressure is most likely a sign of an irritated tensor tympani and that the condition should not be used as an automatic indicator for the insertion of grommets.

4. Subjects with patulous Eustachian tube

Normally, the Eustachian tube opens whenever we swallow (Figure 4), and after a few swallowings the middle ear pressure is usually very close to zero [12, 13]. Virtanen has systematically investigated the opening by placing a sound source in the nostril and a microphone in the ear canal (a method called sonotubometry). Using such a technique, opening of the Eustachian tube can be directly measured by recording a sudden increase in microphone level. This novel approach is useful for finding out what is going on in cases of so-called patulous Eustachian tube where the tube appears to be more or less stuck open, an annoying condition where people complain of their own voice being too loud or echoey, they can hear their own breathing, and everything sounds like their head is an empty barrel. There can also be sensations of fullness or pressure.

Virtanen used sonotubometry to study cases of patulous Eustachian tube in 30 patients who had a wide range of symptoms and response patterns [12,13]. There were 8 subjects who appeared to have Eustachian tubes that were more or less continuously open, and, remarkably, one subject had a measured middle ear pressure of –200 mm water, which seems physically impossible. This supports the interpretation being put forward here that really there is an active tensor tympani at work whose steady level of contraction masquerades as negative middle ear pressure under tympanometry. Confirming this interpretation, this same patient also had a perforated tympanic membrane, meaning it was absolutely impossible for a pressure difference to exist between the eardrum and the middle ear. [Note that the hole must have been quite small for the tympanometer not to have recorded a peak at zero pressure during its rapid pressure sweep; the same methodological consideration suggests that the true (hole-free) reading must have been even more negative than the reported value.] Another patient reported by Virtanen had an Eustachian tube that closed only slowly after swallowing, and this patient too had a perforated tympanic membrane; once again, though, the middle ear pressure registered as markedly negative: –100 mm water.

These cases are not just isolated aberrations. A few years later, Virtanen [14] published a survey of 92 healthy adults who were just recovering from a common cold. Some had a blocked Eustachian tube while others did not, and all subjects underwent sonotubometry and tympanometry. Based on mismatches between the two tests, Virtanen concluded that "a negative middle ear pressure evaluated by tympanometry does not always denote a closed tube" (p. 766) [14]. An illustration in his paper shows a tympanogram with a peak at –155 mm water and an accompanying sonotubometry trace showing the Eustachian tube opening. More generally, a supporting table shows that 21 of 28 subjects (75%) had positive sonotubometry (opening of the Eustachian tube) and middle ear pressure of –51 to –75 mm water, with other categories of negative middle ear pressure ranges also showing that an open Eustachian tube is compatible with sustained sub-zero pressures. For example, even when the middle ear pressure was more negative than –75 mm water Virtanen still observed some tubal openings.

Cases of patulous Eustachian tube function are complex, and the tube is more than just a valve that opens and shuts. Ventilation appears to involve complex coordination of muscles – the tensor veli palatini and its close anatomical companion, the tensor tympani [11,15,16]. Magnuson [17] believed the real function of the Eustachian tube to be "pressure regulation", not just periodic ventilation. It seems that dysfunction of these two interrelated muscles somehow leads to patulous Eustachian tubes, where "patulous" can have a range of functional meanings. One interpretation from Virtanen's work is that heightened activity of the tensor veli palatini is accompanied by additional activity of the tensor tympani, and this can be recorded as negative middle ear pressure – even though the true pressure inside the middle ear may be zero.

In this context, Magnuson [17] recorded some interesting side-effects of 42 subjects who seemed to have patulous Eustachian tubes and who used a "sniffing" technique to temporarily overcome their symptoms. Sniffing, which is a way of evacuating air from the middle ear, was found to have two notable side-effects. 1) It overcame hyperacusis: with "drums out" after swallowing, the subject's voice was over-loud; with "drums in" hearing was more comfortable, natural, and distinct. 2) It reduced "fluttering" of the eardrums, which is the same sensation most people experience when they activate their tensor tympani by yawning or tightly closing the eyes. Finally, there was a third related observation: 3) instead of sniffing, performing a Valsalva enabled most patients to increase their hearing acuity and perceive weak sounds. Importantly, all three side-effects are compatible with the intralabyrinthine pressure theory [3] which suggests that middle ear muscles adjust hearing gain through varying the pressure of fluid inside the cochlea. Understandably, if the tensor tympani is not operating properly, another way to control intralabyrinthine pressure (and hearing acuity) is to vary middle ear pressure – by sniffing or Valsalva manoeuvre – actions which will cause the ear drum to bulge out or be pulled in, in turn causing the ossicular chain and ultimately the stapes to increase or decrease intracochlear pressure. Viewed in this light, patulous Eustachian tube could be a misnomer: it is actually the dysfunction of the tensor tympani which is giving rise to the annoying symptoms.

Clearly, much more work is needed to work out exactly what is going on, but the accumulated evidence is that, across a range of subjects, contraction of the tensor tympani can, through pulling the eardrum inwards, accurately mimic negative middle ear pressure.

Evidence that changes in pressure are really changes in tension

The central hypothesis put forward here is that changes in middle ear pressure as measured tympanometrically are really a reflection of changes in the state of tension of the tensor tympani muscle. Like any isometric muscle, that tension will tend to vary from one individual to another and from time to time. The evidence can be categorised under the following headings.

1. Rapid changes in values

A good indication that a tympanometer measures muscle tension rather than pressure of the middle ear is the magnitude and rapidity with which meter readings change. Gaihede and Ovesen [18] reported pairs of tympanometric measurements made immediately after one another (without removing the probe). Among a set of 80 ears they found a standard deviation of the difference of 7 daPa, a measure that was larger than the resolution of their tympanometer (5 daPa). When follow-up readings were made several months later on 20 ears, the standard deviation was now 17 daPa. In one case, the initial reading was –45 daPa and it increased to –240 daPa at follow-up. The authors mention literature reports where middle ear pressures have been found to change by up to 30 daPa within minutes, and that day-to-day shifts from Type A to Type C are well known. Clearly, we are dealing with a dynamic system, and this is more likely to reflect the action of a muscle than absorption of gas from a mucous lining.

2. Findings from Meniere's disease

A striking result that reinforces the muscle interpretation is the study by Park [19] of middle ear pressure in normal and in Meniere's patients. It has already been suggested that Meniere's disease is caused by excessive activity of the tensor tympani [4], so it is highly relevant that the mean middle ear pressure of 30 normals was found to be 4 daPa (±5 daPa) whereas the figure for 33 Meniere's patients was –43 daPa (±75 daPa). It would be revealing to follow the course of tympanometric pressure prior to, and after, a Meniere's attack.

Historically, it is of interest that there has long been a suspected association between middle ear pressure and Meniere's disease, beginning perhaps with the insights of Tumarkin in 1966 [20]. This author rejected the orthodox notion that the source of "labyrinthopathies" (including Meniere's disease) must be found in the inner ear. Instead, he boldly suggested, following some earlier opinions, that the cause of Meniere's disease may result from middle ear dysfunction. It should be noted that Tumarkin's explanation does not explicitly mention the tensor tympani, but he does point to negative middle ear pressure as a key factor. Unconventionally, he tried inserting grommets into the tympanic membrane to treat 20 cases of vertigo, and reported that the results were "little short of startling", with vertigo disappearing entirely in practically every case.

Tumarkin's paper attracted considerable interest, and at least 36 surgeons tried the procedure, with generally positive outcomes [21]. However, enthusiasm waned and grommets have not turned out to be a cure for Meniere's disease, although they have remained popular for treating otitis media. The point of interest, however, is that many investigators have confirmed that a large proportion of Meniere's patients have what has been called "intermittent Eustachian tube blockage". Hall and Brackmann [22] found that 25 of 81 Meniere's patients (31%) had a negative middle ear pressure of –100 mm water or greater, which, following the conventional picture, is interpreted as "a blocked Eustachian tube". Given what has been said to this point, however, it becomes questionable whether the Eustachian tube really is blocked.

Hall and Brackmann's paper is particularly illuminating, however, in finding that the middle ear pressure varies in line with the strength of the symptoms. For example, one case showed no symptoms when the middle ear pressure was zero, mild symptoms (slight feeling of fullness, vertigo, tinnitus) when the pressure was measured as –125 mm water, and with strong symptoms the pressure was –175 mm water. They also relate how the low-frequency hearing of some patients could by temporarily improved by 5–15 dB by increasing middle ear pressure via a Valsalva manoeuvre.

These are strong indications. Intriguingly, however, a later paper by one of the same authors [23] backs away from the association between negative middle ear pressure and Meniere's, claiming only that, based on a larger sample, the incidence of Type C (or B) tympanograms was only about the same as what Jerger found in normals. We are not told much about the change in outlook apart from enlarged numbers, but given the strong findings of the earlier work, and the equally strong findings reported later by Park (2012) [19], one might reasonably suspect that something peculiar is going on and that perhaps a key factor may have been missed. Closer investigation might provide the necessary clues, and in any such work the tensor tympani hypothesis stands as a good candidate for resolving these peculiar anomalies.

The difficulty associated with Meniere's disease is that it is such a multifactorial disease, and, despite dissenting voices, most research is still focused on the inner ear (see the review by Oberman [24]). Nevertheless, it is encouraging that Oberman's review reminds us that the middle ear muscles remain in the picture (p. 259). It is suggested that the pressure model as described by Bell [4] might help in resolving the matter.

3. Voluntary contraction of middle ear muscles

Logically, perhaps the clearest and most direct evidence in support of the hypothesis under consideration is that gathered from those rare individuals who can voluntarily contract their middle ear muscles.

An earlier paper [4] has already set out an array of evidence that voluntary contraction of the middle ear muscles leads to a loss in hearing sensitivity at low frequencies – both for air conduction and, tellingly, bone conduction. The explanation offered was that contraction of the tensor tympani increased hydraulic pressure in the labyrinth, and a reduction in gain of the cochlear amplifier (see Bell [3] for

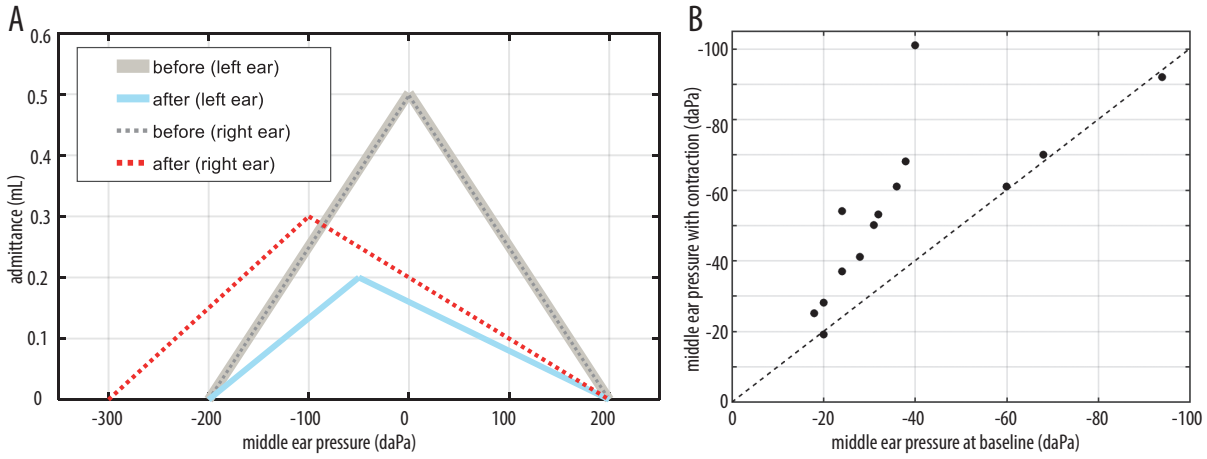

Figure 7. Effect of middle ear muscle contraction on tympanometric peak pressure. (A) Data for a single subject before and during voluntary contraction: the peak shifted from 0 to –75 daPa for the left ear and 0 to –100 daPa for the right. (B) Data from 14 ears of 8 subjects who could voluntarily contract their middle ear muscles. The x-axis shows middle ear pressure before contraction; y-axis shows middle ear pressure with contraction. The diagonal is the 1:1 line. [Data in A from [25]; B from [26]; with permission]

more detail). The general point was that Meniere's disease might be due to dystonia or cramp of the middle ear muscles. In line with that thinking, the question now becomes does voluntary contraction also cause an apparent increase in negative middle ear pressure as measured tympanometrically? Indeed, to get straight to the point, all the indications are generally positive, and the rest of this section outlines published work that directly supports this relationship.

A prime example of what happens when a subject contracts their tensor tympani is shown in Figure 7A. The data comes from Angeli [25] and shows the negative shift in tympanometric peak pressure when the subject's middle ear muscles are contracted. When the muscle is relaxed the peak occurs at zero pressure, but when the muscle is contracted it occurs at –50 daPa (in the left ear, blue) and –100 daPa (in the right, red). An average figure of –75 daPa corresponds to the pressure needed to counteract an inward force of about 4 g wt, a direct and credible interpretation.

More substantial evidence comes from a study by Aron and colleagues [26] which investigated 8 subjects who were also able to contract their middle ear muscles. The tympanometric results for 14 of their ears are shown in Figure 7B which shows middle ear pressure readings before and during contraction. In most ears tested, the apparent middle ear pressure became more negative during contraction, shifting from an average baseline pressure of –38 daPa to –54 daPa, a change which was statistically significant ($p < 0.01$). Of course, a natural inference is that even the initial baseline values represent a standing level of tension of the tensor tympani. As it happened, the main focus of Aron and colleagues was not so much on middle ear pressure as on middle ear compliance, particularly how it may be affected by the stapedius reflex. Nevertheless, despite the slightly different focus, the researchers do explicitly conclude, after conducting analogous experiments in cadavers, that "Pulling on the TT [tensor tympani] resulted in a more negative measured peak MEP [middle ear pressure] measurement" (p. 377) [26]. They also made the hypothesis that "a TM [tympanic membrane] tensed by contraction of the TT requires relatively more negative EAC [external ear canal] pressure to bring it back to its most compliant state, so that peak admittance is measured at a more negative EAC pressure" (p. 379).

Given this insight, it is surprising that there has not been a thorough investigation of the role of the tensor tympani in establishing negative middle ear pressure. Part of the difficulty involves understanding that the tensor tympani could exist in a state of continuous long-term contraction. In analogy with the acoustic reflex of the stapedius, the thinking seems to have been that the tensor tympani undergoes reflex contraction in a similar way, and that there does not seem to be any point in having the muscle contracted for any extended period of time. However, the idea of the muscle being in an isometrically contracted state so that it can immediately respond to ambient sound and appropriately adjust hearing sensitivity casts fresh light on the true function of this long-neglected and misunderstood muscle.

Another part of the perceived difficulty may derive from the unappreciated greater stiffness of dead muscle compared to that which is living. Thus, Aron and colleagues find (Fig. 9 [26]) that a temporal bone specimen required a substantial force of 60 g wt to resist an ear canal pressure of 100 daPa, whereas calculations based purely on ear drum area and an active, compliant muscle indicate that a force of 5 g wt should be sufficient to counteract the forces involved. It appears that living tissue is much more responsive than dead, and this seems reasonable.

Further work on the tympanometric effects of tensor tympani contraction can be found in Bance [27] and Wickens [28], but the key measure in these investigations is again compliance rather than middle ear pressure as such. Not unexpectedly, there is a general correlation between the two, so that higher pressures (positive and negative) are associated

with greater stiffness (lower compliance), but inspection of the two reports just cited indicates that there sometimes appears to be a disjunct. Nevertheless, the findings are generally consistent with an active tensor tympani. In Bance [27], tympanograms were collected before and after voluntary contractions from 2 subjects, but unfortunately not during them (sustaining a strong contraction isn't easy). Stimulation of the tensor tympani by a puff of air to the eye produced increases in impedances and in air- and bone-conduction thresholds, but, curiously, other means of stimulation – such as stroking the subject's cheek or asking them to subvocalise (count silently) – did not. The second work [28] reported on 5 subjects who could all voluntarily contract their tensor tympani muscles for seconds at a time, allowing tympanometry to be done during a contraction, and again air- and bone-conduction thresholds were affected.

4. Multifrequency tympanometry

More recently, use of tympanometry has in many cases been replaced by tympanometry based on a range of frequencies rather than just the standard 226 Hz impedance measures [29]. Often the technique is called multifrequency tympanometry [30], wideband acoustic immittance [31], or wideband absorbance (WBA) tympanometry [32]. The technique supplies an extra frequency dimension to the conventional tympanogram, providing a colourful 3D plot and additional diagnostic information. However, there is a ridge-line corresponding to maximum admittance where the middle ear pressure balances the ear canal pressure, and this ridge-line is, as expected, largely independent of frequency. Thus, the middle ear pressure can be recorded in much the same way as with the traditional method.

Useful insights come from a recent paper [32] where Karuppannan and Barman used WBA tympanometry to explore cases of abnormal middle ear pressure, including 30 cases of negative middle ear pressure in 25 adults (average pressure of –207 daPa). The adults had no active ear discharge, so the confounding factor of otitis media, often seen in children, could be ruled out. Moreover, the inference can be made that the subjects did not have 'blocked Eustachian tubes', which is the usual diagnosis made in pediatric cases when high negative middle ear pressures are recorded. Although it is true that a blocked Eustachian tube can lead to negative middle ear pressure, that does not mean the reverse is the case, and it seems that conflating the two has led to erroneous conclusions. This paper has sought to establish that negative middle ear pressure can occur quite normally as a consequence of sustained tensor tympani contraction, and it can be presumed that this is what happened in Karuppannan and Barman's subjects.

The authors measured wideband absorbance under two conditions: at zero ear canal pressure and at a pressure that exactly counterbalanced the tympanometrically measured middle ear pressure. In this way, they were able to see the effect that middle ear pressure (or, as it is suspected, tensor tympani contraction strength) was having on the ear drum. Their results (their Fig. 2; Figure 8 here) showed that there was a large difference in absorbance between the two conditions at low frequencies (0.2–1 kHz); however, we see that the absorbance is almost identical from 3–6 kHz. Importantly, this suggests a possible way of separating the effects of a tensor tympani contraction from the effects of actual air pressure within the middle ear. Although both factors will naturally stretch the ear drum and change its acoustic impedance, it is clear from Figure 8 that they operate over different frequency ranges. It is suggested that the eardrum's tension (and impedance) changes as a function of middle ear pressure, and this can be seen as a change in absorbance from 0.2–1 kHz. However, for subjects whose tensor tympani is already contracted, the absorbance at 3–6 kHz is independent of pressure differences across the eardrum – the WBA has plateaued – and so in normal subjects it might be expected that this range is still somewhat sensitive to the state of the tensor tympani.

The above interpretation can be supported by comparison with the work of Shaver and Sun [33] which involved normal subjects who were able to adjust their actual middle ear pressure via a Toynbee manoeuvre (swallowing while keeping the nose pinched). The authors found (their Fig. 1 and 2; Figure 8 here) that the change in actual middle ear pressure, to a mean of –160 daPa, changed ear canal absorbance in the sensitive 0.2 and 1 kHz range, but also, it should be noted, over the 3–6 kPa range, where Karuppannan and Barman saw no change. (Actually, Shaver and Sun measured reflectance, but turning their plots upside down gives absorbance.) That is, both sets of experiments involved similar middle ear pressures, but in one case it was actual Toynbee-induced pressure while in the other it was a persistent condition. The difference in WBA at 3–6 kHz, it is suggested, relates to the state of the tensor tympani: in the clinical group the muscle is strongly active, but in the normals it is more relaxed, and so the difference in this frequency range could be a sensitive way of separating out the degree of tensor tympani involvement.

The conclusion is that it should be possible, by using a combination of techniques and subject groups, to separate the individual effects of actual middle ear pressure and tensor tympani contraction. This would be a real achievement in unpicking the paradoxes that have accumulated within the tympanometric field.

Discussion and conclusions

The above text suggests that negative middle ear pressure should, in the first instance, be interpreted as an inwardly directed force created by steady contraction of the tensor tympani. Physically, there is no actual negative pressure in the middle ear; it's just that a tympanometer will react to any muscle-generated force in the same way as if there were. If true, there are a number of major implications of this new interpretation.

It is surprising that negative middle ear pressure has been such a common measure in audiology but has always been interpreted so literally despite persistent anomalies that directly indicate otherwise. As suggested, one reason for the neglect comes from our lack of appreciation of the important role the tensor tympani plays in regulating inner ear pressure, and thereby controlling auditory sensitivity. The muscle, because it is isometric, doesn't appear to 'do' much (other than just tensing the tympanum) and so it has generally been overlooked. Together with the

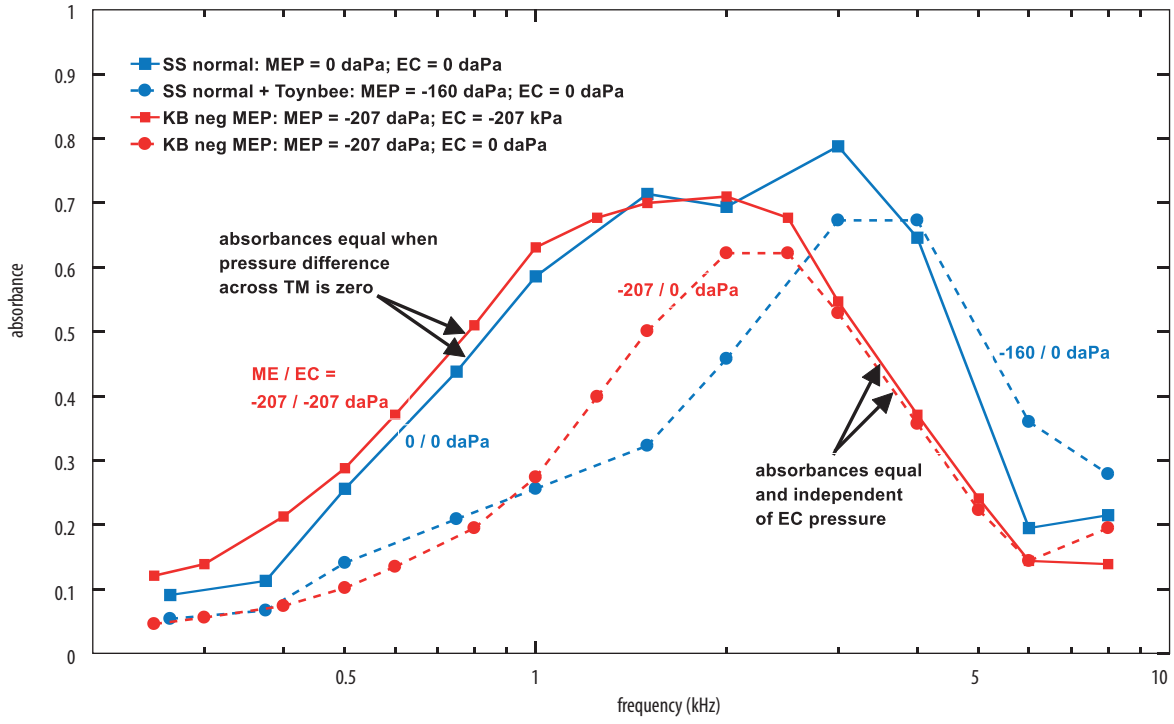

Figure 8. Similarities and differences in wideband absorbance (WBA) between two groups of subjects, both with negative middle ear pressure but of different origins. The first group were 11 normal adults who used a Toynbee manoeuvre to generate an average middle ear pressure of –160 daPa (blue lines, Shaver and Sun [33]); the second group were 25 adults (30 ears) from an audiological clinic diagnosed with negative middle ear pressure averaging –207 daPa (red lines, Karuppannan and Barman [32]). Note the similarity in WBA between 0.2 and 1 kHz (continuous lines) when there was equal pressure on either side of the eardrum. Similarly, the WBAs for both groups are seen to be similar between 0.2 and 1 kHz when the pressure is about 180 daPa across the eardrum (dashed lines). Note however the differences between 3 and 6 kHz: WBA is virtually independent of ear canal pressure for the clinical group, but there are appreciable differences for the normal group. It is suggested that the WBA differences at 3–6 kHz relate to the state of the tensor tympani: for the clinical group the muscle is strongly contracted but for the normals it is not.

unappreciated role of intralabyrinthine pressure, the two elusive factors have prevented research from moving forward. Clearly, much more research is needed to explore the details of these subtle mechanisms.

The central idea behind this paper is that tympanometric peak pressure is made up of two components: actual middle ear pressure, which in most commonly around zero, and tensor tympani tension, which gives rise to an effective "negative pressure" as it pulls the eardrum and whole ossicular chain inwards. Up until now, these two components have been conflated – the tympanometer reading has been interpreted as being solely due to pressure inside the middle ear – which has given rise to the paradoxical findings set out above. Otitis media has often been diagnosed on the basis of negative middle ear pressure; however, it is suggested here that the negative middle ear pressure may be in fact be a side-effect of bacterial infection: irritation of the middle ear muscles due to infection causes the tensor tympani to contract, and this contractile force is likely to be what the tympanometer is reacting to [11].

In order to set matters straight, we will need to have a way of measuring actual middle ear pressure separately from the contractile state of the tensor tympani. Invasive measures like penetrating the middle ear space with a needle [8] are far from satisfactory, and our text suggests that careful use of wideband absorbance techniques – selecting frequency ranges which are characteristic of the impedance of the eardrum and of the tensor tympani – may provide a solution. The ranges 0.2–1 kHz and 3–6 kHz may be good starting points.

It is hoped this paper may help overcome some obstacles in tympanometry and allow further progress to be made. The tensor tympani is a neglected, but essential, component of our intricate hearing system – it is at the heart of an active gain-regulation loop – and tympanometry is a sensitive but powerful tool for understanding how it works.

References

1. Jerger J. Clinical experience with impedance audiometry. Arch Otolaryngol, 1970; 92: 311–24.

1a. Renvall U, Holmquist J. Tympanometry revealing middle ear pathology. Ann Otol Rhinol Laryngol, 1976; 85 (Suppl. 25 Pt 2): 209–215.

2. Bell A. A fast, "zero synapse" acoustic reflex: middle ear muscles physically sense eardrum vibration. J Hear Sci, 2017; 7(4): 33–44.
3. Bell A. How do middle ear muscles protect the cochlea? Reconsideration of the intralabyrinthine pressure theory. J Hear Sci, 2011; 1(2): 9–23.
4. Bell A. Middle ear muscle dysfunction as the cause of Meniere's disease. J Hearing Sci, 2017; 7(3): 9–25.
5. Sadé J. Hyperectasis: the hyperinflated tympanic membrane. The middle ear as an actively controlled system. Otology and Neurotology. 2001; 22: 133–9.
6. Tideholm B, Jonsson S, Carlborg B, Welinder R, Grenner J. Continuous 24-hour measurement of middle ear pressure. Acta Otolaryngol, 1996; 116: 581–8.
7. Grøntved A, Krogh H-J, Christensen P-H, Jensen PO, Schousboe HH, Hentzer E. Monitoring middle ear pressure by tympanometry. Acta Otolaryngol, 1989; 108: 101–6.
8. Dirckx JJJ, Marcusohn Y, Gaihede M. Quasi-static pressures in the middle ear cleft. In: Puria S, Fay RR, Popper AN, editors. The Middle Ear: Science, Otosurgery, and Technology. New York: Springer; 2013. p. 93–133.
9. Gaihede M, Lildholdt T, Lunding J. Sequelae of secretory otitis media: changes in middle ear biomechanics. Acta Otolaryngol, 1997; 117: 382–9.
10. Magnuson B. The atelectatic ear. Int J Pediatr Otorhinolaryngol, 1981; 3: 25–35.
11. Abdelhamid MM, Paparella MM, Schachern PA, Yoon TH. Histopathology of the tensor tympani muscle in otitis media. Eur Arch Otorhinolaryngol, 1990; 248: 71–8.
12. Virtanen H. Patulous Eustachian tube: diagnostic evaluation by sonotubometry. Acta Otolaryngol, 1978; 86: 401–7.
13. Virtanen H. Sonotubometry: an acoustical method for objective measurement of auditory tubal opening. Acta Otolaryngol, 1978; 86: 93–103.
14. Virtanen H. Middle-ear pressure and eustachian tube function. Arch Otolaryngol, 1982; 108: 766–8.
15. Ramirez Aristeguieta LM, Ballesteros Acuna LE, Sandoval Ortiz GP. Tensor veli palatine and tensor tympani muscles: anatomical, functional and symptomatic links. Acta Otorrinolaringol Esp, 2010; 61: 26–33.
16. Kierner AC, Mayer R, Kirschhofer Kv. Do the tensor tympani and tensor veli palatini muscles of man form a functional unit? Hear Res, 2002; 165: 48–52.
17. Magnuson B. Tubal closing failure in retraction type cholesteatoma and adhesive middle ear lesions. Acta Otolaryngol. 1978;86:408–17.
18. Gaihede M, Ovesen T. Precision of tympanometric measurements. J Speech Lang Hear Res, 1997; 40: 215–22.
19. Park JJ-H, Luecke K, Luedeke I, Emmerling O, Westhofen M. Long-term middle ear pressure measurements in inner ear disorders. Acta Otolaryngol, 2012; 132: 266–70.
20. Tumarkin A. Thoughts on the treatment of labyrinthopathy. J Laryngol Otol, 1966; 80: 1041–53.
21. Lall M. Meniere's disease and the grommet (a survey of its therapeutic effects). J Laryngol Otol, 1996; 83: 787–91.
22. Hall CM, Brackmann DE. Eustachian tube blockage and Meniere's disease. Arch Otolaryngol, 1977; 103: 355–7.
23. Forquer BD, Brackmann DE. Eustachian tube dysfunction and Meniere's disease: a report of 341 cases. Am J Otol, 1980; 1: 160–2.
24. Oberman BS, Patel VA, Cureoglu S, Isildak H. The aetiopathologies of Meniere's disease: a contemporary review. Acta Otorhinolaryngol Ital, 2017; 37: 250–63.
25. Angeli RD, Lise M, Tabajara CC, Maffacioli TB. Voluntary contraction of the tensor tympani muscle and its audiometric effects. J Laryngol Otol, 2013; 127: 1235–7.
26. Aron M, Floyd D, Bance M. Voluntary eardrum movement: a marker for tensor tympani contraction? Otol Neurotol, 2015; 36: 373–81.
27. Bance M, Makki FM, Garland P, Alian WA, van Wijhe RG, Savage J. Effects of tensor tympani muscle contraction on the middle ear and markers of a contracted muscle. Laryngoscope, 2013; 123: 1021–7.
28. Wickens B, Floyd D, Bance M. Audiometric findings with voluntary tensor tympani contraction. J Otolaryngol Head Neck Surg, 2017; 46: 2.
29. Diniz Hein TA, Hatzopoulos S, Skarzynski H, Colella-Santos MF. Wideband tympanometry. In: Hatzopoulos S, editor. Advances in Clinical Audiology. London, UK: InTech; 2017. p. 29–45.
30. Sugasawa K, Iwasaki S, Fujimoto C, Kinoshita M, Inoue A, Egami N, et al. Diagnostic usefulness of multifrequency tympanometry for Meniere's disease. Audiol Neurootol, 2013; 18: 152–60.
31. Kobayashi M, Yoshida T, Sugimoto S, et al. Effects of endolymphatic hydrops on acoustic energy absorbance. Acta Otolaryngol, 2020; 140(8): 626–31.
32. Karuppannan A, Barman A. Evaluation of wideband absorbance in adults with abnormal positive and negative middle ear pressure. J Hear Sci, 2020; 10(4): 40–7.
33. Shaver MD, Sun X-M. Wideband energy reflectance measurements: effects of negative middle ear pressure and application of a pressure compensation procedure. J Acoust Soc Am, 2013; 134: 332–41.
34. Sadé J, Halevy A, Hadas E. Clearance of middle ear effusions and middle ear pressures. Ann Otol Rhinol Laryngol, 1976; 85 (Suppl 2): 58–62.