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**An exploratory multi-method investigation of middle ear muscle contraction**

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**ABSTRACT**

It has been suggested that tensor tympani muscle (TTM) contraction may be involved in the development of ear-related pathologies such as tinnitus, hyperacusis and otalgia, called the tonic tensor tympani syndrome (TTTS). However, as there is no precise measure of TTM function under normal and pathological states, its involvement remains speculative. When the TTM or the stapedius muscle (SM) contracts, they both generate an increase of middle ear stiffness that can be measured through middle ear admittance. However, this technique cannot differentiate the contraction between the two muscles. On the other hand, the air pressure measured in a sealed external auditory canal can provide a measure of the eardrum displacement that may be able to differentiate SM from TTM contraction. TTM is attached to the malleus, and its contraction causes a retraction of the eardrum inside the middle ear cavity, while SM can have a small but reversed effect on TTM displacement. To investigate this issue, we compared the middle ear admittance and air pressure in a sealed external ear canal upon auditory stimulation (sMEMC) and voluntary middle ear muscle contraction (vMEMC). In addition, we assessed the perceptual effect of vMEMC, including pitch and loudness matching of the fluttering noise produced by vMEMC and the hearing threshold shifts, were measured. Out of the 14 ears tested, sMEMC was associated with a decrease of admittance in 93% (mean peak average: -0.06 ml, SD:0.04) and an increase of air pressure in 29% of ears (mean peak average: 8.1 Pa, SD:5.1). No decrease in air pressure was found upon sMEMC. For vMEMC (n=8 ears), decreases were found for both admittance and air pressure in 100% and 88%, with a mean peak average of -0.38 ml, SD: 0.54 and -149 Pa, SD:156, for admittance and pressure respectively. These results suggest that SM and TTM are involved in sMEMC and vMEMC, respectively. In addition, vMEMC was associated with perceptual effects including a low-frequency sound, pitch-matched at ~30 Hz (>15 dB SL), and a low-frequency hearing loss of at least 10 dB between 20-200 Hz. In conclusion, admittance and air pressure recordings provide useful and complementary information on middle ear muscle contraction and can be used to explore the middle ear function.

Keywords: Tensor Tympani, Stapedius Muscle, Tensor Tympani Syndrome, Middle ear, Acoustic shock, Hyperacusis, Otagia

## INTRODUCTION

The human ear is a complex organ composed of different structures regrouped in three main parts: the outer ear, the middle ear, and the inner ear. The middle ear's main function is to improve the transmission of the sound to the cochlea by compensating for the impedance change from air to liquid (Møller, 1963). Within the middle ear lies the two smallest skeletal muscles of the human body: the stapedius (SM) and the tensor tympani (TTM) (Mansour et al., 2013). The former is, on average, only 5.8 mm in length, and the latter is around 19.7 mm (Aristeguieta et al., 2010). The stapedius muscle is attached from the middle ear's posterior wall up to the stapes' head, where it is fixed by its tendon (Loeb, 1964; Mansour et al., 2013). The tensor tympani runs from the Eustachian tube's cartilaginous portion to the malleus' handle (Loeb, 1964; Mansour et al., 2013). Interestingly, they are not innervated by the same cranial nerve: the tensor tympani is innervated by the trigeminal nerve and the stapedius muscle by the facial nerve (Loeb, 1964; Mansour et al., 2013). When a stimulus, either a loud sound or else, generates contractions of those muscles, their actions limit the ossicular chain vibrations and thus reduce the level of the sound transmitted to the cochlea. Studies that have activated the TTM and SM independently have shown that they both generate low-frequency attenuation, but this attenuation seems to be more pronounced for the SM than the TTM in both humans and cats (Neergaard et al. 1962; Teig 1973, respectively). However, it has been found that TTM caused a greater change of acoustic compliance when pulling on the TTM compared to SM on humans (Aron et al., 2015) suggesting a greater reduction of sound transmission for the TTM. The source of this discrepancy is unknown but warrants further research. The SM also attenuates high frequencies, although the attenuation is less than for the low frequencies (Neergaard et al., 1962). Besides, the simultaneous pull of these two muscles was associated with almost the same results as the equal sum of the two attenuations obtained independently (Neergaard et al., 1962), but a more complex relationship has also been reported (Teig, 1973). However, there has been some evidence that a lower level of contraction leading to a limited

amount of tension can increase sound transmission in the medium frequency range (Teig, 1973; Wever & Vernon, 1956; Wiggers, 1937). The TTM pulls the umbo inwards by about 100 microns resulting in a retraction of the eardrum into the middle ear cavity (Hüttenbrink, 1989; Loeb, 1964; Møller, 1964). Additionally, the TTM contraction pushes the stapes inside the vestibular canal by around 10 microns because of the malleus-incus joint's gliding motion. Interestingly, the SM's pull on the stapes is antagonistic to the TTM: it pulls the stapes backward; the anterior border of the footplate moves outwards and the posterior border inwards (Mansour et al., 2013). Consequently, SM contraction may produce in some individuals an eardrum extrusion, a movement towards the external ear canal cavity (Lidén et al., 1970; Loeb, 1964; Marchbanks, 1984, 1984; Møller, 1964; Phillipps et al., 1989). However, it has also been reported that SM contraction has only an effect on the stapes and not on the other ossicles (Pang and Peake 1986), thus questioning movements of the eardrum by SM contraction. In addition, the movement of the eardrum following stapedial muscle contraction is cochlear pressure-dependent in humans: the displacement is outward when the cochlear pressure is low and inward when the cochlear pressure is high (Marchbanks, 2002). This may explain conflicting results of tympanic movement caused by the SM contraction.

There is no scientific consensus regarding the respective function of the SM and the TTM. As the SM contracts to sound, it has been proposed to play an important role in the protection of the ear against loud noises (Møller, 1984). It also contracts to vocalization (Møller, 1984). The TTM is activated by face stroking, air puffs against the eyes, electrical stimulation of the tongue, swallowing, head movements and cutaneous stimulation (Salomon & Starr, 1963). TTM does not usually contract in response to sounds (Jones et al., 2008). When it does, the contraction seems to be mostly phasic (tonic contraction lasts less than 1 sec) (Salomon and Starr, 1963; Djupesland, 1965; Lidén et al., 1970; the present study), highly fatigable (the present study) and with relatively long latency (100-200 msec). These properties are not consistent with a protective role of the inner ear from sudden and prolonged loud sounds. On the other hand, it has been shown that the TTM contraction is caused by sound stimulation when a startle reflex is also triggered. Considering that the startle reflex plays a role in the

protection of vulnerable parts of the body (Yeomans et al., 2002), it can be speculated that the TTM may protect the integrity of the middle ear, such as the protection of the ossicular chain from head trauma. This putative TTM function does not exclude other roles it may have, such as improving middle ear aeration for instance (Kierner et al., 2002).

Dysfunction and/or defect of any of the two middle ear muscles, but most notably the TTM, has long been proposed to be responsible, or at least involved, in developing multiple auditory pathologies such as progressive deafness, tinnitus, and vertigo (Goodfriend, 1947). As an example, it was postulated that malocclusion could irritate the trigeminal nerve and disturb the sensory distribution within the fifth cranial nerve enough that it could lead to sporadic reflex contractions of the TTM and even maintain its contraction, causing low-frequency hearing loss and tinnitus (Goodfriend, 1947; Seaver, 1937). Moreover, the involuntary myoclonic activity of the middle ear muscles are responsible for some rare form of objective tinnitus usually described as loud audible clicks (Abdul-Baqi, 2004; Badia et al., 1994; Bance, 2019; Brigo et al., 2013; Dang & Carol Liu, 2019; Ellenstein et al., 2013; Freund, 1956; Hornibrook & Keast, 2019; Howsam et al., 2005; Kim et al., 2013; Kwee & Struben, 1972; Lee et al., 2012; Oliveira et al., 2003; Phillipps et al., 1989; Rajah, 1992; Rock, 1995; Salehi et al., 2019; Watanabe et al., 1974; Zipfel et al., 2000). In particular cases, the middle ear muscle contractions (MEMC), and the objective clicks, are only triggered by forceful eyelid closure, a phenomenon that has been called *Forceful Eyelid Closure Syndrome (FECS)* (Kim et al., 2013; Lee et al., 2012; Rajah, 1992; Rock, 1995). A similar phenomenon has recently been described following an acoustic shock injury (ASI) (Londero et al., 2017). After being exposed to a firearm detonation that mostly affected the right ear, a patient reported chronic symptoms, both within and outside the ears (tinnitus, otalgia, tingling-tension in the ear, red tympanum, blocked nose, pain in the neck-temporal region). Follow-up examination revealed that non-forceful eyelid closure generated eardrum movements in the most symptomatic ear only (Londero et al., 2017). The patient reported that he had never experienced that phenomenon before the acoustic shock. The myoclonic activity of the stapedius muscle has also been reported in a 20-year-old soldier exposed to intense

artillery noise during a fierce battle and immediately afterward complained of bilateral tinnitus. In this case, the eardrum's rhythmic movement was visible during the microscopic examination, and all the symptoms disappeared after the stapedius muscle tenotomy (Golz et al., 2003). Overall, there is some evidence that abnormal middle ear muscle activity can be triggered after acoustic trauma and that they may be associated with a cluster of symptoms.

Regarding acoustic shock injuries (ASI), it has been previously suggested that the TTM could play a central role in the onset of symptoms (e.g., tinnitus, otalgia, hyperacusis, ear fullness, dizziness, vertigo) that follows an acoustic incident defined as a loud, brief and unexpected sound, but non-necessarily damaging for the cochlea (Westcott, 2006, 2016; Westcott et al., 2013). As early as the 1970s, Klockhoff (1979) blamed the tensor tympani's spontaneous tonic muscle activity for the emergence of these symptoms. He was able to assess spontaneous TTM contractions in more than 250 patients by using an impedance bridge; the phenomenon was called "the tonic tensor phenomenon" and the associated symptoms, the "tonic tensor tympani syndrome" (TTTS). Still, the mechanisms underlying the large variety of symptoms observed after acoustic shocks remain elusive and the ability of the TTM to sustain a contraction has not been demonstrated. A model has been recently elaborated on the hypothesis that the tensor tympani, the trigeminal nerve (TGN), and the trigeminal cervical complex (TCC) could play a role in generating these symptoms (Noreña et al., 2018). The authors suggested that TTM overuse (due to the acoustic shock), TTM overload (due to muscle tension), and TTM injury (due to hypoxia and "energy crisis") could lead to inflammation, thereby activating the TGN, the TCC, and the cortex. The activation of these centers could thus generate a wide variety of symptoms experienced after an ASI (Noreña et al., 2018). This speculative model, partly developed from an acoustic shock injury (ASI) case study in which the patient suffered from multiple symptoms and displayed signs of tympanum inflammation and middle ear muscle dysfunction (Londero et al., 2017), is awaiting further confirmation. New innovative tests to assess the integrity of the TTM and its normal/abnormal functioning are crucially needed to test and refine this hypothesis. As mentioned by previous authors (Aron et al., 2015): « without

definitive markers for TTM contraction, it becomes a matter of "faith" to assign many otologic pathologies to the TTM ».

Currently, the most used technique to assess the integrity of MEM is admittance measurements. In brief, this technique consists in presenting a sound in the sealed auditory ear canal and measuring the quantity of sound energy that is reflected when a MEM is contracted. This measure is an indicator of the stiffness of the middle ear: the muscle contraction will generate an increase of middle ear stiffness, modifying the quantity of sound energy reflected (vs .transmitted). Since both the TTM and the SM increases ME stiffness, this technique does not differentiate between the contractions of these two muscles. It has been shown that air pressure measured within a sealed auditory ear canal is more suited to differentiate these two muscles than admittance measurements because it assesses the movement of the eardrum upon a contraction rather than the stiffness of the ME. Møeller (1964) found in rabbits that TTM contraction always produced a decrease in air pressure while SM contraction produced either no change or a considerably smaller increase or decrease of air pressure. He also concluded that an increase in air pressure could only be caused by SM contraction. Similar results were found by Lidén and collaborators (1970) in humans: in a sample of 78 normal hearing subjects, they found that 51% of participants showed increased air pressure deflection upon acoustic stimulation, 31% present a broad and shallow decrease and 13% present abrupt and phasic decrease in air pressure. The authors interpreted this last finding as a sign of TTM contraction and thus concluded that, in some individuals, a loud sound ( $\geq 100$  dB HL) could trigger TTM contraction. As TTM usually does not contract to sound except startling sound (Jones et al., 2008) it is plausible that the loud sound stimulation triggered the startle reflex for these individuals. Other methods to trigger TTM contraction should be used in research. Using a similar approach, Robert Marchbanks developed a device called the Tympanic-Membrane Displacement system (TMD) (Marchbanks, 1984). He found that SM contraction could be associated with inward, outward or inward/outward movement of the eardrum (Marchbanks, 1984) and that the configuration could be mediated by changing the posture from sitting to supine (Marchbanks, 2002). Philipps and collaborators used the TMD to assess the presence of objective tinnitus described as spontaneous clicks in the right ear of a 10-year-old girl.



They found increased air pressure upon auditory stimulation and decrease air pressure upon the click symptoms. They concluded that SM was unlikely to be the source of the click symptoms since the tympanic movement displacement associated with the sound stimulation was different from the ones associated with the click symptoms (Phillipps et al., 1989). Despite the diagnostic potential of the air pressure technique to investigate MEMC in animals and humans, no studies have yet replicated and extended these findings. One of the goals of the current paper was to define the specific characteristic of SM and TTM contraction through admittance and air pressure measurements.

The first objective of the current study was to define SM contraction admittance and air pressure hallmarks by using auditory stimulation. The TTM is known to be non-responsive to sound for the sound level used here (Jones et al., 2008) and thus the measured modifications will be only attributed to SM. The expected modifications upon stimulation were decreased admittance and increased or null air pressure change. The second objective of the present study was to define TTM admittance and air pressure hallmarks in participants who can voluntarily contract their middle ear muscles. Most studies suggested that TTM was one of the muscles under voluntary control (Aron et al., 2015; Bance et al., 2013; Bance, 2019; Lidén et al., 1970; Wickens et al., 2017) but some others reported cases of SM voluntary contraction alone (Burns et al., 1993; A. R. Møller, 1961). The hypothesis is that if the TTM is indeed involved in vMEMC, the admittance and air pressure results upon contraction should be different from the ones found following sMEMC. Considering that the TTM is anatomically connected to the malleus handle, a contraction of this muscle would lead to a retraction of the eardrum and negative pressure. We hypothesize that TTM contraction upon vMEMC would result in decreased admittance and air pressure.

*In fine*, the final goal was to build a sensitive assessment battery of middle ear muscle function for patients reporting the symptoms listed above (Pollard, 2019a, 2019b).

## METHODS

### 1.0 Participants:

A total of 18 ears from 12 participants (Mean age: 28 y.o., F/M: 6/6) were tested in this study: four participants (Mean age: 31 y.o., F/M: 2/2) were able to voluntarily contract their middle ear muscles bilaterally (vMEMC, n=8 ears) and eight participants could not (Ctrl, n= 12 ears). The perceptual effect of vMEMC, including pitch and loudness matching of the fluttering noise produced by vMEMC and the hearing threshold shifts, were assessed on three out of the four participants with vMEMC ability: participant 09 (male, 36 y.o, Ears #11, #12), participant 10 (female, 25 y.o., Ears #13, #14) and participant 11 (female, 28 y.o., Ears #15, #16).

The characteristics of all the ears tested are displayed in Table I. The static tympanometry results revealed that most ears had normal movements of the eardrum-ossicular chain complex displaying type A tympanogram (n=11), three had reduced mobility displaying type AS tympanogram (Ear #7, #8, #13), and two had highly compliant middle ear system displaying AD tympanogram (Ear #15, #16) (Jerger, 1970). For two of the ears, static tympanometry was not performed (Ear #17, #18). In addition, in most ears (n=15), ipsilateral stapedial reflex thresholds were obtained. In one ear, it was not possible to obtain a threshold for any of the tested sound frequencies due to the high compliance of the middle ear system in this ear generating too many artifacts (Ear #16). For two of the ears, the test was not performed (Ear #17, #18).

Finally, all ears displayed normal hearing thresholds ( $\leq 20$  dB HL) between 250 Hz to 8 kHz. Every participant that was able to produce a vMEMC (they were not aware of what was going on) reported that it was associated with a kinesthetic or physical sensation of "their eardrum" moving in their ears associated with a low-frequency fluttering sound.

The vMEMC conditions were tested over several days. The testing time was adapted for each participant with his ability: if they reported fatigue within a testing session, the test was stopped and then resumed the following day. All participants reported fatigue after one or several hours of testing involving repeated episodes of vMEMC. The "Comité de Protection des Personnes Nord Ouest IV" review board approved this research. Reference number: 2018 A01183 52.

## 2. Materials:

### 2.1 Screening tests

#### 2.1.1 *Audiometry*

Hearing thresholds (HT) were assessed monaurally, for both ears, by presenting pure tones from 0.25 to 8 kHz by half-octave steps using the conventional clinical procedures with Sennheiser HDA-300 connected to an Astera audiometer (Natus Medical; formerly Otometrics, Denmark) (Harrell, 2002). The test was performed in a soundproof room.

#### 2.1.2 *Tympanometry*

All the tympanometry measurements were recorded in a soundproof room or a quiet room using the Madsen Zodiac tympanometer (Natus Medical, formerly Otometrics, Denmark). A probe was inserted in the external ear until an airtight seal was achieved. For static tympanometry, the pressure scale was set from -400 to 200 daPa, and the sound probe was set at 226 Hz. The patient is asked not to move during the measure. The pressure is modified quickly (200 daPa per sec) from positive to negative pressure, and the admittance of the 226 Hz probe tone is recorded.

#### 2.1.3 *Stapedial reflex threshold*

Stapedial reflex thresholds were tested using tonal stimuli of 0.5, 1, 2, and 4 kHz and broadband noise of 2.4 seconds' duration in ipsilateral conditions for both ears (5.5 ms fall/rise time) using the Madsen Zodiac tympanometer (Natus Medical, formerly Otometrics, Denmark). The presentation level started at 60 dB HL and increased by 5 dB steps until a .02 ml change in admittance was automatically detected twice at the same level.

#### 2.2.1 *Admittance measurements*

The Madsen Zodiac's Y-recorder option was used for continuous admittance recordings with the frequency probe set at 226 Hz for most conditions and some specific conditions at 1000 Hz. This feature allows for continuous recording of admittance change over a duration of 30 seconds. Recordings were made under two different conditions of

external ear canal air pressure: 0 and -50 daPa. This modification in auditory ear canal air pressure had been made available to improve the acoustic stapedius reflex threshold measurements in ears with high static acoustic admittance: in those cases, stapedius reflex thresholds are detected at lower stimulation level with -50 daPa pressure compared to normal pressure (0 daPa) (Atcherson & Stoody, 2011; DiGiovanni & Ries, 2007). In the present study, the pressure was varied to add an argument to differentiate SM contraction from TTM contraction (Aron et al., 2015). The Y-recorder feature was used to assess the admittance of one ME continuously while presenting a sound ipsilaterally or contralaterally (sMEMC). It was also used to assess admittance modification of one ME during each voluntary MEM contraction.

### *2.2.2 Air pressure captor*

A device was developed to measure the air pressure inside the airtight sealed external ear canal. The device was calibrated using an artificial external ear cavity. This cavity was an open cylinder made out of a plastic syringe of 25 mm in length and 8 mm in diameter, mimicking an average human external ear canal (Hammershøi & Møller, 1996). As shown in Figure 1, the two sides were plugged with a tympanometry tip: one side was connected to a pressure pump (Madsen Zodiac Tympanometer, Natus Medical), and the other side was connected to the air pressure captor. The pressure sensor is fully calibrated, and temperature compensated for sensor offset, sensitivity and temperature effects. The pressure range measured by the sensor should be  $\pm 498.16$  Pascal (Pa) and the noise floor is below 5 Pa. The pressure pump was used to precisely change the air pressure in the cavity from -600 to 600 Pa (slightly above the “regular” pressure range) by 50 Pa steps and record the output with the air pressure captor. As displayed in Figure 1, the pressure input (pressure pump) and output (pressure captor) were linearly correlated, suggesting that the pressure captor is sensitive to pressure change and provides a precise measure of air pressure. If sensitive enough to detect SM and TTM contractions, the pressure recording offers several advantages over the admittance recording: 1) it does not require the presentation of a sound (which is convenient for severe hyperacusis patients) and 2) it can be obtained simultaneously in both ears. In participants, the external ear canal was airtight sealed using an admittance ear tip

connected to a pressure sensor by a plastic tube of 10 cm in length. The sensor was connected to an Arduino that was connected to a computer. The MegunoLink software (Number Eight Innovation Limited, New Zealand) allowed visualization and data recording.

## 2.2 Experimental tests

The first objective of the study was to compare ME admittance and air pressure modifications following auditory stimulus-evoked middle ear muscle contraction (sMEMC) in all ears. As auditory sMEMC is associated with SM contraction (Møller, 1984), this allows us to validate the presence of SM contractions through admittance modifications and compare those responses to air pressure changes. Following an auditory stimulation, a decrease of admittance and either, an increase or no effect on air pressure was expected. The second objective was to compare ME admittance and air pressure modification during vMEMC. As this type of contraction is most likely associated with TTM contraction (Aron et al., 2015), this allows us to explore admittance and air pressure modifications associated with TTM contraction: a decrease for both admittance and pressure were expected.

### *2.2.3 Middle ear measurements*

The acoustic stimulation was presented through Sennheiser HD-600 supra-auricular headphones connected to a Sound Blaster X-fi HD model SB1240 sound card. A MATLAB program created in-house allowed to manually control all the stimulus parameters such as the duration, the intensity level, the center frequency, and the bandwidth of any sound presented (Fournier et al., 2018). For 14 of the 18 ears, a two-second white noise was presented four times (ISI= 2s) at 90 dB SPL in one ear while measuring admittance in the contralateral ear. This was performed twice for a total of eight sMEMC responses per ear: the responses were reliable and stable. The mean average response was calculated from the eight responses for both admittance and air pressure. White noise was chosen because it is the stimulus eliciting the stapedial reflex at the lowest intensity level in all ears. The level was set at 90 dB SPL as it is loud

enough to elicit a clear sMEMC in all ears and is still acceptable for participants (not too loud).

In addition, in two ears of one participant (#9), the reflex response growth was measured using a burst of white noise (1.2 s duration) presented in one ear and increasing by 3 dB steps from 66 to 99 dB SPL while recording either admittance or pressure in the contralateral ear. For three participants with the ability to voluntarily contract the MEM, additional stimulation was used to validate the presence of sMEMC in both of their ears. In their case, a 1000 Hz tone at 100 dB SPL was presented continuously in a pulse sequence with 1.2s stimulation duration and 2s gaps of silence (0.5s fall/rise time) in one ear while the air pressure or admittance change was measured in the other ear. The mean average response was calculated from the recordings for both admittance and air pressure. For the assessment of voluntary middle ear muscle contraction, we simply asked the participants to voluntarily contract their muscles every 10 seconds for a minute while continuously measuring admittance or air pressure in their ears.

The reflex decay test allowed for continuous admittance recording for 12.5s. After 1s of silence, a loud 500 or 1000 Hz pure tone is presented either ipsilaterally (same side of the recording) or contralaterally for 10s. Typically, a change in the admittance is seen after the beginning of the sound presentation and maintained during the stimulation (Hall, 1977) and then returns to normal after the sound presentation (within 1.5s). This change in the admittance is known to be due to SM contraction (Hall, 1977). A baseline measure was taken: the participant was asked to stay quiet during the measurement and refrained from moving and swallowing during the recording 12.5 sec. The Madsen Zodiac's reflex decay feature allows modification of the air pressure inside the external ear canal by +/- 50 daPa. Recordings were made under two different pressure conditions: 0 and -50 daPa, to differentiate TTM from SM contractions. It has been previously shown that SM and TTM contraction causes a decrease in admittance when the recording is performed at normal pressure (0 daPa) (Aron et al., 2015). However, when the same measurements are performed at negative pressure (-50 daPa), the admittance is increased when TTM contraction is present and decreased for SM contraction alone (Aron et al., 2015). In the vMEMC condition, the participants were asked to contract their MEM every 3s after the beginning of the sound presentation.

### 2.3 Hearing Thresholds Shift During vMEMC

The perceptual effect of vMEMC was measured on three participants with the ability to voluntarily contract their MEM: participants 09, 10 & 11. The measurements were performed using Sennheiser HD-600 supra-auricular headphones connected to a Sound Blaster X-fi HD model SB1240 sound card. The same MATLAB program created in-house, previously described in the "*air pressure captor*" section, was used here. For air conduction thresholds (ACT), pure tones ranging from 22 Hz up to 8 kHz by  $\frac{1}{4}$  octave steps were presented in a "5s stimulation – 1s of silence" sequence that was continuously running (0.1-sec rise/fall time). The 5s stimulus presentation allowed enough time to produce a vMEMC and measure its effect. For each frequency, the hearing threshold was first measured without vMEMC: the sound was first presented at a moderate level and decreased by 2 dB steps until the sound was not heard and then increased by 1 dB steps until it was just audible again. This procedure was repeated at least twice per frequency to obtain a stable threshold response. For measuring the hearing threshold under the vMEMC condition, the sound was also first presented at a moderate audible level. The participant contracted his MEM during the 5s stimulation: if the sound was still perceived during the contraction, the sound intensity was decreased by 2 dB. The procedure was repeated until the sound was not heard during the vMEMC, then the intensity was raised by 1 dB until the sound was audible. This procedure was repeated at least twice per frequency to obtain a stable threshold response. The same technique was applied for bone conduction thresholds: the bone oscillator (Natus Medical, formerly Otometrics, Denmark) was plugged into the Sound Blaster X-fi HD model SB1240 sound card and placed on the mastoid of each ear of the participant, this system was thus not calibrated in dB HL. The bone conduction thresholds (BCT) for the low frequencies, including 125, 176 and 250 Hz, were tested in both ears.

### 2.4 Psychoacoustic Characterization of the vMEMC Fluttering Sound

A pitch and loudness matching task was used to characterize the fluttering sound heard during vMEMC in each ear of participants 09, 10 and 11. The same Matlab program and equipment used to measure the hearing threshold shifts were used to

characterize the fluttering sound. Since vMEMC sounds were heard differently in each ear for most participants, the psychoacoustic measures were performed ipsilaterally: the vMEMC sound heard in the LE was measured with sound stimulation in the LE, and the sound heard in the RE with sound stimulation in the RE. Pure tones ranging from 10 Hz up to 1000 Hz were used and presented in a "5s stimulation – 5s of silence" sequence that was continuously running (0.1-sec rise/fall time). The sound was first presented at an audible level, and the participant changed the frequency of the presented sound until it was almost like the sound emitted during vMEMC. The participants could contract their MEM during the 5s silence intervals to compare the fluttering sound's timbre to the sound presented during the 5s stimulation. After a proper match, the participant measured the hearing threshold for the frequency obtained in the pitch-matching task using a conventional stepwise method (-3 dB when the sound is heard, and + 1 dB when it is inaudible). Finally, the loudness of the fluttering sound was estimated by increasing the sound level by 1 dB step from the threshold measured at the pitch matching frequency until the target tone's sound level was judged similar to the sound produced by vMEMC. The participant could again contract their MEM during the 5s silence intervals and compare the sound level of the fluttering sound emitted to the sound level of the one presented in the earphone.

### 2.5 Video recording

Microscopic video recordings of vMEMC were made by an ENT at the ORL and Skull Base Surgery Department of the Clairval Hospital in Marseille (France). The videos were recorded using the otoendoscopy and digital recording system (Wolf, Germany).

## **RESULTS**

### *1. sMEMC: admittance and air pressure results*

Admittance and air pressure responses upon a contralateral white noise set at 90 dB SPL and measured in 14 ears are presented in Figures 2A and 2B, respectively. The sound onset is at 0 seconds on the figure. For admittance, a decrease of at least two standard deviations under the baseline was seen in 13 out of the 14 ears tested (93%) with



a mean peak average: -0.06 ml (SD:0.04). For the air pressure, however, an increase of pressure of at least two standard deviations above the baseline following the onset of the sound stimulation was observed in only 4 out of 14 ears, representing only 29% of all the ears tested. The mean peak average for the responses in these four ears is 8.1 Pa (SD:5.1). These results suggest that SM contraction in a normal functioning ear almost always causes an increase of ME stiffness reflected by a decrease in admittance for a 226 Hz tone. On the other hand, SM contraction causes eardrum movement in about a third of the ears.

To further validate that the positive pressure was indeed caused by SM contraction, two ears displaying positive pressure with the 90 dB SPL white noise were tested for response growth to a gradual increase in sound levels. Admittance (226 Hz probe tone) and air pressure growth responses following the increase in intensity levels from 66 to 99 dB SPL by 3 dB steps of a contralateral white noise are presented in Figure 3B and 3A, respectively. Similar results are presented in Figures 4A and 4B, with the sole exception that admittance was measured with a 1 kHz probe tone instead of the standard 226 Hz. For both ears, the maximum air pressure increase with the gradual rise in sound levels. Similarly, the absolute value of the peak of the admittance variation increases for the 226 Hz and 1kHz probe tones with the rise of sound level. In addition, the air pressure and the admittance modifications were highly correlated.

## 2. *vMEMC vs. sMEMC: admittance and air pressure results*

The mean average response of air pressure and admittance (226 Hz, 0 and -50 daPa) during vMEMC and sMEMC are presented in Figure 5 left and right column, respectively. For eight of the ears with the ability to contract voluntarily their MEM, all of them displayed, upon voluntary contraction, decrease admittance deflections at 0 daPa (100%), with a mean peak average of -0.38 ml (SD: 0.54). When the pressure was set at -50 daPa, 4 out of seven ears (57%) displayed increased spike-like admittance deflections two out of seven displayed bi-phasic (decrease-increased) admittance deflection (29%) and one displayed a decrease of admittance (14%). For one ear, it was not possible to change the pressure within the external ear cavity and thus to measure the admittance under -50 daPa condition. For the measure of air pressure, 7 out of 8 ears (88%)

displayed decreased pressure spike-like deflection upon vMEMC while one ear displayed increased air pressure with a mean peak average of -149 Pa (SD:15) for the ears displaying decreased air pressure. The duration of the vMEMC responses of around 0.5s is similar across the different ears. Interestingly, the ear displaying vMEMC increased air pressure deflection was the same ear displaying decreased admittance deflection for both 0 and -50 daPa. More results on the participant displaying voluntary SM contraction in his LE and voluntary MEMC involving the TTM on the RE are provided in the supplementary material (Figure 1).

The results in some of the same ears following contralateral stimulation are presented in Figure 5 right column. As expected for SM contraction, decreased admittance was seen for all the ears tested at 0 and -50 daPa. Contrary to vMEMC responses, the change in pressure did not cause a reversal of admittance for sMEMC responses, from decreased to increased admittance. In addition, air pressure responses to sMEMC were either increased air pressure deflections or no effect.

As shown in Figure 6, no correlation was found between admittance and air pressure for both vMEMC and sMEMC. These results suggest that the stiffness of the ME, as measured by admittance, and the movement of the eardrum, as measured by air pressure, are two distinct phenomena. From this figure, it is apparent that admittance change with vMEMC is of a larger magnitude than sMEMC. This is in agreement with the results of a previous study on vMEMC (Aron et al., 2015). It is also very clear that the changes in air pressure with vMEMC are much larger than with sMEMC.

### *3. Three detailed cases of vMEMC*

#### *3.1 Standard measurements and sMEMC*

Static tympanometry and sMEMC results for three participants with the ability to voluntarily contract their MEM are presented in Figure 7. Static tympanometry was used to assess the tympano-ossicular chain's integrity in both ears of the participants as the tympano-ossicular chain's abnormal integrity could influence some of the measurements. The admittance recordings of SM reflexes to brief and sustained sounds (reflex decay) were used to validate the presence of SM reflexes and compare SM contraction admittance with air pressure results. Figure 7 top panel shows participants' RE (red) and

LE (blue) static admittance results. For participant 09, both ears display Jerger type A tympanograms with normal pressure and compliance, suggesting a normal middle ear function bilaterally (Fowler & Shanks, 2002). Participant 10 displayed a Jerger type A tympanogram for the LE and type AS tympanogram for the RE. Participant 11, however, displayed high static acoustic admittance for both ears (Jerger type Ad). Type Ad tympanograms are typically recorded from ME having ossicular disarticulation or thin membranes having no middle or fibrous layer (Fowler & Shanks, 2002; Holt et al., 1982). This heightened peak of admittance was most prominent in the LE, and this limited the testing on this ear. Stapedial muscle contraction was triggered by repetitive short, loud stimulation (1.1 s, 100 dB SPL, 1 kHz) in the contralateral ear and measured alternatively by the pressure captor and the admittance probe in the other ear. Figure 7 middle panel shows the air pressure and admittance modifications measured after each of the five loud sound presentations. Both ears of participant 09 show a clear increase of air pressure after each sound presentation. The pressure modifications following the stimulation are replicable and clearly above the baseline: it is on an average of 25 Pa and 33 Pa for the LE and RE, respectively. As expected, a brief sound stimulation is also causing a replicable reduction of admittance in both ears: around -0.05 and -0.1 ml for the RE and LE, respectively. Similar results were obtained in Participant 10 LE. For her RE, there was no modification of pressure following the onset of the stimulation. However, there was a clear change in admittance in both ears. For participant 11 RE and LE, there was no modification in air pressure caused by brief auditory stimulation. On the other hand, the admittance was strongly reduced in the right ear (it was not possible to do the test in the left ear since the high static admittance generated too much noise). Altogether, these results suggest that SM contraction is associated with a reduction in admittance but not necessarily with a change in air pressure (or eardrum movement). Figure 7 bottom panel shows the modification of admittance and air pressure following sustained loud acoustic stimulation for all the participants (1 kHz, 100 dB HL, 10s, reflex decay test of the Madsen Zodiac). At rest, the admittance is close to 0 ml for the first second, and then, the admittance drops (when the sound is presented) and stays negative for 10 sec before coming back to 0 ml around the 11<sup>th</sup> second. For the reflex decay test, as expected, all ears' admittance became negative as soon as the sound was presented, and this negativity

was maintained during the sound stimulation. This change in admittance during the sound presentation is associated with SM contraction (Hall, 1977). In terms of air pressure, a sawtooth pattern can be observed in the three subjects. This periodic fluctuation at a rate of 1-2 Hz reflects the effects of heartbeats on the tympanic membrane movements. Also, the results following sustained stimulation yielded results like the one obtained for the short stimulation, that is, visible increase pressure for participant 09 LE and RE and participant 10 LE only. In these three ears, there was an increase of air pressure with the beginning of the sound, and this positive pressure is sustained during the sound presentation. However, for participant 10 RE and participant 11 LE and RE, no sustained change of pressure was seen during the prolonged stimulation. In sum, the results obtained in participants 09 LE and RE and participant 10 LE are consistent for all the measurements: brief and sustained loud auditory stimulation resulted in reduced admittance and increased positive air pressure. These results suggest that SM contractions can be assessed by air pressure only in some ears. However, as seen in participant 10 RE and participant 11 RE and LE, the lack of increased pressure cannot be interpreted as an absence of SM reflexes but as an absence of tympanic movement during SM contraction.

### *3.2. vMEMC assessments*

vMEMC were assessed using the same methods previously validated with sMEMC. These measurements were performed under two conditions: vMEMC alone (Figure 8) and vMEMC with sound stimulation (Figure 9). This last condition was chosen to disentangle SM from TTM contraction in the same recording as one would be triggered by sound (SM) and the other by the participant will (presumably involving TTM). To further disentangle SM from TTM contraction, admittance recording was performed under different auditory external canal pressure: 0 and -50 daPa. As mentioned earlier, the admittance decrease upon a TTM contraction for pressure set at 0 daPa but increases when pressure is set at -50 daPa (Aron et al., 2015). Figure 8 shows the change in pressure and admittance following vMEMC alone. For the air pressure recordings, vMEMC is associated with a spike-like decrease of pressure for all ears except participant 09 LE. These results suggest that, in all five ears, TTM contraction is present during vMEMC. The admittance recording under different pressure further supports this

hypothesis: vMEMC is associated with a spike-like decrease of admittance at 0 daPa pressure (dark red or blue line) and with a spike-like increase of admittance at -50 daPa pressure (light red or light blue line). Those results further support that TTM is contracted in those four ears. Interestingly, participant 09 LE does not show the same pattern: the pressure peak observed for this ear during each vMEMC is positive, suggesting that the muscle voluntarily contracted is the SM. The vMEMC admittance recordings further support this hypothesis: there is no inversion of the admittance negative spike-like peak at -50 daPa. Participant 10 also displays interesting admittance results when the external ear canal pressure is set at -50 daPa: there is a biphasic response with a decrease of admittance followed by an increase of admittance. This result suggests that both the SM and the TTM are contracted in this participant upon vMEMC: SM is first contracted followed by TTM.

Figure 9 shows the change in pressure and admittance following vMEMC during sustained auditory stimulation in the contralateral ear (reflex decay). vMEMC is associated, again, with a clear decrease of air pressure for all ears except participant 09 LE. The RE of participant 09 displays both a sustained increase of air pressure that is maintained during the sound stimulation and a rapid spike-like decrease of air pressure due to vMEMC, suggestive of SM and TTM contraction, respectively. For the admittance, very large phasic peaks of decreased admittance are superimposed on the stable reduction of admittance caused by the sound presentation for participants 09 and 11 RE and both ears of participant 10. These phasic peaks of decreased admittance are reverted (increased admittance) when the pressure is set at -50 daPa. However, the stable reduction in admittance caused by the sound presentation is constant despite the change in external ear canal pressure. These results suggest that the TTM is voluntarily contracted in participants 09 & 11 RE, as it is the only change in admittance affected by the difference in pressure. These changes are somewhat less obvious for participant 10 ears who displays biphasic decrease and increase of admittance upon vMEMC. These results suggest again that both MEMs are contracted in this case. To note, the change in admittance caused by the TTM is at least two times bigger than the one caused by SM. Participant 09 RE was the only ear displaying the contrast in pressure between the SM (a sustained increase of pressure) and the TTM (a sudden decrease of pressure). In his case,

the change of pressure caused by the TTM is at least ten times bigger than the one caused by SM. Finally, participant 09 LE showed increased phasic pressure peaks associated with vMEMC superimposed on the sustained increased pressure associated with the continuous auditory stimulation. Similar results were observed for the admittance recordings: the sudden decreases of admittance associated with vMEMC were superimposed on the already sustained reduction of admittance triggered by the constant stimulation. The modification of admittance caused by vMEMC was not affected by the change of external ear canal pressure (-50 daPa ). In sum, these results suggest that participant 09 contract voluntarily is TTM in his RE and his SM in his LE. For participant 10, both muscles seem to be contracted by the vMEMC. Finally, for participant 11, the TTM is contracted voluntarily in both ears. It is worth mentioning that TTM and SM contractions are more easily recognizable by air pressure and admittance, respectively. Moreover, one notes that TTM does not react (contract) to loud sounds, at least for the auditory stimulation provided in this study.

### *3.3 Perceptual effect of vMEMC*

The hearing thresholds under vMEMC conditions were measured bilaterally in the three cases to assess the perceptual impact of vMEMC. Using standard audiometry, previous studies have shown that vMEMC is associated with low-frequency hearing loss, mostly measured at 250 and 500 Hz (Angeli et al., 2013; Bance et al., 2013; Nomura et al., 1979; Reger, 1960; Reger et al., 1962; Smith, H.D., 1943; Wickens et al., 2017). We thus wanted to confirm these results and extend them by measuring the hearing threshold shift at a much lower frequency (up to 22 Hz) and with more precise frequency steps ( $\frac{1}{4}$  octave steps) (see the methods section for the procedure used). As expected, the low frequencies' hearing thresholds were affected by vMEMC for all participants and both ears: it is associated with a clear elevation in thresholds (Figure 10). The highest threshold shift is seen for participant 09 RE: in this ear, the air conduction thresholds (ACT) are mainly affected for frequencies below 1000 Hz with a maximal change of around 39 dB at 88 Hz and 38 dB at 125 Hz. Similar frequencies (1000 Hz and below) are also affected by vMEMC in participant 09 LE, but to a lesser extent: the maximal threshold shift in this ear is 10 dB at 100 Hz.

Similar results were found for the bone conduction thresholds (BCT) under the vMEMC condition with an increase in hearing thresholds for the low frequencies below 1000 Hz. The mean average BCT elevation across all participants' ears was 9, 6 and 4 dB for 250, 176 and 125 Hz frequencies, respectively. The highest elevation of 18 dB was found at 250 Hz in the RE and the lowest of 3 dB in the LE of participant 09.

After hearing threshold measurement, participants' pitch matched the sound produced by vMEMC to an external sound. Participants reported that the frequency of the sound emitted by vMEMC was of similar frequency (low frequency) across ears, but the loudness differed: Participant 09 reported that the sound was much louder in the RE compared to the LE and Participant 11 reported the reverse case with a louder vMEMC sound emitted in the LE compared to the RE. For participant 10, the sound was similar across ears. The sound that best matched their perception was all very low in frequency, 28, 40 and 32 Hz bilaterally for participants 09, 10 and 11, respectively (Figure 10). Moreover, the loudness level in dB SL well matched their perception: the vMEMC sound was matched to a 28 Hz played at 26 dB SL and 10 dB SL for participant 09 RE and LE respectively. For participant 10, it was matched to a 40 Hz tone set at 13 and 18 dB SL in the RE and LE, respectively and to a 32 Hz at 21 and 37 dB SL for the RE and LE of participant 11, respectively. All the psychoacoustic measurements were made ipsilaterally. They all reported that very low-frequency sound (around 30 Hz) at a level above 80 dB SPL produced a similar kinesthetic sensation of movement of their ears as the one produced by vMEMC contraction. Overall, these results suggest that vMEMC, produces a very low-frequency sound. However, the loudness of the sound emitted seems to be more important when the TTM is involved in the contraction (above 15 dB SL) compared to SM contraction alone (around 10 dB SL measured in Participant 09 LE).

#### *4. Video recording of a vMEMC case*

We had the opportunity to video record the vMEMC that involved TTM contraction in the RE of participant 09; it was thus the occasion to validate that TTM contraction could be seen with bare eyes (Loeb, 1964). This observation would further validate the negative pressure peaks during vMEMC associated with TTM contraction. Supplementary video 1 presents the video recording of the movements of the eardrum

during vMEMC. The first recording was performed without cleaning the RE of participant 09: this video is presented because the hair stuck on the eardrum facilitates the visualization of the eardrum's retraction during each voluntary vMEMC contraction. The second recording is simply vMEMC contractions of the same ear, after the wax and the hair were removed. The amplitude of the eardrum retraction is visible with bare eyes, suggesting that the TTM muscle contraction is substantial: the retraction of the anteroinferior quadrant of the pars tensa is observed in both recordings.

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Video 1 about here  
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## **DISCUSSION**

Our goal was to develop efficient methods that can be used to objectify putative abnormal TTM contractions in patients reporting symptoms that may involve the TTM. We believed that air pressure, providing an indirect measure of the eardrum movement following MEMC, could be a potential novel method to detect TTM contraction: as the eardrum is retracted by the contraction, a negative pressure peak could act as a marker of TTM contraction. To verify these assertions, we first wanted to compare middle ear admittance to air pressure modifications following auditory stimulus-evoked middle ear muscle contraction. We've presumed that the SM would be the strained muscle under this condition as the TTM does not contract to sound (Jones et al., 2008). The measure of admittance is currently the gold standard in detecting the presence of SM contraction and was thus the measure of choice for the comparison (Fowler & Shanks, 2002). For this condition, we found the expected decrease of admittance in most ears (93%) and an increase of air pressure deflection in 29% of ears. To note, no decreased air pressure deflections were found for all ears tested under sMEMC conditions. Despite the lack of decreased air pressure deflection, these results were consistent with previous studies on sMEMC measuring air pressure (Lidén et al., 1970; A. R. Møller, 1964). One might wonder if the presence of eardrum movement following SM contraction in some ears may give information about the connection of the SM muscle to the stapes and/or about a specific organization of the ossicles and the eardrum in those individuals. It has been



posited that the stapes undergo a rocking motion inside the oval window when the stapes muscle tendon pulls on it (Hüttenbrink, 1989; Marchbanks, 2002). The net effect is expected to be rather neutral on the eardrum unless the position of the stapes footplate moves outward such as in cases of increased intracranial pressure. Unfortunately, it is not known if the participants with eardrum displacement had increased intracranial pressure. We then tested, with a similar method, the ears of participants who can voluntarily contract their MEM. We knew, from previous research (Aron et al., 2015; Lidén et al., 1970), that the TTM was usually involved in these types of contractions. For vMEMC (n=8 ears), negative deflections were found for both admittance and air pressure in 100% and 88%, respectively. These results are consistent with an inward displacement of the tympanic membrane caused by TTM contraction. Additional admittance testing under negative auditory external canal pressure (-50 daPa) confirmed this interpretation (Aron et al., 2015).

To replicate and extend previous work on vMEMC, we also measured the perceptual effect of vMEMC by measuring the hearing threshold shift produced by vMEMC and the psychoacoustic properties of the fluttering sound emitted during the contraction. These perceptual attributes could also be used to identify the clinical manifestation of TTTS in patients. A low-frequency hearing loss with a maximal shift for frequencies below 150 Hz was found during vMEMC for all ears. The fluttering sound produced by vMEMC was pitch matched to a very low frequency, around 30 Hz. However, the sound's loudness was estimated to be more than a sound presented at 15 dB SL when TTM was involved in the voluntary contraction and 10 dB SL for SM alone. These perceptual attributes, i.e., the low-frequency hearing loss, the pitch and loudness of the fluttering sound, could be used to identify the clinical manifestation of the middle ear muscles abnormal contraction in patients and potentially TTTS. We also had the opportunity to video record vMEMC when TTM contraction was present, and the results showed apparent retraction of the eardrum as previously described in the literature (Loeb, 1964). The visible retraction of the eardrum, the negative peak of air pressure, and the change of admittance produced by vMEMC, including the inverted admittance peak for -50 daPa pressure, suggest that TTM is activated during vMEMC. However, the possibility that SM is also contracted during vMEMC cannot be ruled out.

### **The experience of voluntary TTM contraction**

The prevalence of individuals who can voluntarily contract their MEM muscle in the general population has been roughly estimated at around 1-2% of the general population (Reger, 1960). This estimation is based on Reger's observation, "from 120 students in the Junior Medical Class at the State of the University of Iowa, that only one or two are found each year who can contract their middle ear muscles voluntarily, but usually in a limited degree" (Reger, 1960). Based on our own experience, we believe that this underestimates the true prevalence of individuals who can voluntarily contract their MEM. At the end of one of our studies assessing middle ear function by continuous admittance recordings, we asked each of the 15 participants if they could produce a voluntary movement of their eardrums. If they answered "yes", we registered the peak of admittance produced by vMEMC every 5 seconds similarly to the results presented for participants 09, 10 & 11 of the present study. Four out of fifteen participants (27%) verbally reported that they could "move their eardrum", and admittance peaks attested to this ability during continuous admittance recordings. More so, on most occasions when we met researchers or clinicians who reported that they were able to "move their eardrums", either peak of contractions visible on the continuous admittance or pressure captor recordings attested to their claim. It was reported that, by guided practice, it is possible to develop such an ability (Reger et al., 1962). According to Reger and collaborators (1962), there seem to be different ways to transfer MEM contraction from a reflex over to voluntary control: "Those who in addition also experienced some sort of kinesthetic sensation in their ears while yawning found it relatively easy to learn to contract their middle ear musculature at will" (Reger, 1960).

In the current study, all the participants described a kinesthetic sensation during vMEMC: they all reported a physical sensation of movement inside their ear associated with a fluttering low-frequency noise. The fluttering or trembling noise was proposed to be generated by the muscle tremor during the contraction (Reger et al., 1962). In addition, "it was found that the sound produced by a 20 Hz tone reproduced in an earphone approximated the sound resulting from the tremor of voluntary contractions when this tone was presented at 18 dB sensation level" (Reger, 1960). The results obtained here are

completely in agreement with Reger's results: Participant 09, 10 and 11 pitch matches the fluttering noise to a 28 Hz, 40 Hz and 32 Hz tone in both ears, respectively. In dB SPL, the sound's loudness was matched at 81 and 95 dB SPL for the LE and RE of participant 09, respectively, and 96 and 86 dB SPL for the LE and RE of participant 11, respectively. Considering that a 30 Hz tone at 90 dB SPL has the same loudness as a 1 kHz tone at 60 dB SPL, one can say that the sound emitted during the vMEMC is quite loud for these two participants. The lowest loudness match was made by Participant 10 with 69- and 66-dB SPL for her LE and RE, respectively. The values obtained in dB SL of 26, 10, 13, 18, 21 & 27 dB SL, for participants 09, 10, 11 RE and LE respectively, are similar to the 18 dB SL reported in one case by Reger (1960). When assessing the psychoacoustic measurements of the fluttering sound, participant 09 reported that the 28 Hz sound presented at high levels of stimulation ( $> 80$  dB SPL) was producing a kinesthetic sensation similar to the one perceived during vMEMC (but of lower intensity). It can be speculated that the 28 Hz frequency sound at high intensity generates large movements of the eardrum. For the 4 participants ( $n=7$  ears) tested with negative pressure in the auditory ear canal during admittance recording, the -50 daPa change of pressure did not affect the ability to produce vMEMC. Only TTM in a hyperactive state (such as the TTTS) may be responsive to pressure variation.

It is worth mentioning that similar tremors and fluttering noise has been reported after an acoustic shock injury (Londero et al., 2017). In that case, the tinnitus pitch was measured at 12 kHz, and the tremolo, estimated from amplitude-modulated stimulus at the tinnitus frequency, was found at 32 Hz. This tremolo is similar to the rate of the fluttering noise reported here (28-40 Hz) and in previous cases (20 Hz) of the voluntary MEM contractions (Reger, 1960). It is thus possible that the tinnitus's tremolo at 32 Hz in the acoustic shock injury case has the same origin as the fluttering and tremor sensation observed for voluntary contractions, e.g., the TTM and/or the SM. Additionally, the patient reported that non-forceful eyelid closure produced eardrum movements associated with a physical kinesthetic sensation in the most symptomatic ear only, attested by admittance peaks. vMEMC were also influenced by the closure or the opening of the eyes: 1) participants 09, 10 and 11 all reported that it was easier to produce vMEMC when closing the eyes than when the eyes were kept open. Participant 09 can still contract

his TTM in his RE with his eyes open, while he cannot contract his SM voluntarily in his LE for the same condition. This might be explained by the fact that SM contraction and closure of the eye movements are both generated through the facial nerve, while TTM contraction is triggered by the trigeminal nerve. It is known that the eyeblink reflex and the TTM contractions are both involved in the startle reflex response (Klockhoff, Ingmar & Anderson, Henry, 1960). These results suggest a connection between the TTM and the eye blink; this connection is usually not apparent in a normal state but may become unmasked during a pathological state. Altogether, these results emphasize the complex interactions between the TTM and the SM and between the facial and trigeminal nerve (Sanders, 2010).

### **Objective validation of TTM voluntary contractions**

In the present study, TTM contraction was validated by two main measures: admittance and air pressure. Previous studies on vMEMC using continuous admittance had shown that it was possible to record vMEMC and demonstrate that these voluntary contractions involved the TTM (Aron et al., 2015). At normal pressure, TTM and SM contraction increases the tympano-ossicular chain's stiffness: for a low-frequency probe such as the 226 Hz tone, the admittance is negatively affected by this stiffness increase resulting in a spike-like decrease admittance deflection for every contraction. However, by applying negative pressure to the auditory ear canal, the eardrum is artificially pulled outward. In this unusual position of the eardrum, TTM contraction will pull the eardrum back in its usual position before pulling it even more inside the middle ear cavity leading to a transitory decrease of stiffness observed as an increased admittance deflection. This negative pressure will only influence the admittance recording if there is TTM contraction and not if the sole muscle contracted is the stapedius. The new position of the eardrum caused by the negative pressure did not affect SM contraction as seen in the present study. The results obtained here agree with Aron and colleagues' previous results (2015): vMEMC recordings produced a spike-like decrease of admittance for almost all ears (except one) at normal pressure and a spike-like increase of admittance for negative pressure, suggesting that TTM is involved in vMEMC. As expected, SM contractions, either triggered by sound or voluntary control, were only slightly affected by the change

of pressure. This technique can be used to identify the presence of TTM contraction. However, it has several disadvantages: 1) two separate recordings of the same phenomenon as to be made, one at 0 daPa and the other at -50 daPa, which is not convenient and time-consuming, 2) simultaneous recording of the two ears cannot be performed, at least with standard tympanometry equipment, 3) a sound is continuously presented (226 Hz probe tone) at a loud level which is not convenient for patients with loudness hyperacusis and 4) admittance recordings can be contaminated by environmental noise.

The second measurement used in this study was the air pressure measured within an airtight sealed ear canal. The first attempt to use this type of system to objectify TTM voluntary contraction goes back to Mangold and Eckstein in 1913; they used a liquid manometer sealed in the ear canal. More than fifty years later, and using more sophisticated equipment, Lidén and collaborators reported the results of a simultaneous recording of changes in relative impedance and air pressure in two normal subjects who could voluntarily contract their MEM (Lidén et al., 1970). For the air pressure, they observed a sudden spike-like decrease of pressure for vMEMC that they attributed to the TTM. The results obtained here are almost identical to the ones obtained by Lidén and his colleagues. First, they found sudden spike-like decrease pressure repeated on several occasions for vMEMC, the value of these contractions being on average around -100 Pa, like the ones found here. Secondly, during the reflex decay test, the acoustic stimulation produced a small increase in pressure maintained during the sound stimulation and when vMEMC was performed, a sudden spike-like decrease of pressure was superimposed on this small increase of air pressure, like the results of participant 09 RE.

In sum, from the current study results and previous research, it seems reasonable to conclude that admittance under negative pressure and air pressure measurements can reliably assess TTM contraction present during vMEMC. In addition, vMEMCs were associated with a large amplitude retraction of the eardrum visible on video recording; it seems plausible that these retractions are responsible for the air pressure change. It has long been known, by direct observation, that TTM contraction produces a large amplitude retraction of the eardrum (Loeb, 1964). The air pressure measurement is a complementary measurement of tympanic membrane displacement. However, it's not

possible with this technique to identify if the TTM is the only muscle contracted or if both the SM and the TTM are contracted.

### **Elevation of hearing thresholds for the low frequencies during vMEMC contraction**

The evaluation of hearing thresholds under vMEMC revealed that it causes both bone and air conduction threshold elevation for frequency under 1000 Hz for as long as the contraction is maintained. There is, however, a large variability between ears and individuals: Participants 09 RE ACT had a maximal hearing threshold shift of 39 dB at 100 Hz while participants 10 RE & LE had a maximal shift of 17 dB at 40 and 30 Hz, respectively. The same is true for BCT, with participant 09 RE displaying the largest shift of 18 dB at 250 Hz and Participant 10 displaying more modest shifts of 5 dB in both ears for the same frequency. These results are in line with previous reports of low-frequency hearing loss in humans during vMEMC in participants who were voluntarily able to contract their MEM (Angeli et al., 2013; Bance et al., 2013; Nomura et al., 1979; Reger, 1960; Reger et al., 1962; Smith, H.D., 1943; Wickens et al., 2017). The average values obtained here are like those obtained by Wikens et al. (2017), where, from five years, the average threshold shift for the AC threshold at 250 Hz was around 20 dB and around 10 dB for the BC. However, for most of these studies mentioned above, it is unclear how the test was adapted (if adapted) to measure the hearing thresholds during vMEMC. Indeed, despite mentioning that these tests were run only on participants who were able to contract their MEM for a long duration of time, it seems that the conventional clinical hearing test procedure was used. Despite the claims that some participants can maintain the contraction for 20 seconds up to a few minutes (Reger et al., 1962), there is no objective recording of such long time duration vMEMC. Even here, some participants were convinced they could maintain their contractions for a long duration, and still, for none of them, the maintenance in the admittance nor air pressure change was obtained. This raises doubt on the ability of the TTM to sustain his contraction over time. The fluttering sound lasts more than a few seconds suggesting that it is partially independent of the eardrum movement. However, voluntary MEM contraction may be different from triggered and/or spontaneous contractions. For example, participant 09 could voluntarily produce a phasic contraction of SM in his LE but could not sustain the contraction.

Conversely, SM contraction is sustained in that same ear during prolonged auditory stimulation. The procedure to measure hearing threshold shift caused by vMEMC used here that is, using a pulse sequence of long pure tone duration and asking subjects whether they hear a “gap” in the continuous sound could be used in further research for all vMEMC participants but who are not able to sustain the contraction for long periods.

The threshold shifts obtained during vMEMC here are also in line with the threshold shift seen in animals' studies using pulls of different weights applied on the TTM muscle (Neergaard et al., 1962; Teig, 1973). Teig (1973) observed a reduction of the cochlear microphonics for frequencies below 750 Hz when the cat's tensor tympani were at 10 to 15% of its maximal contraction force, while higher frequencies could be either slightly enhanced or reduced. Higher contraction amplitude resulted in a more widespread cochlear microphonics reduction across frequencies, with the low frequencies displaying the highest thresholds. The results obtained in participant 09 RE are like those obtained in cats with threshold shifts for frequencies under 1000 Hz up to 20 Hz. A small increase in the threshold was also seen for some higher frequencies, 4 to 8 kHz, for ACT only. Interestingly, both participants (09 & 11) reported a clear decrease in the hearing threshold at 1.6 and 2 kHz for participant 09 RE and at 1.2 kHz for participant 11 RE during TTM contraction: a sound was presented a few dB under the threshold and was only heard during TTM contractions. This is in agreement with the first case study published on the audiometric effect of TTM contractions: the participant reported that the 2000 Hz tone was increased slightly in loudness during vMEMC (Smith, 1943). The small decrease (improvement) in hearing threshold near 2000 Hz was also reported in cats only for lightweight applied to the TTM (Teig, 1973). We cannot exclude the possibility that the threshold shifts may be caused entirely or partially by SM contraction (Pang & Peake, 1986).

As previously shown by Wickens et al. (2017), the ears of two participants displayed during vMEMC a low frequency mixed hearing loss with conductive and sensorineural components. The stiffening of the tympano-ossicular chain by TTM contraction is most probably the primary mechanism responsible for conductive hearing loss. The possible contribution of the SM cannot be excluded as there is no way to ascertain that SM is not contracted with the TTM during vMEMC. Many different

mechanisms may be responsible for the sensorineural component (BCT threshold shift). A TTM contraction pushes the stapes inside the vestibular canal by around 10 microns which may increase the inner ear pressure (Bell, 2011, 2017). Ultimately, this pressure increase could modify the cochlear micromechanics leading to threshold elevation and transient hearing loss. An increase in intralabyrinthine pressure was also proposed to explain the BC threshold shift, mainly affecting the low frequencies (<2000 Hz) when BCT was measured under positive and negative external ear canal pressure in normal hearing participants (Rasmussen, 1948). The effect of MEMC on the stapes footplate into the oval window, stretching of the annular ligament and round window membrane may have changed the cochlear impedance load resulting in BC threshold elevation. Finally, the threshold shift may have occurred due to the masking of the target sound by the sound produced by the muscle contraction (Wickens et al., 2017).

### **Clinical implications**

The current research has definite clinical implications. First, several of the methods used here could be implemented in clinics to assess middle ear muscle dysfunction in patients suffering from symptoms associated with TTTS (Fournier et al., submitted). As previously mentioned, hearing threshold shifts for low frequencies, especially in the 40-200 Hz frequency region, could be a clinical manifestation of the TTTS (Pau et al., 2005; Wickens et al., 2017).

In addition, TTM dysfunction may be uncovered by somatic maneuvers and sound stimulation. Middle ear muscle contractions have been long known to contract in association with other somatic reflexes such as head turns in animals and men (Simmons, 1964). After MEM injury, the muscle may be in a hyperactive state and react more strongly to other somatic reflexes or even sound, generating adverse effects and bizarre symptoms.

To date, there is no evidence suggesting that the TTM can sustain a contraction and thus, that symptoms associated with TTTS are in fact due to a tonic state of the TTM. More so, most participants with the ability to contract voluntarily their TTM were not able to sustain the contraction arguing against the tonic tensor tympani explanation. However, there is evidence that patients with TTTS symptomatology including



hyperacusis, otalgia, tinnitus and ear fullness display middle ear muscle dysfunction such as spontaneous, somatic, and sound-evoked TTM phasic contractions (Fournier et al. submitted). We believe that the term Tonic Tensor Tympani Syndrome should be abolished and replaced by another term, such as Hyper-Reactive Tensor Tympani Syndrome, for instance (see Fournier et al., submitted). The pressure captor could be used to objectively measure TTM phasic contractions in patients with TTTS symptomatology. In addition, it could be used to assess tinnitus resulting from myoclonic TTM or SM contractions. In these rare cases, the TTM or the SM have been reported to contract sporadically, producing audible clicks for the patients at a level that is sometimes audible for an external observer (Abdul-Baqi, 2004; Badia et al., 1994; Bance, 2019; Brigo et al., 2013; Dang & Carol Liu, 2019; Ellenstein et al., 2013; Freund, 1956; Hornibrook & Keast, 2019; Howsam et al., 2005; Kim et al., 2013; Kwee & Struben, 1972; Lee et al., 2012; Oliveira et al., 2003; Phillipps et al., 1989; Rajah, 1992; Rock, 1995; Salehi et al., 2019; Watanabe et al., 1974; Zipfel et al., 2000). One of the main challenges in these cases is to differentially diagnose the origin of the myoclonic activity between the TTM and the SM. Considering that cutting the muscle's tendon is one known therapeutic approach (Golz et al., 2003), a precise differential diagnosis is critical to guide the intervention. The pressure captor can be very useful to identify TTM myoclonic activity: a large negative pressure peak would indicate TTM contractions. However, if both muscles are in a myoclonic mode, only the activity of the myoclonic TTM would be apparent. If no pressure peaks are recorded, TTM involvement could be ruled out and the investigation could be directed toward other potential sound sources such as the SM or a palatal myoclonus (Oliveira et al., 2003; Seidman & Babu, 2003). It is worth noting that it has been anecdotally reported that many symptomatic patients described that pressurizing their ear canals gets rid of or reduces their middle ear myoclonus symptoms. In these cases, the pressure captor would not be useful. The pressure captor could also be useful to objectify the presence of TTM contractions in *Forceful Eyelid Closure Syndrome (FECS)* (Kim et al., 2013; Lee et al., 2012; Rajah, 1992; Rock, 1995) or myoclonus triggered by palpation of skeletal muscles of the jaw and the face such as the sternocleidomastoid and the trapezius (Salehi et al., 2019). It has been reported that

tinnitus can be triggered and/or modulated by pressure applications on myofascial trigger points (Rocha & Sanchez, 2007, 2007; Teachey et al., 2012).

Finally, air pressure measurement may be more suited to assess TTM contractions than continuous admittance recording for patients with loudness and/or painful hyperacusis (Noreña et al., 2018; Pollard, 2019a, 2019b; Westcott, 2016; Westcott et al., 2013). The former has been defined to be present « when moderately intense sounds are judged to be very loud compared with what a person with normal hearing would perceive » and the latter, « when one with hyperacusis experiences pain at much lower sound levels than listeners with normal hearing » (Tyler et al., 2014). Performing tympanometry or continuous admittance recording can be a highly unpleasant experience for these patients (Tidball & Fagelson, 2017). Considering that the level of the probe tone is around 74 dB SPL (85, 72, 70, and 69 dB SPL, for 226, 678, 800, and 1000 Hz probe tone, respectively; Madsen Zodiac Reference Manual) and above the loudness discomfort level of some patients (Aazh & Moore, 2017), it is very difficult to assess middle ear function using tympanometry in these patients - they are afraid that the probe tone may exacerbate their symptoms of pain and sound sensitivity for several days (Littwin, 2017). The pressure captor has the advantage of assessing eardrum movements without requiring any sound presentation: patients feel safer with this type of measurement. Other advantages of the pressure captor include the relative immunity of the measurements to sound artifact and simultaneous recordings of the two ears.

## CONCLUSIONS

Tensor tympani muscle contractions can be measured through air pressure and admittance recordings in participants who can voluntarily contract their middle ear muscles and possibly patients with symptomology consistent with TTTS. vMEMC involving TTM contraction was associated with decreased admittance and pressure, while SM contraction was associated with a reduction in admittance and either an increase of air pressure or no modification of pressure. We hope that the new tests developed here will pave the way for new approaches to investigate the MEMs.

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## References

- Aazh, H., & Moore, B. C. J. (2017). Incidence of Discomfort During Pure-Tone Audiometry and Measurement of Uncomfortable Loudness Levels Among People Seeking Help for Tinnitus and/or Hyperacusis. *American Journal of Audiology*, 26(3), 226. [https://doi.org/10.1044/2017\\_AJA-17-0011](https://doi.org/10.1044/2017_AJA-17-0011)
- Abdul-Baqi, K. J. (2004). Objective high-frequency tinnitus of middle-ear myoclonus. *The Journal of Laryngology & Otology*, 118(3), 231–233. <https://doi.org/10.1258/002221504322928044>
- Angeli, R. D., Lise, M., Tabajara, C. C., & Maffacioli, T. B. (2013). Voluntary contraction of the tensor tympani muscle and its audiometric effects. *The Journal of Laryngology & Otology*, 127(12), 1235–1237. <https://doi.org/10.1017/S0022215113003149>
- Aristeguieta, L. M. R., Acuña, L. E. B., & Ortiz, G. P. S. (2010). [Tensor veli palatini and tensor tympani muscles: Anatomical, functional and symptomatic links]. *Acta Otorrinolaringologica Espanola*, 61(1), 26–33. <https://doi.org/10.1016/j.otorri.2009.08.006>
- Aron, M., Floyd, D., & Bance, M. (2015). Voluntary Eardrum Movement: A Marker for Tensor Tympani Contraction? *Otology & Neurotology*, 36(2), 373–381. <https://doi.org/10.1097/MAO.0000000000000382>
- Atcherson, S. R., & Stoodly, T. M. (2011). Acoustic Stapedial Reflex Test Modification in Ears with High Static Admittance and Asymmetric Hearing Loss: A Case Study. *Asia Pacific Journal of Speech, Language and Hearing*, 14(2), 129–138. <https://doi.org/10.1179/136132811805334821>
- Badia, L., Parikh, A., & Brookes, G. B. (1994). Management of middle ear myoclonus. *The Journal of Laryngology & Otology*, 108(5), 380–382. <https://doi.org/10.1017/S0022215100126866>
- Bance, M. (2019). Middle ear muscle disorders: Presentation, diagnosis and management. *ENT and Audiology News*, 28(1), 2.
- Bance, M., Makki, F. M., Garland, P., Alian, W. A., van Wijhe, R. G., & Savage, J. (2013). Effects of tensor tympani muscle contraction on the middle ear and markers of a contracted muscle: Markers of Tensor Tympani Muscle Contraction. *The Laryngoscope*, 123(4), 1021–1027. <https://doi.org/10.1002/lary.23711>
- Bell, A. (2011). How the middle ear muscles protect the cochlea? Reconsideration of the intralabyrinthine pressure theory. *Journal of Hearing Science*, 1(2), 16.
- Bell, A. (2017). Middle Ear Muscle Dysfunction as the cause of Meniere's Disease. *Journal of Hearing Science*, 3, 9–25. <https://doi.org/10.17430/904674>
- Brigo, F., Storti, M., Lochner, P., & Nardone, R. (2013). Transitory stapedial myoclonus in a patient with benign fasciculation syndrome. *The Journal of Laryngology & Otology*, 127(6), 605–606. <https://doi.org/10.1017/S0022215113000297>
- Burns, E. M., Harrison, W. A., Bulen, J. C., & Keefe, D. H. (1993). Voluntary contraction of middle ear muscles: Effects on input impedance, energy reflectance and spontaneous otoacoustic emissions. *Hearing Research*, 67(1–2), 117–127. [https://doi.org/10.1016/0378-5955\(93\)90239-W](https://doi.org/10.1016/0378-5955(93)90239-W)
- Dang, J., & Carol Liu, Y.-C. (2019). Treatment of objective tinnitus with transpalatal Botox® injection in a pediatric patient with middle ear myoclonus: A case report.

- International Journal of Pediatric Otorhinolaryngology*, 116, 22–24.  
<https://doi.org/10.1016/j.ijporl.2018.09.024>
- DiGiovanni, J. J., & Ries, D. T. (2007). Stapedial reflex and ears with high static acoustic admittance. *American Journal of Audiology*, 16(1), 68–74.  
[https://doi.org/10.1044/1059-0889\(2007/006\)](https://doi.org/10.1044/1059-0889(2007/006))
- Ellenstein, A., Yusuf, N., & Hallett, M. (2013). Middle ear myoclonus: Two informative cases and a systematic discussion of myogenic tinnitus. *Tremor and Other Hyperkinetic Movements (New York, N.Y.)*, 3. <https://doi.org/10.7916/D8RX9BS1>
- Fournier, P., Cuvillier, A.-F., Gallego, S., Paolino, F., Paolino, M., Quemar, A., Londero, A., & Norena, A. (2018). A New Method for Assessing Masking and Residual Inhibition of Tinnitus. *Trends in Hearing*, 22, 233121651876999.  
<https://doi.org/10.1177/2331216518769996>
- Fowler, C. G., & Shanks, J. E. (2002). Tympanometry. In *Handbook of Clinical Audiology* (Fifth, pp. 175–204). Lippincott Williams & Wilkins.
- Freund, E. M. (1956). Objective Audible Clicking in Ear: Presentation. *Archives of Otolaryngology - Head and Neck Surgery*, 64(2), 129–133.  
<https://doi.org/10.1001/archotol.1956.03830140045006>
- Golz, A., Fradis, M., Martzu, D., Netzer, A., & Joachims, H. Z. (2003). Stapedius muscle myoclonus. *The Annals of Otology, Rhinology, and Laryngology*, 112(6), 522–524. <https://doi.org/10.1177/000348940311200607>
- Goodfriend, D. J. (1947). Deafness, Tinnitus, Vertigo and Neuralgia. *Archives of Otolaryngology - Head and Neck Surgery*, 46(1), 1–35.  
<https://doi.org/10.1001/archotol.1947.00690020008001>
- Hall, C. M. (1977). Stapedial reflex decay in retrocochlear and cochlear lesions. Review of procedures and methods for conducting SRD tests. *The Annals of Otology, Rhinology, and Laryngology*, 86(2 pt. 1), 219–222.  
<https://doi.org/10.1177/000348947708600213>
- Hammershøi, D., & Møller, H. (1996). Sound transmission to and within the human ear canal. *The Journal of the Acoustical Society of America*, 100(1), 408–427.  
<https://doi.org/10.1121/1.415856>
- Harrell, R. W. (2002). Puretone evaluation. In *Handbook of Clinical Audiology* (Katz, J., pp. 71–87). Lippincott Williams & Wilkins.
- Holt, G. R., Watkins, T. M., & Yoder, M. G. (1982). Assessment of tympanometry in abnormalities of the tympanic membrane. *American Journal of Otolaryngology*, 3(2), 112–116. [https://doi.org/10.1016/S0196-0709\(82\)80041-0](https://doi.org/10.1016/S0196-0709(82)80041-0)
- Hornibrook, J., & Keast, A. (2019). Bilateral clicking tinnitus due to myoclonus of middle ear muscles. *The New Zealand Medical Journal*, 132(1492), 67–69.
- Howsam, G. D., Sharma, A., Lambden, S. P., Fitzgerald, J., & Prinsley, P. R. (2005). Bilateral objective tinnitus secondary to congenital middle-ear myoclonus. *The Journal of Laryngology & Otology*, 119(6), 489–491.  
<https://doi.org/10.1258/0022215054273223>
- Hüttenbrink, K. B. (1989). [Movement of the ear ossicles by middle ear muscle contraction]. *Laryngo- Rhino- Otologie*, 68(11), 614–621.
- Jerger, J. (1970). Clinical experience with impedance audiometry. *Archives of Otolaryngology (Chicago, Ill.: 1960)*, 92(4), 311–324.  
<https://doi.org/10.1001/archotol.1970.04310040005002>

- Jones, S. E. M., Mason, M. J., Sunkaraneni, V. S., & Baguley, D. M. (2008). The effect of auditory stimulation on the tensor tympani in patients following stapedectomy. *Acta Oto-Laryngologica*, *128*(3), 250–254. <https://doi.org/10.1080/00016480701509925>
- Kierner, A. C., Mayer, R., & Kirschhofer, K. (2002). Do the tensor tympani and tensor veli palatini muscles of man form a functional unit? A histochemical investigation of their putative connections. *Hearing Research*, *165*(1–2), 48–52. [https://doi.org/10.1016/s0378-5955\(01\)00419-1](https://doi.org/10.1016/s0378-5955(01)00419-1)
- Kim, B. G., Bang, M.-Y., Kim, D. H., & Moon, I. S. (2013). Tinnitus related to eyelid blinking. *Auris Nasus Larynx*, *40*(5), 518–520. <https://doi.org/10.1016/j.anl.2012.11.007>
- Klockhoff, Ingmar. (1979). Impedance Fluctuation and a “Tensor Tympani Syndrome.” *Proc 4th International Symposium on Acoustic Impedance Measurements*, 69–76.
- Klockhoff, Ingmar, & Anderson, Henry. (1960). Reflex activity of the tensor tympani muscle recorded in man. *Acta Oto-Laryngologica*, *51*(1–2), 184–188.
- Kwee, H. L., & Struben, W. H. (1972). Tinnitus and myoclonus. *The Journal of Laryngology & Otology*, *86*(3), 237–241. <https://doi.org/10.1017/S0022215100075198>
- Lee, G.-H., Bae, S.-C., Jin, S.-G., Park, K.-H., Yeo, S.-W., & Park, S.-N. (2012). Middle ear myoclonus associated with forced eyelid closure in children: Diagnosis and treatment outcome. *The Laryngoscope*, *122*(9), 2071–2075. <https://doi.org/10.1002/lary.23420>
- Lidén, G., Peterson, J. L., & Harford, E. R. (1970). Simultaneous recording of changes in relative impedance and air pressure during acoustic and non-acoustic elicitation of the middle-ear reflexes. *Acta Oto-Laryngologica. Supplementum*, *263*, 208–217. <https://doi.org/10.3109/00016487009131562>
- Littwin, R. (2017). Hyperacusis Management: A Patient’s Perspective. In *Hyperacusis and Disorders of Sound Intolerance: Clinical and Research Perspectives* (pp. 241–264).
- Loeb, M. (1964). Psychophysical correlates of intratympanic reflex action. *Psychological Bulletin*, *61*(2), 140–152.
- Londero, A., Charpentier, N., Ponsot, D., Fournier, P., Pezard, L., & Noreña, A. J. (2017). A Case of Acoustic Shock with Post-trauma Trigeminal-Autonomic Activation. *Frontiers in Neurology*, *8*. <https://doi.org/10.3389/fneur.2017.00420>
- Mansour, S., Magnan, J., Haidar, H., Nicolas, K., & Louryan, S. (2013). *Comprehensive and Clinical Anatomy of the Middle Ear*. Springer Berlin Heidelberg. <https://doi.org/10.1007/978-3-642-36967-4>
- Marchbanks, R. J. (1984). Measurement of Tympanic Membrane Displacement Arising from Aural Cardiovascular Activity, Swallowing, and Intra-aural Muscle Reflex. *Acta Oto-Laryngologica*, *98*(1–2), 119–129. <https://doi.org/10.3109/00016488409107543>
- Marchbanks, R. J. (2002). Measurements of inner ear fluid pressure and clinical applications. In *A Textbook of Audiological Medicine* (pp. 289–307). <https://doi.org/10.1201/b14730-20>
- Møller, A. (1963). Transfer function of the Middle Ear. *Journal of The Acoustical Society of America*, *35*(10), 1526–1534. <https://doi.org/10.1121/1.1918742>

- Møller, A. R. (1961). LVI Bilateral Contraction of the Tympanic Muscles in Man: Examined by Measuring Acoustic Impedance-Change. *Annals of Otology, Rhinology & Laryngology*, 70(3), 735–752. <https://doi.org/10.1177/000348946107000309>
- Møller, A. R. (1964). Effect of Tympanic Muscle Activity on Movement of the Eardrum, Acoustic Impedance and Cochlear Microphonics. *Acta Oto-Laryngologica*, 58(1–6), 525–534. <https://doi.org/10.3109/00016486409121413>
- Møller, A. R. (1984). Neurophysiological Basis of the Acoustic Middle-Ear Reflex. In *The Acoustic Reflex, Basic Principles and Clinical Applications* (Shlomo Silman). Academic Press, Inc.
- Neergaard, E., Andersen, H. C., Hansen, C. C., & Jepsen, O. (1962). Experimental Studies on Sound Transmission in the Human Ear. *Acta Oto-Laryngologica*, 54(1–6), 511–520. <https://doi.org/10.3109/00016486209126969>
- Nomura, Y., Harada, T., & Fukaya, T. (1979). Dynamic tympanometry. *Journal of the American Auditory Society*, 4(5), 190–194.
- Noreña, A. J., Fournier, P., Londero, A., Ponsot, D., & Charpentier, N. (2018). An Integrative Model Accounting for the Symptom Cluster Triggered After an Acoustic Shock. *Trends in Hearing*, 22, 2331216518801725. <https://doi.org/10.1177/2331216518801725>
- Oliveira, C. A., Negreiros Júnior, J., Cavalcante, I. C., Bahmad, F., & Venosa, A. R. (2003). Palatal and middle-ear myoclonus: A cause for objective tinnitus. *The International Tinnitus Journal*, 9(1), 37–41.
- Pang, X. D., & Peake, W. T. (1986). How Do Contractions of the Stapedius Muscle Alter the Acoustic Properties of the Ear? In J. B. Allen, J. L. Hall, A. E. Hubbard, S. T. Neely, & A. Tubis (Eds.), *Peripheral Auditory Mechanisms* (pp. 36–43). Springer. [https://doi.org/10.1007/978-3-642-50038-1\\_5](https://doi.org/10.1007/978-3-642-50038-1_5)
- Pau, H. W., Punke, C., Zehlicke, T., Dressler, D., & Sievert, U. (2005). Tonic contractions of the tensor tympani muscle: A key to some non-specific middle ear symptoms? Hypothesis and data from temporal bone experiments. *Acta Oto-Laryngologica*, 125(11), 1168–1175. <https://doi.org/10.1080/00016480510012408>
- Phillipps, J. J., Reid, A., Marchbanks, R. J., & Jarrar, R. F. (1989). Objective tinnitus and tympanic membrane displacement. *The Journal of Laryngology & Otology*, 103(9), 872–873. <https://doi.org/10.1017/S0022215100110357>
- Pollard, B. (2019a). Unravelling the mystery of hyperacusis with pain. *ENT and Audiology News*, 27(6), 2.
- Pollard, B. (2019b). Clinical Advancements for Managing Hyperacusis with Pain: *The Hearing Journal*, 72(10), 10. <https://doi.org/10.1097/01.HJ.0000602900.16223.0e>
- Rajah, V. (1992). Tinnitus related to eyelid blinking. *The Journal of Laryngology & Otology*, 106(1), 44–45. <https://doi.org/10.1017/S0022215100118560>
- Rasmussen, H. (1948). 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-Laryngologica*, 36(sup74), 54–64. <https://doi.org/10.3109/00016484809131987>
- Reger, S. N. (1960). Effect of middle ear muscle action on certain psychophysical measurements. *Annals of Otology, Rhinology & Laryngology*, 69, 1–20. <https://doi.org/10.1177/000348946006900426>

- Reger, S. N., Menzel, O. J., Ickes, W. K., & Steiner, S. J. (1962, May 7). Changes in air conduction and bone conduction sensitivity with voluntary contraction of the middle ear musculature. *Proceedings of the Middle Ear Function Seminar*. Middle Ear Function Seminar, Fort Knox, Kentucky.
- Rocha, C. A. C. B., & Sanchez, T. G. (2007). Myofascial trigger points: Another way of modulating tinnitus. In B. Langguth, G. Hajak, T. Kleinjung, A. Cacace, & A. R. Møller (Eds.), *Progress in Brain Research* (Vol. 166, pp. 209–214). Elsevier. [https://doi.org/10.1016/S0079-6123\(07\)66018-X](https://doi.org/10.1016/S0079-6123(07)66018-X)
- Rock, E. H. (1995). OBJECTIVE TINNITUS AND THE TENSOR TYMPANI MUSCLE. *International Tinnitus Journal*, 1(1), 8.
- Salehi, P. P., Kasle, D., Torabi, S. J., Michaelides, E., & Hildrew, D. M. (2019). The etiology, pathogeneses, and treatment of objective tinnitus: Unique case series and literature review. *American Journal of Otolaryngology*, 40(4), 594–597. <https://doi.org/10.1016/j.amjoto.2019.03.017>
- Salomon, G., & Starr, A. (1963). Electromyography of middle ear muscles in man during motor activities. *Acta Neurologica Scandinavica*, 39(2), 161–168. <https://doi.org/10.1111/j.1600-0404.1963.tb05317.x>
- Sanders, R. D. (2010). The Trigeminal (V) and Facial (VII) Cranial Nerves. *Psychiatry (Edgmont)*, 7(1), 13–16.
- Seaver, E. P. (1937). Temporomandibular Joint Malocclusion and the Inner Ear, a Neuromuscular Explanation. *Annals of Otolaryngology, Rhinology & Laryngology*, 47(1), 140–149.
- Seidman, M. D., & Babu, S. (2003). Alternative medications and other treatments for tinnitus: Facts from fiction. *Otolaryngologic Clinics of North America*, 36(2), 359–381. [https://doi.org/10.1016/S0030-6665\(02\)00167-6](https://doi.org/10.1016/S0030-6665(02)00167-6)
- Simmons, F. B. (1964). Variable Nature of the Middle Ear Muscle Reflex. *International Audiology*, 3(2), 136–146. <https://doi.org/10.3109/05384916409074078>
- Smith, H.D. (1943). Audiometric effects of voluntary contraction of the tensor tympani muscles. *Archives of Oto-Rhino-Laryngology*, 38(4), 369–372.
- Teachey, W. S., Wijtman, E. H., Cardarelli, F., & Levine, R. A. (2012). *Tinnitus of myofascial origin*. 17(1), 4.
- Teig, E. (1973). Differential effect of graded contraction of middle ear muscles on the sound transmission of the ear. *Acta Physiologica Scandinavica*, 88(3), 382–391. <https://doi.org/10.1111/j.1748-1716.1973.tb05467.x>
- Tidball, G., & Fagelson, M. A. (2017). Audiological Assessment of Decreased Sound Tolerance. In *Hyperacusis and Disorders of Sound Intolerance: Clinical and Research Perspectives* (pp. 15–32).
- Tyler, R. S., Pienkowski, M., Roncancio, E. R., Jun, H. J., Brozoski, T., Dauman, N., Coelho, C. B., Andersson, G., Keiner, A. J., Cacace, A. T., Martin, N., & Moore, B. C. J. (2014). A Review of Hyperacusis and Future Directions: Part I. Definitions and Manifestations. *American Journal of Audiology*, 23(4), 402–419. [https://doi.org/10.1044/2014\\_AJA-14-0010](https://doi.org/10.1044/2014_AJA-14-0010)
- Watanabe, I., Kumagami, H., & Tsuda, Y. (1974). Tinnitus due to abnormal contraction of the stapedial muscle. *ORL; Journal for Oto-Rhino-Laryngology and Its Related Specialties*, 36, 217–226.



- Westcott, M. (2006). Acoustic shock injury (ASI). *Acta Oto-Laryngologica Supplementum*, 556, 54–58. <https://doi.org/10.1080/03655230600895531>
- Westcott, M. (2016). Hyperacusis-induced Pain: Understanding and Management of Tonic Tensor Tympani Syndrome (TTTS) Symptoms. *Journal of Pain & Relief*, 05(02). <https://doi.org/10.4172/2167-0846.1000234>
- Westcott, M., Sanchez, T., Sharples, T., Diges, I., Saba, C., Dineen, R., McNeill, C., Chiam, A., & O'Keefe, M. (2013). Tonic tensor tympani syndrome in tinnitus and hyperacusis patients: A multi-clinic prevalence study. *Noise and Health*, 15(63), 117. <https://doi.org/10.4103/1463-1741.110295>
- Wever, E. G., & Vernon, J. A. (1956). The Control of Sound Transmission by the Middle Ear Muscles. *Annals of Otology, Rhinology & Laryngology*, 65(1), 5–14. <https://doi.org/10.1177/000348945606500101>
- Wickens, B., Floyd, D., & Bance, M. (2017). Audiometric findings with voluntary tensor tympani contraction. *Journal of Otolaryngology - Head & Neck Surgery*, 46(1). <https://doi.org/10.1186/s40463-016-0182-y>
- Wiggers, H. C. (1937). The functions of the intra-aural muscles. *American Journal of Physiology-Legacy Content*, 120(4), 771–780. <https://doi.org/10.1152/ajplegacy.1937.120.4.771>
- Yeomans, J. S., Li, L., Scott, B. W., & Frankland, P. W. (2002). Tactile, acoustic and vestibular systems sum to elicit the startle reflex. *Neuroscience and Biobehavioral Reviews*, 11.
- Zipfel, T. E., Kaza, S. R., & Greene, J. S. (2000). Middle-ear myoclonus. *The Journal of Laryngology & Otology*, 114(3), 207–209. <https://doi.org/10.1258/0022215001905120>

FIGURE CAPTIONS

FIGURE 1. A) Schematic drawing of a set-up for the recording of the pressure captor. B) Schematic drawing of the calibration set-up of the pressure captor. C) Figure displaying the air pressure output measure (air pressure captor) when the air pressure input is modified by 50 Pascal steps from -600 to 600 Pascal (air pressure pump).

FIGURE 2. A) Admittance in ml (with a probe tone set at 226 Hz and 0 daPa) and B) Air pressure in Pascal recordings of 12 ears following contralateral white noise stimulation of 90 dB SPL. The sound onset is at 0 seconds for both, admittance and air pressure. C) Distribution of the maximal values of the admittance deflections. The dotted line is the baseline +/- 2 standard deviation (SD). D) Distribution of the maximal values of the increased pressure deflections. The dotted line is the baseline plus one standard deviation. E) Maximal values of the admittance deflections plotted against the maximal values of the air pressure deflection for all ears.

FIGURE 3. A) Air pressure responses (dark line = first measure, gray line = the second measure) to increase of contralateral white noise simulation from 66 to 99 dB SPL by 3 dB steps for both ears of participant 09. B) Admittance responses (226 Hz probe at 0 daPa pressure) to increase of contralateral white noise simulation from 66 to 99 dB SPL by 3 dB steps. Dark line = first measure, Gray line = second measure. C) Correlation between air pressure and admittance responses (226 Hz probe at 0 daPa) to increase of contralateral white noise simulation from 66 to 99 dB SPL by 3 dB steps. Dark circle = first measure, Gray circle = second measure.

FIGURE 4. A) Air pressure responses (dark line = first measure, gray line = the second measure) to increase of contralateral white noise simulation from 66 to 99 dB SPL by 3 dB steps for both ears of participant 09. B) Admittance responses (1 kHz probe at 0 daPa pressure) to increase of contralateral white noise simulation from 66 to 99 dB SPL by 3 dB steps. Dark line = first measure, Gray line = second measure. C) Correlation between air pressure responses and admittance responses (1 kHz probe at 0 daPa) to increase in contralateral white noise simulation from 66 to 99 dB SPL by 3 dB steps. Dark circle = first measure, Gray circle = second measure.

FIGURE 5. Admittance in ml with a probe tone set at 226 Hz and 0 daPa (upper panel) and -50 daPa (middle panel) following either vMEMC (left panels) or sMEMC (right panels). The air pressure levels in Pa are presented for vMEMC and sMEMC on the bottom panels. sMEMC was produced by a 90 dB SPL contralateral white noise.

FIGURE 6. Distribution of the maximal air pressure in Pa and admittance in ml (for a 226 Hz probe tone and both 0 and -50 daPa) for vMEMC and sMEMC. Only the ears (n=6) with results for all the measurements were included.

FIGURE 7. A) Static tympanometry results of participants 09, 10 and 11 for their RE (red) and LE (blue). The gray dotted line represents the area of normative values. B) Pressure and admittance recordings for participants 09, 10 and 11 RE (red) and LE (blue) following brief contralateral stimulation (1 kHz, 1.1 s duration at 100 dB SPL). Participant 11 LE admittance recordings could not be obtained. Each

admittance and pressure measurement are superimposed on each other. The thicker blue and red lines represent the mean average. C) Air pressure and admittance recordings for participants 09, 10 and 11 RE (red) and LE (blue) following sustained contralateral auditory stimulation (1 kHz, 10s duration at 100 dB SPL). Participant 11 LE admittance recordings could not be obtained.

FIGURE 8. Air pressure and admittance recordings for participants 09, 10 and 11 RE (red) and LE (blue) following middle ear muscle voluntary contractions (vMEMC). The thicker blue and red lines represent the mean average. Admittance was measured at 0 daPa (dark red and blue line) and -50 daPa external ear canal pressure (light red and blue).

FIGURE 9. Air pressure and admittance recordings for participants 09, 10 and 11 RE (red) and LE (blue) following contralateral auditory stimulation (1 kHz, 10s duration at 100 dB SPL) with vMEMC every 3 seconds. Admittance was measured at 0 daPa (dark red and blue line) and -50 daPa EAC pressure (light red and blue). Simultaneous sMEMC and vMEMC admittance recordings could not be obtained in participant 11 LE.

FIGURE 10. A) Air conduction thresholds (ACT) measured with and without VMEMC (w/o) contraction for both ears (RE & LE) of participants 09, 10 and 11. B) The threshold shift caused by vMEMC contraction is shown for each participant's ear in the bottom panel. The star represents the psychoacoustic characteristics (pitch and loudness) of the fluttering sound emitted during vMEMC for each ear.

Table I. Characteristics of all the ears tested.

#Ear	Laterality	#Participant	Group	Static tympanometry				Stapedial reflex thresholds (dB HL)				Hearing thresholds (dB HL)			
				TPP (daPa)	ECV (ml)	SA (mmho)	Type	0.5 kHz	1 kHz	2 kHz	4 kHz	BBN	PTA low	PTA mid	PTA high
1	RE	1	Ctrl	-10	1.1	0.5	A	85	90	85	80	65	8	11	2
2	RE	2	Ctrl	-15	0.9	0.6	A	80	85	90	95	65	2	4	0
3	RE	3	Ctrl	-7	1	0.5	A	85	85	85	85	60	3	3	2
4	RE	4	Ctrl	-8	0.7	0.4	A	95	95	95	85	80	7	4	2
5	RE	5	Ctrl	1	1.1	0.7	A	95	100	95	100	75	13	8	3
6	RE	6	Ctrl	5	1.4	0.5	A	95	95	95	105	85	7	4	-8
7	RE	7	Ctrl	-11	0.6	0.2	AS	85	80	85	85	60	10	7	-5
8	LE	7	Ctrl	-7	0.7	0.1	AS	80	90	95	95	55	11	7	-5
9	RE	8	Ctrl	-44	1.2	0.7	A	95	95	90	95	80	10	2	0
10	LE	8	Ctrl	-34	1.2	0.9	A	92	90	90	92	80	5	8	-1
11	RE	9	vMEMC	-7	0.7	0.4	A	95	95	95	90	70	10	11	0
12	LE	9	vMEMC	-47	0.6	0.6	A	100	100	95	95	80	17	13	7
13	RE	10	vMEMC	-12	0.7	0.3	A	85	85	95	95	65	0	1	0
14	LE	10	vMEMC	-7	0.6	0.3	AS	80	85	95	95	70	3	4	-2
15	RE	11	vMEMC	-23	0.9	2.8	AD	90	85	80	85	NT	8	9	5
16	LE	11	vMEMC	-11	0.7	6.9	AD	NO	NO	NO	NO	NO	10	14	5
17	RE	12	vMEMC	NT	NT	NT	NA	NT	NT	NT	NT	NT	10	0	2
18	LE	12	vMEMC	NT	NT	NT	NA	NT	NT	NT	NT	NT	5	5	-2

RE = Right ear, LE = Left ear, Ctrl = Control, vMEMC = Voluntary Middle Ear Muscle Contraction, NT = Not tested, NO = Tested but not obtained, PTA = Pure tone average, low = thresholds average at 0.25, 0.5, 0.75 kHz, mid = thresholds average at 1, 1.5, 2, 3 kHz, high = thresholds average at 4, 6, 8 kHz

Figure 1.

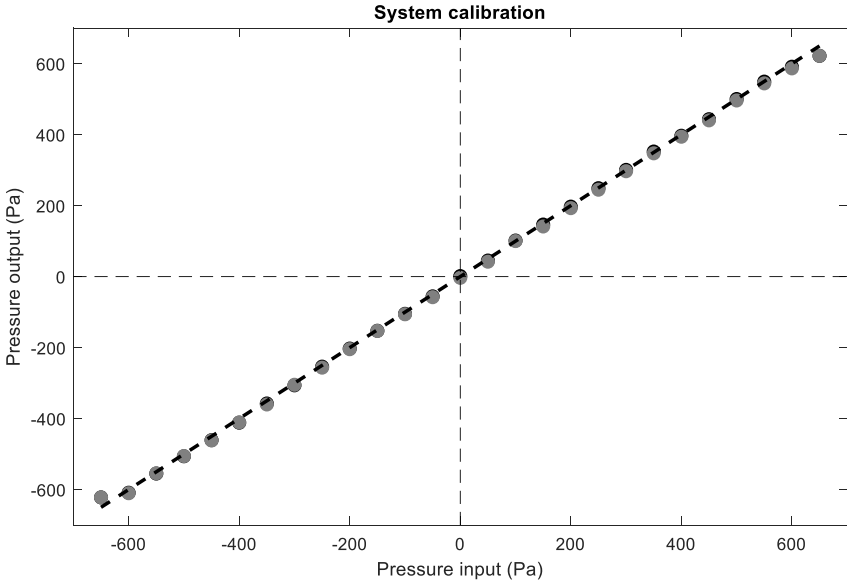
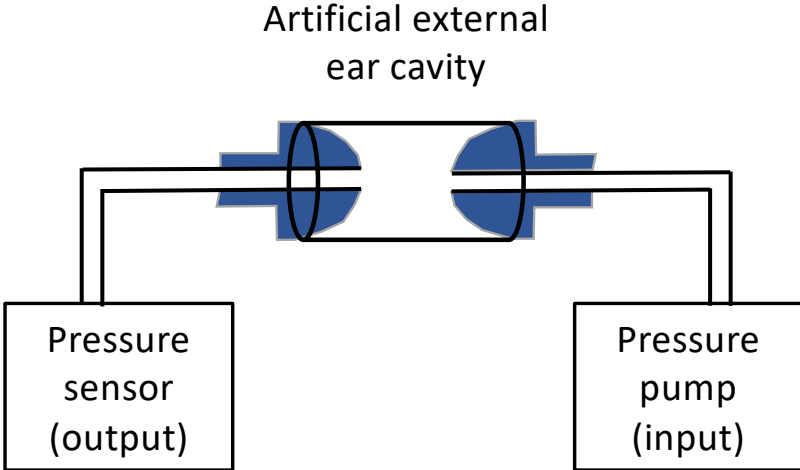
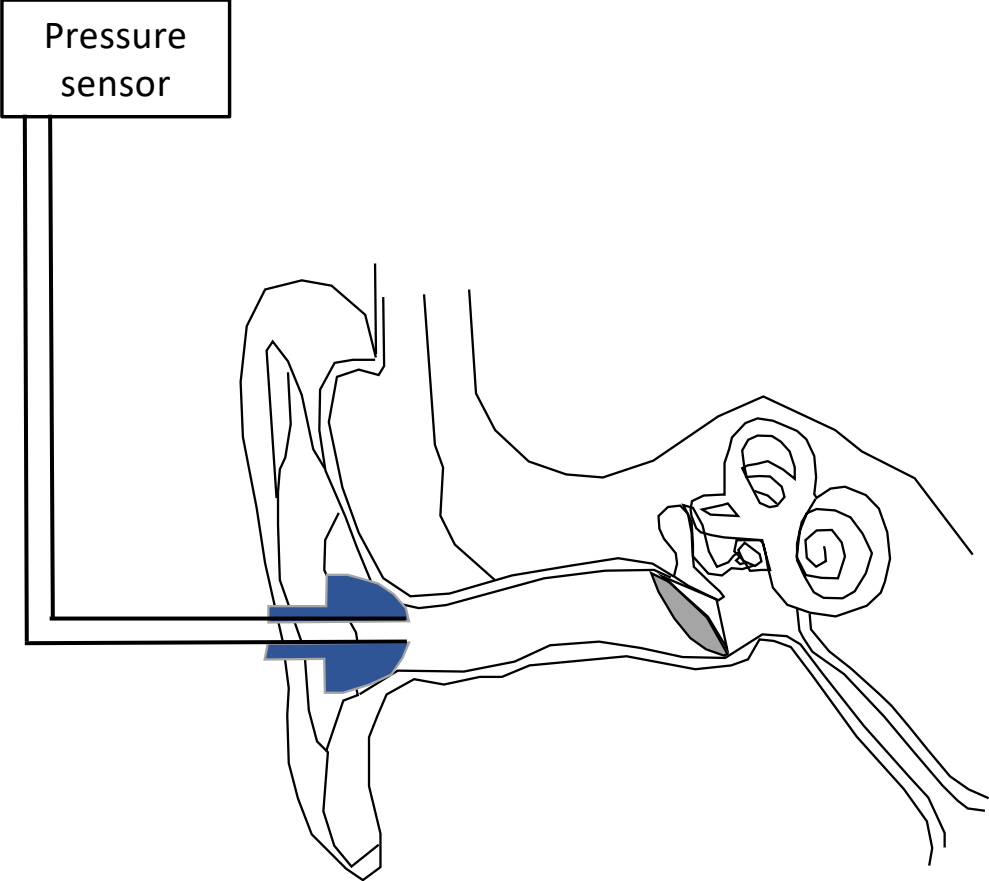


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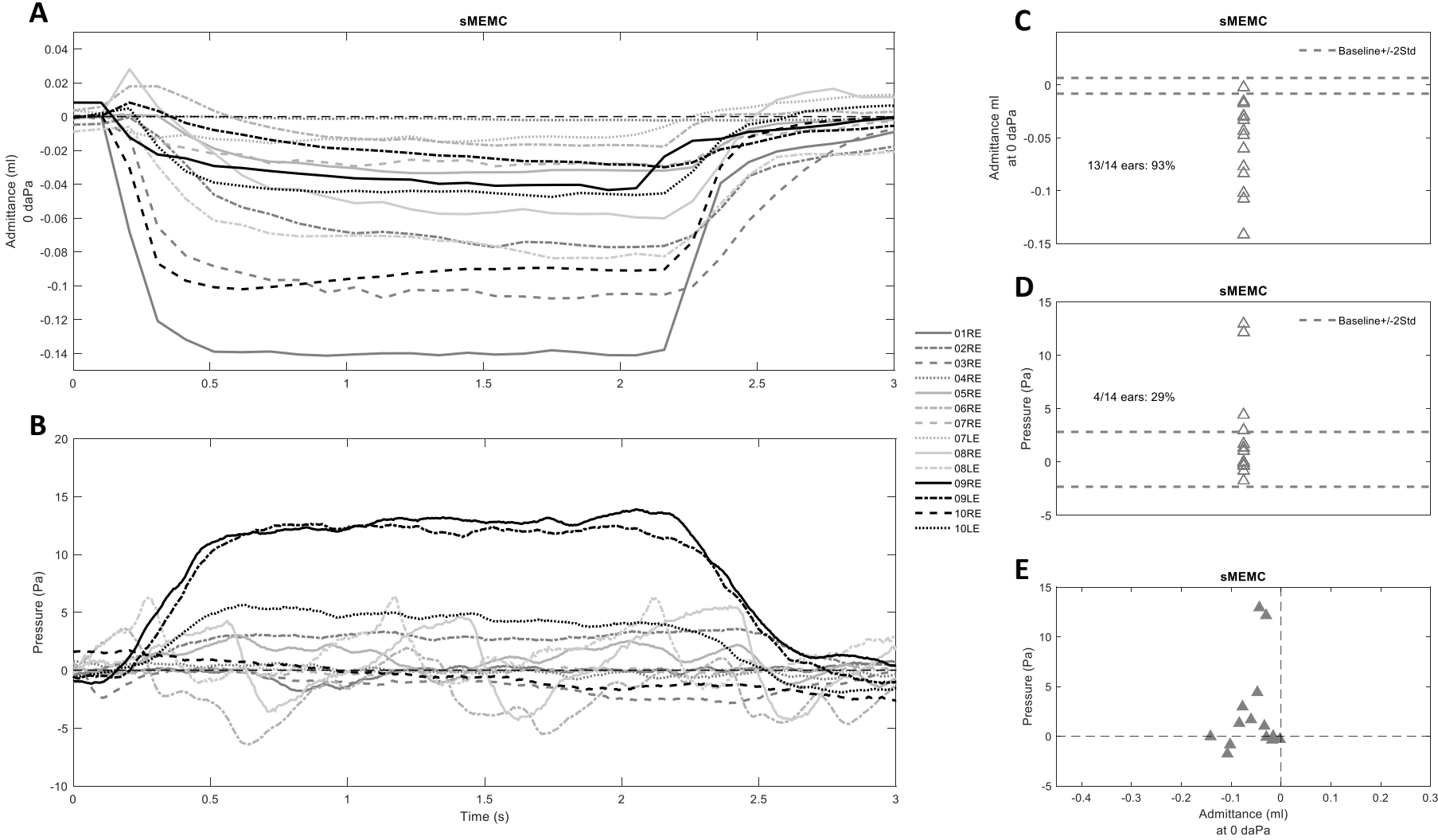


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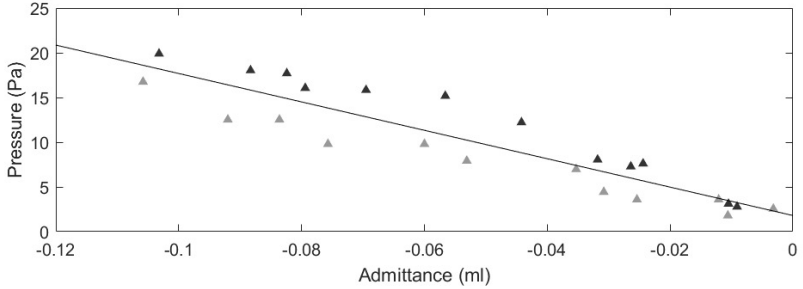
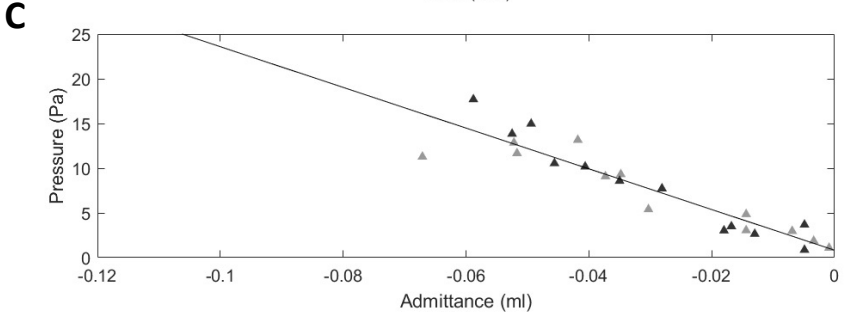
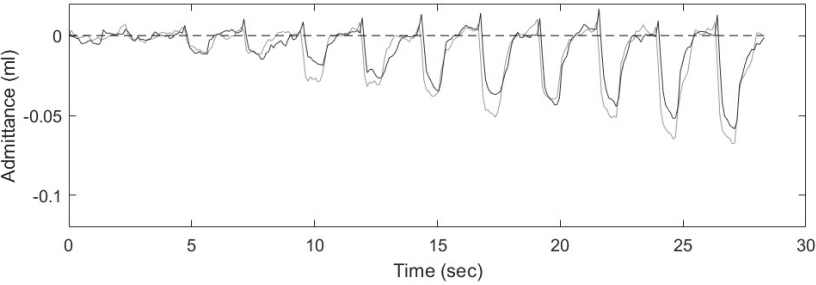
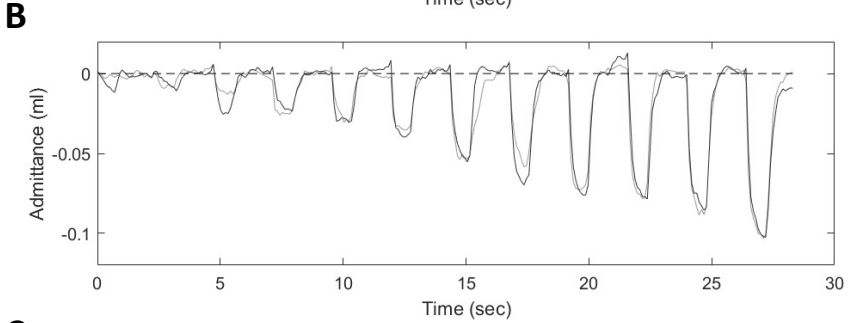
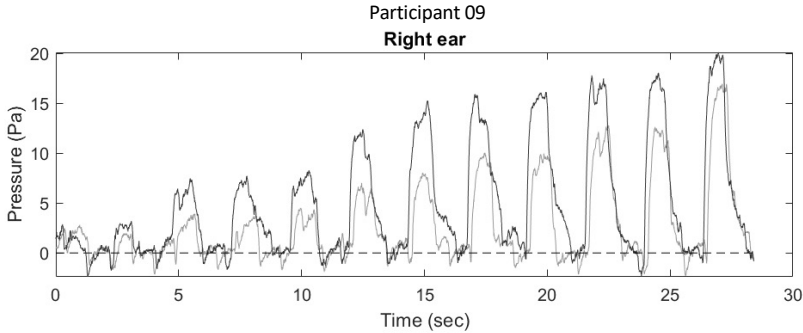
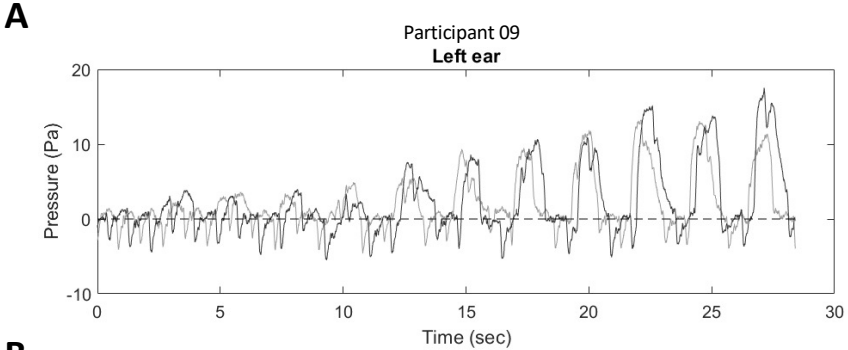


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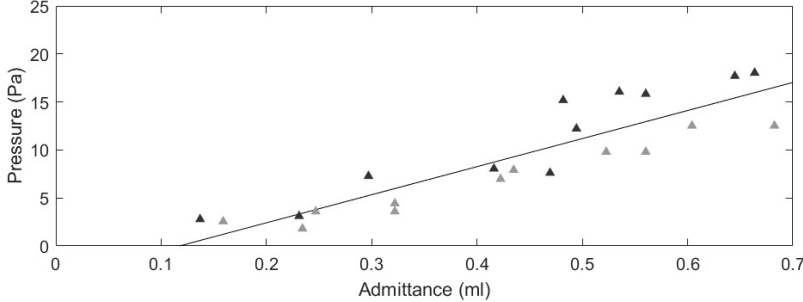
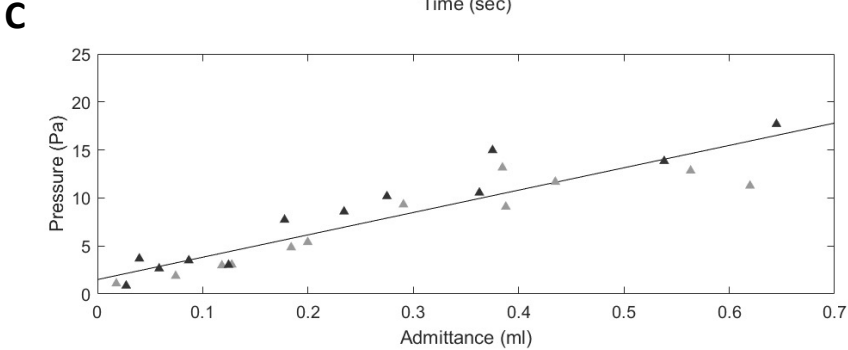
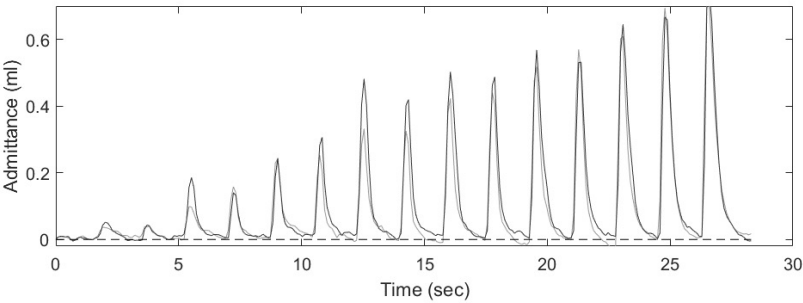
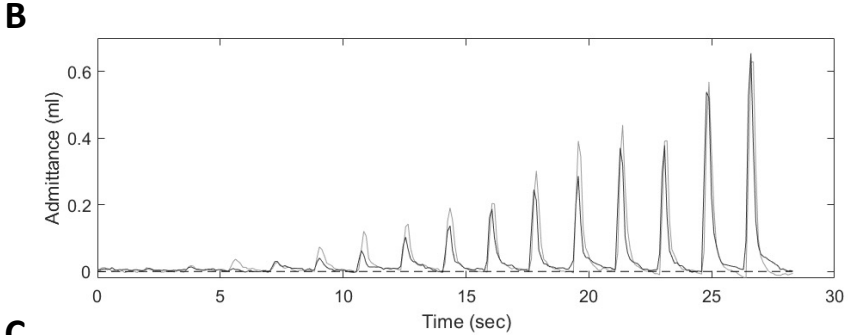
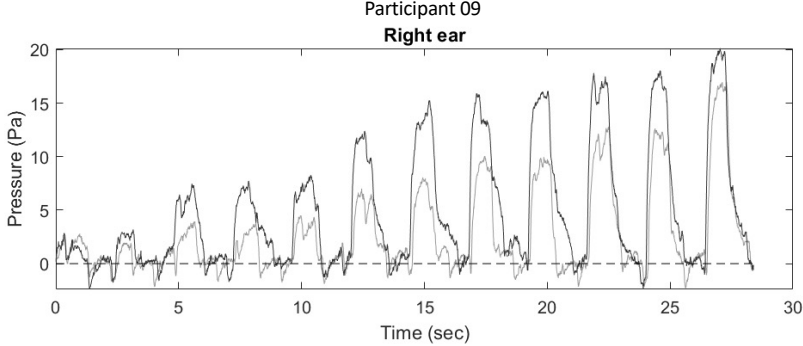
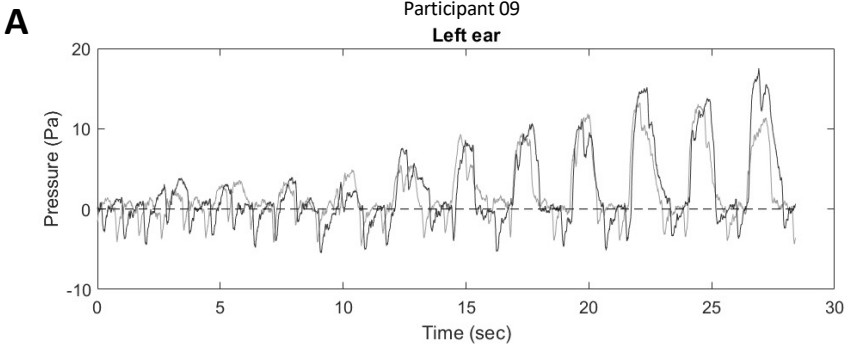




Figure 5.

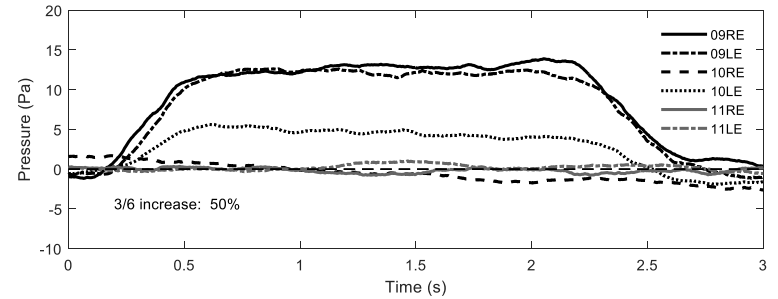
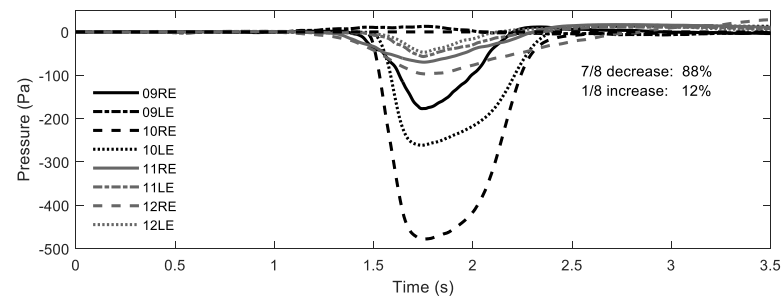
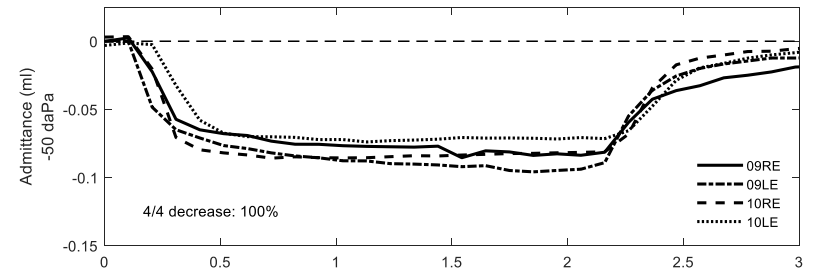
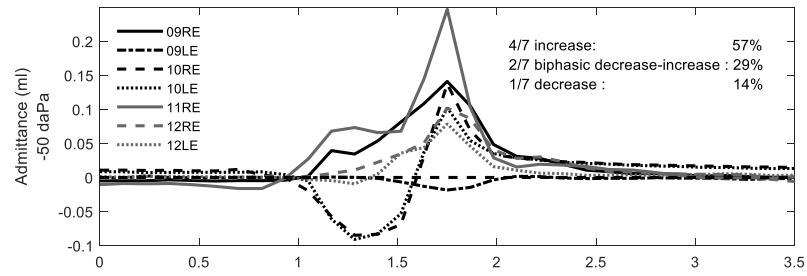
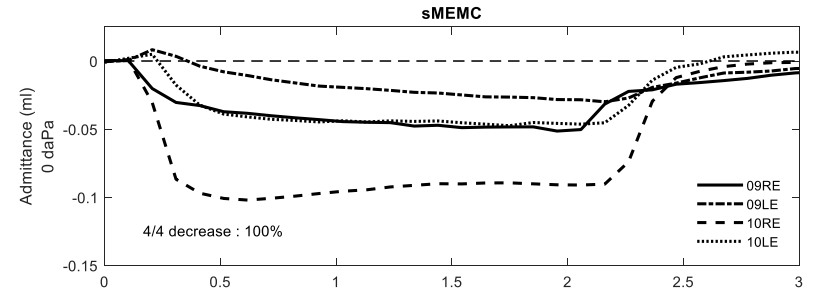
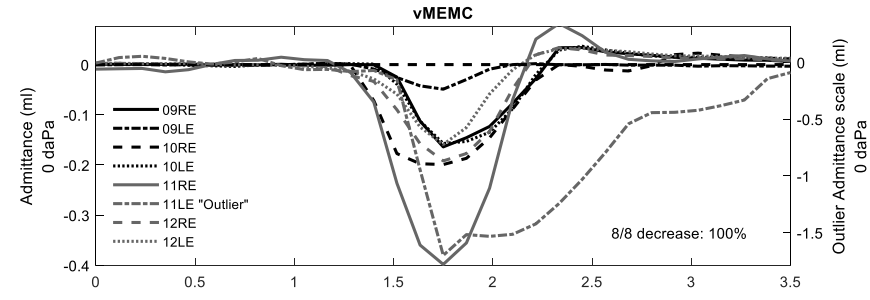


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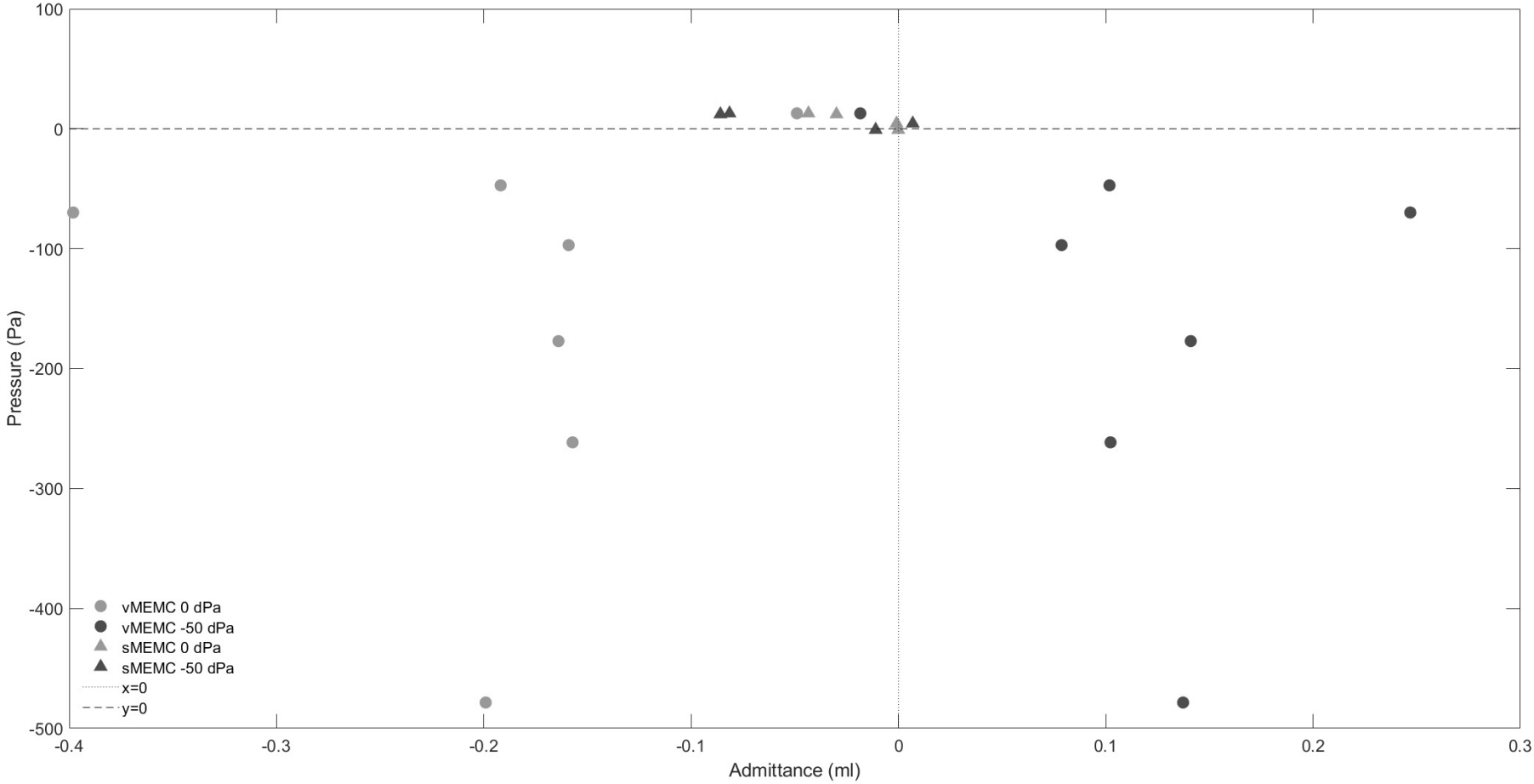


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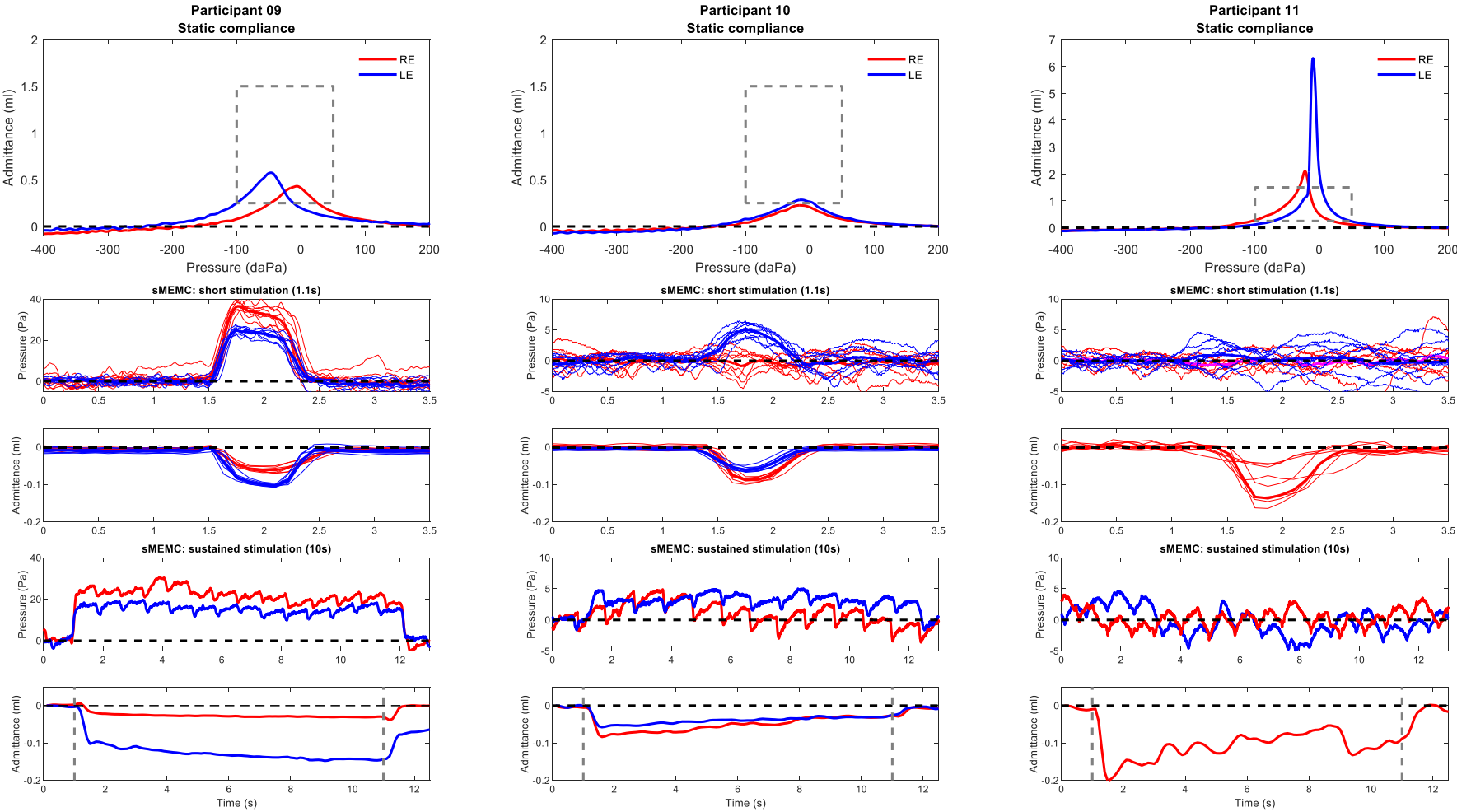


Figure 8.

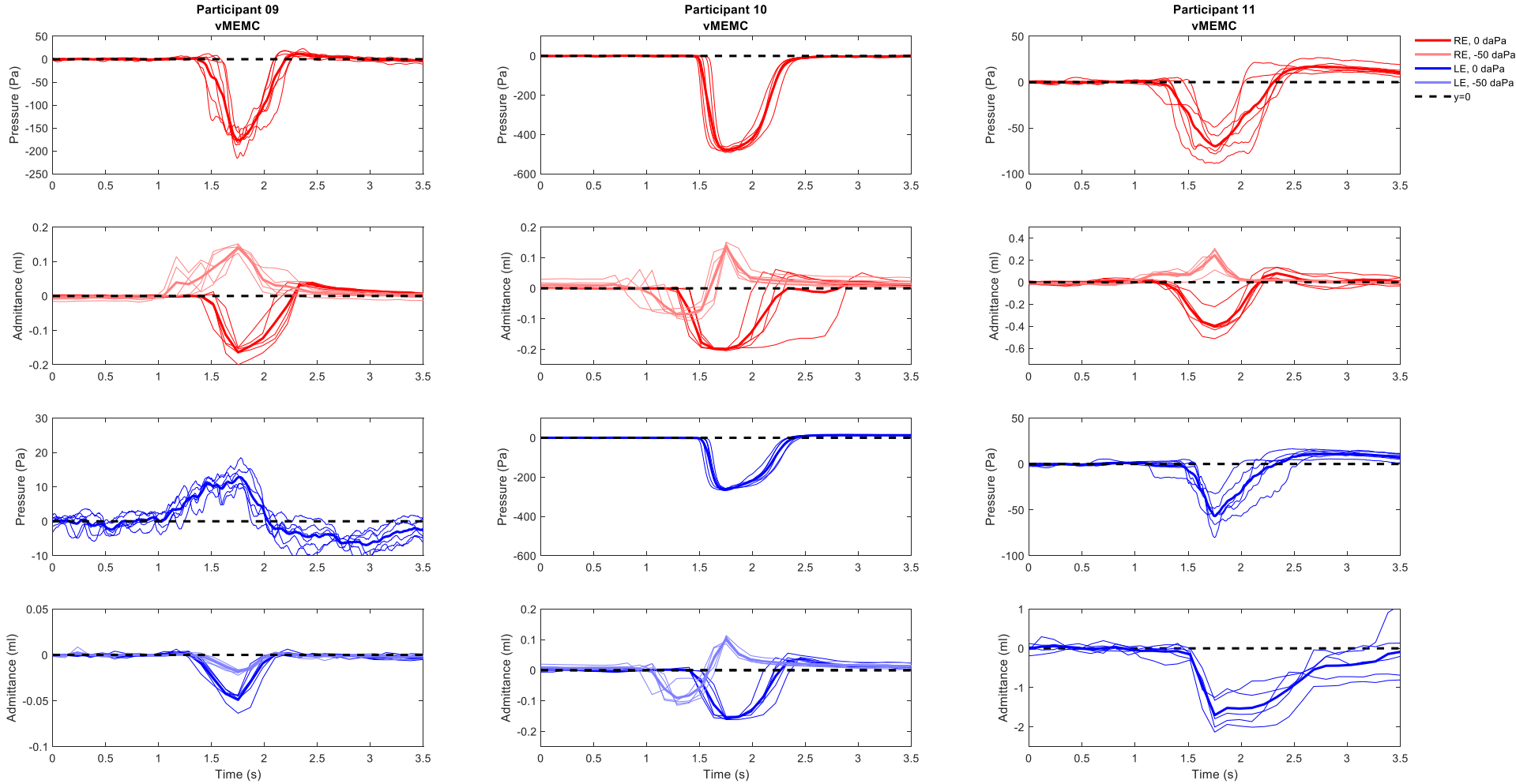


Figure 9.

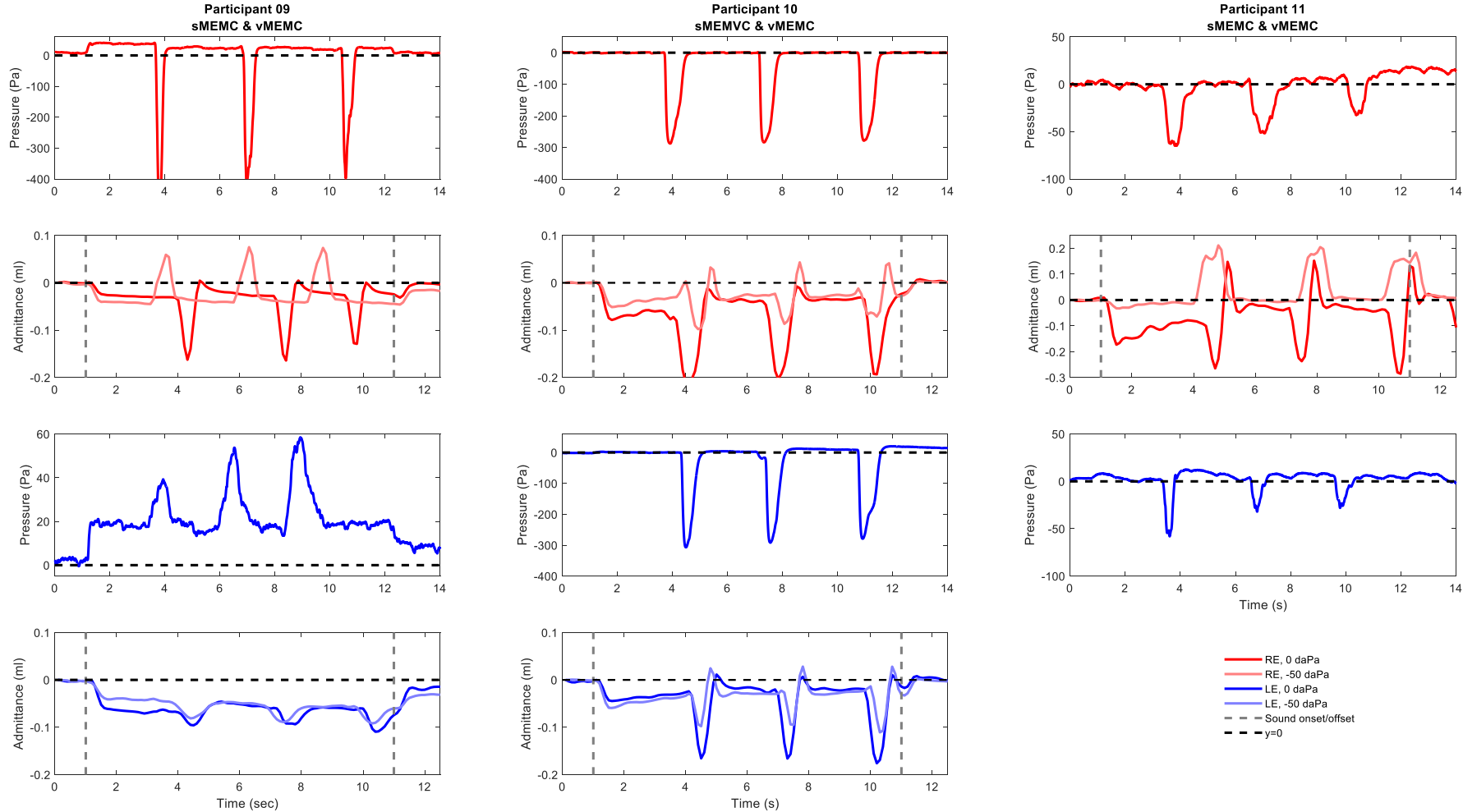


Figure 10.

