The occlusion effect and its reduction

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The occlusion effect is a major problem for hearing aid wearers. If unsolved, it makes their own voice sound un-natural, and if solved it can severely limit the gain that can be achieved without feedback oscillation. It appears to be caused by the jaw vibrating to a much greater magnitude than the other bones of the skull, because of the much lower weight of the jaw. Jaw vibration then causes one wall of the ear canal to vibrate with respect to the opposing wall. A new solution to the occlusion effect is to add a second, inward-looking microphone to a hearing aid. This microphone senses the occlusion-induced sound pressure within the ear canal, inverts it, and feeds it back to the receiver to partially cancel the occlusion-induced sound pressure. The processing operates according to well-established rules of negative feedback, an important component of which is filtering to ensure that gain around the loop is less than unity for any frequency at which phase shifts cause the feedback to be positive. Occlusion reduction of 15 dB in the target frequency range is achievable, and as this is the same magnitude as the occlusion effect for the average hearing aid wearer, active occlusion reduction can completely remove occlusion for the average wearer, despite having little or no venting. Because the active occlusion reduction also cancels any sound entering the ear canal via any small vent or leakage path, the processing allows electronic amplification to be provided, even with negative gains if desired. This substantially widens the range of frequencies over which directional microphones and adaptive noise suppression can be effective

INTRODUCTION

The occlusion effect refers to the sensation of having a blocked ear that many people experience while wearing a hearing aid or earmold in the ear canal. There are at least two physical causes of this sensation. The first is the physical pressure arising from contact of the hearing aid against the walls of the ear canal. The second is the abnormally high sound pressure that occurs within the space between the medial end of the hearing aid and the ear drum when the person talks or chews, or even walks. It is very common for people to complain about the sound quality of their own voice while wearing hearing aids (Dillon *et al.* 1999). It is also common for people to complain about the indirect result of a clinician trying to improve the clients' perception of their own voice by having an open vent path.

MECHANISM OF OCCLUSION-INDUCED SOUND

It is widely accepted that the extra sound pressure is caused by vibration of soft tissue

in the ear canal walls excited by bone-conducted vibrations in the skull (Carle, 2002; Dillon, 2001; Pirzanski, 1998; Mueller, 1996; Mackenzie, 1989; and Killion, 1988). With nothing in the ear, the acoustic impedance seen by the vibrating wall is low, so the sound pressure generated is also low. With a hearing aid blocking the exit from the ear canal, however, the acoustic impedance seen by the vibrating wall(s) is much higher, and consequently, so too is the sound pressure in the ear canal. These occlusion-induced sounds are most noticeable during the utterance of the vowels /i/ or /u/, both of which have low first-formant energy and which are uttered with a relatively closed jaw position.

While there is no reason to doubt this basic explanation, it does not give a clear picture of just how bone conduction causes the ear canal walls to vibrate, or how the vibrating walls then cause a sound pressure within the enclosed space. The first part of this paper proposes a more detailed mechanism and reports measurements on two subjects that support this mechanism.

In older children and adults, the temporal and mastoid bones are rigidly attached to the parietal and occipital bones and to the maxilla. The combined structure has a relatively high acoustic mass. On the other hand, the mandible is loosely attached to the remaining bones, and its mass is approximately four times smaller than the combined mass of the remaining bones of the skull (Slager, 1997).



Fig. 1: Lateral view of the skull, outlining the major bone structures surrounding the ear canal.

Above the resonant frequency of a structure, the amount of vibration is controlled by the mass of the structure. For every doubling of mass, there is a 6 dB decrease in the level of vibration (Beranek, 1986). Therefore, based on the mass ratio between the different bone structures surrounding the ear canal, and the low resonant frequency of the skull (Hakansson *et al.*, 1986), there should be at least 12 dB more vibration in the mandible than in the temporal bones. This figure of 12 dB assumes that the vocal tract sound pressure fluctuations provide an equal force to the mandible and to the remain-

ing bones, each of which is free-standing. The resulting difference in vibration amplitude is, however, likely to be greater than 12 dB because the temporal/occipital/parietal bones are more closely coupled to other body masses and damping mechanisms (the brain, the neck tissues, the spine) than is the mandible.

As a result of this difference in bone vibration, there should also be at least 12 dB more excitation of the portion of the canal wall closely coupled to the mandible (the inferior and anterior walls of the outer half of the ear canal) than the remaining walls which are more closely coupled to the mastoid and temporal bones. If one portion of the wall moves much more than the other, there will be a net fluctuation of volume within the canal, giving rise to a sound pressure within the canal, especially when the ear canal is blocked at any point lateral to the vibrating wall. Hence, from this theoretical perspective, the bone-conducted component that creates the occlusion effect primarily should be the result of mandible-transmitted vibrations. The following experiment was carried out to confirm that mandible vibration does indeed dominate.

Method

Two subjects were fitted with vibration sensor microphones BU-1771, or low-mass accelerometers, with matched sensitivity. The devices were attached to a subject's skin external to the mastoid and upper portion of the mandible, using adhesive dressing tape. The devices were oriented so that the direction of maximum sensitivity directly faced the centre of the ear canal entrance.

During the experiment, the subjects were asked to sustain the utterance of the vowel /i/ while the outputs from the microphones were recorded and stored in a personal computer. This process was repeated a number of times; the bone conduction sensing devices were removed and re-positioned at slightly different locations (but within a few mm of their original position) to produce multiple independent estimates of the same underlying bone vibrations.

The data were analysed by taking the Discrete Fourier Transform (DFT) of an 800 millisecond segment of the recorded bone-conducted vibrations. The level and phase difference between the two sensing locations were then computed using Equation 1. These computed measures were weighted by ideal rectangular windows to produced 12 logarithmically spaced centre frequencies, ranging from 100 Hz to 1000 Hz, and then each windowed response was averaged across the four recordings obtained from slightly different placements of the microphones.

$$LevelDifference = abs(\frac{Mandible_{DFT}}{Mastoid_{DFT}})$$
(Eq. 1)

Results

The average level differences between the relative sound vibrations sensed at the mandible and mastoid bone locations are shown in Fig. 2. The mandible is vibrating much more intensely, by 15 to 20 dB in the lower frequencies, than the mastoid bone, as anticipated.



Fig. 2: Sound vibration level difference between the mandible and mastoid bone, for a continuous enunciation of 800 ms of the vowel /i/.

Discussion

The results indicate that the mandible vibrates much more strongly than the mastoid process. We can therefore infer that the anterior and inferior wall of the outer half (the cartilaginous portion) of the ear canal will vibrate much more strongly than the remaining walls, which are in close contact with the mastoid process. Consequently, if part of the canal wall vibrates relative to the opposing surface of the wall, the volume of the enclosed space will vary in time, and this fluctuating volume will directly produce a sound pressure in the ear-canal. That is, we are arguing that the mandible and soft tissue that couples the mandible to the ear canal is the most significant source of the own-voice amplified sound noticeable in blocked ears.

The reader can perform three simple experiments to confirm this interpretation: First, with the little finger in the ear canal, open and close the jaw. Movement of the anterior and inferior wall, but not of the posterior or superior wall, is evident. Second, with the little finger still in the ear canal, alternately say the sounds /i/ (as in "he") and /a/ (as in "far"). When /i/ is voiced there is a higher sound pressure, and greater pressure of the mobile portions of the canal wall against the fingers, than when /a/ is sounded. Third, and most directly, close the ear canal by pushing the tragus inwards, and vocalise the sound /i/. While still vocalising, grasp the jaw firmly with the other hand (which damps its vibrations), and note the reduction in loudness. Conversely, no reduction in loudness occurs when the bones of the skull are grasped. A jaw clamp, however, is not a convenient solution to the occlusion problem.

TRADITIONAL SOLUTIONS AND THEIR LIMITATIONS

The traditional solution to the occlusion effect is to open the ear canal with a vent. If the vent has a sufficiently large diameter and/or a sufficiently short length, then its impedance will be much less than the impedance of the small cavity of air trapped

between the hearing aid and the eardrum. The sound pressure generated in the ear canal by the vibrating wall of the ear canal equals the volume velocity of the canal wall (i.e. its velocity times the area vibrating) times the impedance that the vibrating wall sees. Consequently, a sufficiently low-impedance vent lowers the impedance seen by the vibrating wall, which lowers the sound pressure generated within the ear canal. In practice, a 2 mm diameter vent will significantly decrease the occlusion-induced sound, but a 3 mm vent is needed to reduce it to insignificant levels. The magnitude of the occlusion effect (the increase in SPL relative to nothing in the ear) for vents of different diameters is shown in Fig. 3. An open fitting is achieved by putting only a tube into the ear canal. The space around the tube has the same effect as a very large vent. As can be seen from Fig. 3, the occlusion effect is entirely removed.



Fig. 3: The mean increase in SPL (relative to no earmold) in the ear canal for 10 subjects, as they talked while wearing earmolds with vents of different sizes (from Dillon, 2001, by permission).

There are two disadvantages to solving the occlusion problem with a vent (or equivalently an open fitting). The first is that along with the occlusion-induced sound, amplified high-frequency sound travels out through the vent. This leakage creates a feedback path that limits the amount of gain that can be achieved, even when the hearing aid contains a feedback cancellation scheme. The second problem is more subtle. The vent also provides a low-impedance escape route for amplified low-frequency sound, such that a negative gain is likely for "amplified" low-frequency sounds. The negative gain is not itself a problem, as low-frequency sound entering the ear canal through the vent provides a gain of 0 dB. However, the dominance of vent-transmitted sound over the low-frequency range means that the benefits of directional microphones and adaptive noise reduction systems are limited to the high- and possibly midfrequency range, thus limiting their overall benefit. We will return to this point at the end of the paper.

The second solution to the occlusion effect is to insert the hearing aid past the cartilaginous section of the canal and into the bony section. As a result, the vibrating cartilaginous walls make contact with the hearing aid rather than compress and expand the residual volume of the ear canal. Consequently, there is no occlusion-induced sound pressure. The only disadvantage of this solution is that the skin in the bony canal is thin and sensitive. Unless the hearing aid material is very soft and compliant, discomfort is likely. Unfortunately, very soft materials degrade quickly.

ACTIVE OCCLUSION CANCELLATION

The remainder of this paper describes a solution to the occlusion problem that is more practical than a jaw clamp, does not limit the usable gain of the hearing aid, and does not cause discomfort or physically degrade. It needs neither a physical barrier damping the vibration of the cartilaginous tissue in the ear canal walls, nor an open path to the air outside the ear, but rather is based on cancellation of sound within the ear canal. The cancellation is based on sensing the mandible-induced sound pressure directly inside the ear canal and applying principles of active noise control, or active feedback control, to reduce the level of that sound pressure.

Basic principle of active occlusion cancelling

Active occlusion cancelling can be accomplished with the system shown in Fig. 4.



Fig. 4: Block diagram of a hearing aid incorporating an active occlusion system, inside an ear canal.

The external microphone (on the left), hearing aid amplifier (H/A) and receiver, are the same as in any other hearing aid, and the remaining components are those needed for occlusion cancelling. The internal microphone on the right senses the sound pressure in the ear canal, and its output is filtered by block *B*. The output of filter *B* is subtracted from the main signal, and the combination is filtered by block *A*, before being passed to the receiver. Block *C* is a compensation filter whose role will be explained later. The chain of components comprising internal microphone, filter *B*, subtractor, filter *A*, receiver, and ear canal volume form a negative feedback loop. *A* negative feedback loop has a predictable effect of reducing any signal injected anywhere into the loop, such as the occlusion signal injected into the ear canal. The multiplicative effect, *E*, on sound pressure, is shown in Equation 2. This is an easily-derived equation that is the basis of feedback control theory (Benjamin, 1982; Hitay, 1999).

$$E = \frac{1}{1 + |TAB| \cos(\theta^{\circ})}$$
 (Eq. 2)

where, for a given frequency, T is the transducer gain response from the receiver input to the internal microphone output, which is affected by the ear canal and middle-ear impedance, and θ° is the phase response around the open loop.

One of the main concerns in systems with feedback loops is that the feedback loop may become unstable. As the denominator approaches zero, the absolute value of E increases towards infinity. Very large closed-loop gains can occur when a sufficiently large open-loop gain combines with an adverse phase around the loop, which occurs when $\cos(\theta^{\circ})$ becomes negative. Phase shift around the loop is inevitable at very low frequencies (because of leakage within the transducer and around the earmold) and at mid and high-frequencies (because of resonances in the receiver and microphone and the tubing and ear cavity to which they are connected). Instability is avoided by choosing *A* and *B* so that the magnitude of the gain around the loop (*TAB*) is significantly greater than unity only at those frequencies where there is minimal phase shift around the loop. It is evident from Fig. 3 that the occlusion-induced SPL is greatest around 300 Hz, and that the problem region extends from around 100 Hz to 500 Hz. Fortunately, for practical microphones and receivers, phase shift around the loop is minimal over this frequency range. Filters *A* and *B* can therefore be designed to have maximum gain within this frequency range.

As well as reducing the level of the occlusion-induced sound, the feedback loop will reduce the signal and sound level of hearing aid amplified sounds. However, this effect can be corrected by a pre-compensation filter, *C*. If the response of the pre-compensation filter is chosen to be that shown in Equation 3, then the resulting frequency response from the external microphone to the receiver output is no different from when just the regular hearing aid components are present.

$$C = \frac{1}{A} \left(1 + |TAB| \cos(\theta) \right)$$
 (Eq. 3)

Simulations and predicted responses

To enable appropriate filter responses A, B and C to be derived, and occlusion-reduction performance to be predicted, the combined response of a Knowles EH hearing aid receiver and a Knowles FG microphone (as the internal microphone), both connecting to a Zwislocki coupler by short tubes, were simulated using electroacoustic models of each component. These transducers were chosen because of their low degree of phase shift within the frequency range of interest. The predicted responses agreed closely with the measured performance of the same transducers connected to a Zwislocki coupler. The simulated response was then used as T in Equation 2, along with the mathematical transfer functions appropriate to filters A and B, to predict the degree of occlusion reduction that is possible for any particular overall loop gain. The overall loop gain must be sufficient to achieve the desired degree of occlusion reduction, but not so large that instability occurs at any frequency.

The simulated response of the predicted occlusion reduction, following Equation 2 is shown in Fig. 5. For this simulation, the occlusion reduction is predicted to be -18

dB at 200 Hz, and to extend over a range of low frequencies, including the frequency range in which the fundamental frequencies and first formant frequencies of closed vowels usually lie (from 80 Hz to 400 Hz).



Fig. 5: Predicted effective sound pressure reduction in the ear canal |E|, following Equation 2.

In contrast to the occlusion reduction achieved in the low frequencies, amplification is observed at very low and high frequencies, in the region of 10 Hz and 1.3 kHz, respectively. This amplification occurs because the phase shift around the loop causes positive feedback to occur at these frequencies. A greater degree of occlusion reduction could be achieved by increasing the gain at any point within the loop. However, significantly increased gain will eventually produce an instability (i.e. oscillation) in one or both of the 10 Hz or 1.3 kHz regions.

Fortunately, although occlusion reduction appears to be achievable over only a restricted range of frequencies, the range achieved is reasonably well matched to the range of frequencies over which occlusion occurs. Furthermore, across this frequency range, the degree of occlusion reduction achievable is similar to the degree of occlusion that typically occurs in unvented hearing aids, as shown in Fig. 3. Consequently, active occlusion cancelling should be able to completely remove the occlusion effect, with no venting, for the average subject.

Although not shown in this paper, similar final performance results if the hearing aid has leakage or a vent. As the size of the vent increases, phase shifts around the loop increase and the low frequency gain of the loop decreases. Both of these limit the maximum achievable gain around the loop in the target frequency region, so the effectiveness of active occlusion reduction decreases as the vent (or leak) diameter increases. Fortunately, the vent also directly decreases the occlusion-induced SPL, so the active mechanism does not need to be as effective. The end result is that the overall occlusion reduction achievable in the target frequency region changes is not much affected by the vent size.

Measured performance

The effectiveness of a prototype active occlusion system was evaluated by measuring the degree of occlusion reduction possible, and by obtaining subjects' preferences for sound quality with and without the system active.

Twelve subjects with normal hearing were bilaterally fitted with specially designed in-the-ear (ITE) unvented ear shells containing the components shown in Fig 4. The receiver and internal microphone were coupled to the ear canal by tubes and wax-protection devices. The transducers were connected with a 1 m cable to external electronic circuitry. The electronic circuitry comprised of a hearing aid amplifier and a custom-designed analog active occlusion cancelling system. The circuit could be switched between active (feedback loop operating) and passive (feedback loop disabled) modes of operation. A volume control and feedback overall gain control were also available. It was also possible to probe the voltage signal from the external and in-the-ear microphone outputs and connect them to a two channel network signal analyser which was programmed to perform 1/12 octave linear spectral averaging of the recorded signals. A third microphone was connected to a sound level meter and positioned directly in front of the participant.

During the sound recordings, each subject was asked to read an arbitrary paragraph at a normal vocal effort which resulted in 55 - 70 dB SPL, recorded approximately 30 cm away from the mouth, for at least 5 to 8 minutes. Both the external and the in-theear canal microphone were recorded simultaneously. Throughout the recordings the hearing aid amplifier was muted.

Following the recording the subjects were asked about their subjective reaction to the sound of their own voices. In order to form their opinion the subjects were allow to switch freely between the active and passive modes of operation. Subjects were not advised of the mode of operation corresponding to a given location of the control switch. Throughout the subjective assessments, the hearing aid amplifier was adjusted to yield 0 dB ear-simulator gain.

Results

Figure 6 shows the extent to which the sound pressure inside the ear canal on one subject exceeds the long-term sound pressure level recorded immediately outside the ear, with active occlusion disabled, and with it enabled. Without active occlusion there is a 15 dB increase in signal level, and without it, speech levels inside the ear canal are similar to those outside. The difference between active cancelling on and off, i.e. the degree of active occlusion reduction, is also shown in the same figure.



Fig. 6: Long terms sound pressure level in the occluded ear relative to sound pressure level immediately outside the ear for an occluded hearing aid (upper thick line), and with active occlusion cancelling operating (thin line). The degree of occlusion reduction is shown in the lower thick line.



Fig. 7: Measured average sound level reduction in the completely blocked ear canal (solid line) with one standard deviation (faded line) based on sample populations of 22 ears.

Figure 7 shows the amount of occlusion reduction averaged over 22 test ears (measurements could not be obtained on one ear for two of the subjects). Occlusion reduction ranges from 50 Hz to 900 kHz, with the maximum reduction of 15 dB occurring at 300 Hz. There is an increase in level-induced sound below 50 Hz and above 1 kHz.

Ten out of twelve subjects preferred the quality of their own voice when the active occlusion reduction was enabled. One subject described a sensation of pressure during jaw movement. We hypothesize that this subject perceived the increase in sound

pressure caused by the "cancellation" at very low frequencies, in the vicinity of 10 Hz or perhaps lower. Physical measurement confirmed that this subject had a greater than average increase in SPL in this very low frequency range when the occlusion cancelling was activated. At these extreme frequencies, the negative feedback loop is behaving as a positive feedback loop, due to phase shifts around the loop.

DISCUSSION

The first part of this paper presents a probable mechanism by which the aid wearer's own voice causes excessive low-frequency sound pressure in the ear canal, which leads to an unacceptable voice quality for many hearing aid wearers. The second part of the paper presents an active means of removing the excess sound pressure, irrespective of the mechanism by which the occlusion sound is first created,. Our simulations and evaluation on 12 subjects indicate that it is possible for a negative feedback loop to largely eliminate the occlusion-induced sound, without the loop becoming unstable at any frequency.

A disadvantage of the processing, sufficient to cause at least one subject to prefer the occluded sound, is that the negative feedback loop can create additional sound pressure for very low frequencies, in the infra-sound frequency range. These frequencies can occur during jaw movement, such as when chewing. An easy solution to this problem is to fit the active occlusion reduction hearing aid with a small vent, such as 1 mm diameter, which avoids creating these very low-frequency pressure fluctuations in the first place.

The active occlusion system was invented so that the occlusion effect could be avoided without requiring a large vent, and the consequent leakage and limitation in gain that a large vent causes. We have realised, however, that the system offers additional advantages that may be even more important. As explained earlier, hearing aids with large vents deliver unprocessed, acoustically transmitted sound to the aid wearer in the lowfrequency range, which may extend up to 1500 Hz in open canal hearing aids. This means that directional microphones and adaptive noise suppression systems are effective only over the high-, and perhaps mid-frequency part of the speech range, and this excludes the frequency range where noise is usually most intense. The active occlusion cancelling system actually reduces the level of sound reaching the ear canal by any path, including sound transmitted in through the vent. Consequently the system will cause low-frequency sounds transmitted in through the vent to be delivered with a negative insertion gain - as low as -15 dB. This means that the patient can instead be provided with electronically "amplified" sound with a gain of 0 dB, something that is unachievable with conventional hearing aids. If the hearing aid has a directional microphone, or an active noise reduction system then this processing will also be operating over the low-frequency range, or as much of that range as directional microphones are capable of operating over without excessive internal noise.

We think these advantages are very significant now, and will become even more significant when hearing aid signal processing combines the signals picked up from the two

sides of the head to produce even more effective directionality and noise suppression. The advantages seem sufficient to outweigh the increase in complexity associated with an additional microphone in the hearing aid. The internal microphone can also be put to additional uses, such as sensing when the aid wearer is talking, or acting as a pick-up point isolated from external noise for devices of the future where the hearing aid also functions as an interface to telephones and other electronic devices.

Finally, electronic transmission of low frequency sounds into the ear canal means that low- and high-frequency sounds arrive with the same delay, instead of milliseconds apart as happens with current hearing aids. This enables a smoother frequency response to be provided in the transition frequency region, and may lead to more consistent inter-aural time differences to be delivered to the binaural system.

Further research is needed to establish the magnitude of the benefit of having directionality and noise suppression extend over a wider frequency range, and to examine whether there is any patient benefit associated with having a consistent time delay over a much wider frequency range.

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