Drill-induced hearing loss in the nonoperated ear

MELVILLE J. DA CRUZ, FRACS, PAUL FAGAN, FRACS, MD, MARCUS ATLAS, FRACS, and CELENE McNIEL, MA, Sydney, Australia

The reversible hearing loss in the nonoperated ear noted by patients after ear surgery remains unexplained. This study proposes that this hearing loss is caused by drill noise conducted to the nonoperated ear by vibrations of the intact skull. This noise exposure results in dysfunction of the outer hair cells, which may produce a temporary hearing loss. Estimations of outer hair cell function in the nonoperated ear were made by recording the change in amplitude of the distortion-product otoacoustic emissions before and during ear surgery. Reversible drill-related outer hair cell dysfunction was seen in 2 of 12 cases. The changes in outer hair cell function and their clinical implications are discussed.

(OTOLOGYNOL Head Neck Surg 1997;117:555-8.)

The damaging effect of prolonged exposure to loud environmental noise has been studied extensively and is now well established.1,2 In contrast, the contribution that instrument-generated noise makes to cochlea damage during ear surgery has received relatively little attention. The effect of drill-generated noise on the nonoperated ear is discussed even less. This has clinical significance because patients, if asked, will report transient hearing loss in the nonoperated ear after operations in which drilling has been performed for prolonged periods. This study aims to document the transient hearing loss that occurs in the nonoperated ear in patients who undergo temporal bone surgery by means of measuring outer hair cell (OHC) function by use of distortion-product otoacoustic emissions (DPOAEs). The mechanisms of the hearing loss are discussed in terms of the known levels of noise produced by drills.

METHODS AND PATIENTS

Intraoperative recordings of DPOAEs were made in 12 patients who underwent standard temporal bone surgery performed by the senior surgical authors (P. A. F., M. D. A.) at St. Vincent’s Hospital, Sydney, Australia. Each patient had normal hearing in the nonoperated ear (1 to 6 kHz), normal middle ear function, and an ear canal sufficiently wide to accept the recording probe. After the patient was anesthetized, the ear canal of the nonoperated ear was cleared of debris, and the recording probe was inserted and firmly taped in place (Fig. 1). A shaped foam pillow was placed around the ear and probe to minimize the chances of the probe shifting while the recordings were being made. The ventilator device was stopped, alarms were temporarily disabled, and suction devices were turned off to reduce the levels of contaminating background noise. Standard anesthetic techniques were used with long-term muscle paralysis. The muscle paralysis minimized the effect of middle ear reflexes on the recordings.

The duration of drilling ranged from 90 minutes (mastoidectomy, exostosis, acoustic tumor surgery) to several hours (skull base surgery). Two different drill systems were used for the surgery, Bien Air (35,000 rpm; Bien Air, Bienn, Switzerland) and Mednexit (60,000 rpm; Mednexit, Inc., West Palm Beach, Fla.), and each was fitted with various sizes of cutting and
diamond burs. This provided a wide range of noisy conditions for study.

Otoacoustic emissions were chosen as a measure of OHC function for several reasons. They are conveniently recorded by commercially available equipment (Celesta 530; Madsen Electronics) without interfering with standard operative protocol. The amplitude of the DPOAE is stable and is unaffected by the action of efferent pathways, middle ear reflexes, or anesthesia.3,4

In measuring DPOAEs, two pure tones of known frequency are presented simultaneously by speakers placed in the external auditory canal. As a result of the nonlinearity of the cochlea, mainly because of the active function of the OHCs,5-7 a third lower level tone (the distortion product) is produced. This tone is transmitted through the middle ear by the reverse of normal middle ear function and can be detected by a sensitive microphone placed in the canal close to the drum. The practical application of this knowledge is that OHCs of known regions within the cochlea can be studied with confidence by simply adjusting the primary frequencies and measuring the amplitude of the evoked third tone, or DPOAE. Reduced DPOAE amplitude reflects a dysfunction of OHCs and, in the setting of this study, changes in DPOAE amplitude are likely to be the result of the effects of drill-generated noise within the cochlea.

Initial baseline recordings of DPOAEs were made at the 2, 3, 4, and 6 kHz places, followed by multiple recordings after periods of drilling lasting 5 to 20 minutes. Intraoperative recordings were made in 12 patients before and after brief periods of drilling (Table 1). Several weeks after surgery pure-tone and speech-reception thresholds were recorded for comparison with the preoperative audiogram.

**RESULTS**

In two cases (patients 1 and 2; Fig. 2) there was a reduction in DPOAE amplitude with drilling. In the remaining 10 cases there was no change in DPOAE amplitude. In patient 1 the higher speed drill (Mednext, 65,000 rpm) was used, and in patient 2 the lower speed drill was used (Bien air, 35,000 rpm). In both cases the decrease in DPOAE amplitude was easily measurable.

---

**Table 1.** Case summary of intraoperative recordings of DPOAEs

<table>
<thead>
<tr>
<th>Patient no.</th>
<th>Age (yr)</th>
<th>Hearing loss</th>
<th>Operation</th>
<th>Drill</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>26</td>
<td>Yes</td>
<td>Mastoidectomy</td>
<td>Mednext</td>
</tr>
<tr>
<td>2</td>
<td>46</td>
<td>Yes</td>
<td>Acoustic neuroma</td>
<td>Bien air</td>
</tr>
<tr>
<td>3</td>
<td>40</td>
<td>No</td>
<td>Acoustic neuroma</td>
<td>Bien air</td>
</tr>
<tr>
<td>4</td>
<td>45</td>
<td>No</td>
<td>Acoustic neuroma</td>
<td>Bien air</td>
</tr>
<tr>
<td>5</td>
<td>51</td>
<td>No</td>
<td>Skull base</td>
<td>Bien air</td>
</tr>
<tr>
<td>6</td>
<td>54</td>
<td>No</td>
<td>Acoustic neuroma</td>
<td>Bien air</td>
</tr>
<tr>
<td>7</td>
<td>6</td>
<td>No</td>
<td>Mastoidectomy</td>
<td>Bien air</td>
</tr>
<tr>
<td>8</td>
<td>41</td>
<td>No</td>
<td>Exostosis</td>
<td>Bien air</td>
</tr>
<tr>
<td>9</td>
<td>37</td>
<td>No</td>
<td>Exostosis</td>
<td>Bien air</td>
</tr>
<tr>
<td>10</td>
<td>38</td>
<td>No</td>
<td>Mastoidectomy</td>
<td>Bien air</td>
</tr>
<tr>
<td>11</td>
<td>52</td>
<td>No</td>
<td>Mastoidectomy</td>
<td>Bien air</td>
</tr>
<tr>
<td>12</td>
<td>26</td>
<td>No</td>
<td>Canalioplasty</td>
<td>Bien air</td>
</tr>
</tbody>
</table>

*Patients in whom decreases in DPOAE amplitude were seen.*
Fig. 2. Summaries of four intraoperative recordings are presented. Each shows the DPOAE amplitude recorded at different frequencies, before and after brief periods of drilling. Duration of drilling is indicated on the time scale. **A** and **B**, Examples from 10 cases in which no changes in DPOAE amplitude were seen. **C** and **D**, two cases of significant decrease in DPOAE amplitude, very likely representing an intraoperative noise-induced hearing loss.

above the background noise levels in the external ear canal. In all 12 cases the hearing in the nonoperated ear, when measured by pure-tone and speech audiometry, was normal. Similarly, in the postoperative audiogram after a series of mastoid operations, no changes in pure-tone bone conduction levels were found, suggesting that no permanent cochlear changes had occurred.8

The decrease in DPOAE amplitude seen in the patients 1 and 2 is likely to be directly related to instrument-generated noise. This notion is supported by human and animal experiments conducted under controlled conditions, where otoacoustic emissions have been used to study OHC function. In these experiments the primary response to acoustic overstimulation, lasting from minutes to less than 1 hour, was a reduction in emission amplitude.9,10

**DISCUSSION**

Does instrument-generated noise result in hearing loss during ear surgery? This is a clinically relevant question because hearing loss in the nonoperated ear has been noted after ear surgery when drilling has been prolonged.11 Known levels of drill-generated noise, the likely damaging effect of this noise, and an understanding of the mechanisms by which loud sound leads to hearing loss allow this question to be answered with some degree of confidence.

In animal experiments, brief exposures to loud sound at the intensities commonly encountered at ear

**A** *Reversible Hearing Loss and OAE*

\[
\text{normal OHC} \xrightarrow{\text{OHC dysfunction}} \text{reversible hearing loss}
\]

\[
\text{decreased OAE}
\]

**B** *Reversible and Permanent Hearing Loss*

\[
\text{Reversible physiological interruption to OHC function} \xrightarrow{\text{exposure duration and intensity}} \text{Permanent hair cell death}
\]

**Fig. 3.** Relationship between normal hearing and normal OHC function is shown. **A**, With brief exposures to intense sound, a change occurs in the OHCs in the cochlea, resulting in a decrease in DPOAE amplitude and reversible hearing loss. **B**, After prolonged high-intensity noise exposure, permanent changes occur in the OHCs, resulting in irreversible hearing loss.
surgery, around 110 dB SPL,12-15 produce threshold shifts of up to 60 dB after several minutes of exposure.16,17 This, coupled with the negligible attenuation of sound by the intact skull:18-20 suggests that the non-operated ear is exposed to significant levels of sound, which may lead to hearing loss. This hearing loss is further supported by the two cases in which intraoperative reductions in DPOAE amplitude were recorded (patients 1 and 2). A temporary threshold shift in the ipsilateral ear during surgery has been documented by means of serial intraoperative electrocochleogram recordings.21,22

Loud noise produces a hearing loss by changing the way OHCs function within the cochlea. In the case of temporary hearing loss the OHCs are physically intact, but there is a reversible physiologic disruption to their function (Fig. 3). Permanent changes occur with prolonged high-intensity exposures, resulting in irreversible hearing loss.2,22 In this study the postoperative audiogram remained unchanged in all cases, suggesting that, in the two cases in which temporary reduction in DPOAE amplitude occurred (patients 1 and 2), there was no permanent hearing loss. An examination of bone conduction audiograms after mastoid surgery showed no change in threshold from preoperative levels, supporting the notion that drill noise does not produce permanent cochlear damage.8 Similar findings were noted in animals, in which the postexposure evoked response thresholds were normal despite hearing loss being present immediately after exposure to loud noise.23

In this study an intraoperative decrease in DPOAE amplitude in the nonoperated ear was unusual, occurring in only 2 of 12 cases, despite drill-generated noise levels being sufficiently high to readily produce hearing loss under experimental conditions.

CONCLUSIONS

Temporary OHC dysfunction in the nonoperated ear occurs in some cases of temporal bone surgery. This is because of the levels of noise produced by drills and the sound-conducting characteristics of the intact skull. The drill speed and duration of drilling do not seem to be major influencing factors. Commonly available, high-speed otologic drills and burrs are safe from the point of view of permanent hearing loss; however, it is likely that temporary intraoperative hearing loss does occur.

With this sample of subjects, we could not demonstrate any permanent hearing loss in the nonoperated ear caused by drill-generated noise. However, the demonstration of temporary OHC dysfunction in two subjects still raises the possibility of a permanent drill-induced hearing loss in more susceptible ears with underlying fragile cochleae. Further investigation needs to be undertaken in this area.

REFERENCES

16. de Cruz MJ. Quantification of the fundamental mechanisms leading to cochlear hearing loss [master of medical science thesis (physiology)]. University of Western Australia, Perth, 1994.