

Perioperative Hearing Impairment

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PERIOPERATIVE hearing loss is a rarely reported phenomenon. However, it occurs more frequently than most anesthesiologists suspect. Perioperative hearing impairment is often subclinical and may go unnoticed unless audiometry is performed.¹⁻³ It can be conductive or sensorineural, unilateral or bilateral, and transient or permanent.⁴⁻⁸ Hearing loss has been reported following virtually every type of anesthetic technique. The hearing mechanism may be less susceptible to acoustic trauma during general anesthesia,⁹ but other mechanisms are capable of causing both conductive and sensorineural hearing losses (SNHL) in the perioperative period. The etiologies include mechanical, traumatic, noise-induced, changes in cerebrospinal fluid (CSF) pressure, nitrous oxide, embolism, pharmacologic, and other miscellaneous causes.

Brief Overview of Anatomy and Physiology

The cochlea is the auditory portion of the inner ear. It is a snail-shaped structure of 2.75 turns with an uncoiled length of approximately 3 cm (fig. 1). The cochlea is divided into three channels. The two outer channels, the scala vestibuli and the scala tympani, contain perilymphatic fluid and communicate at the apex through the helicotrema. The middle channel, the scala media (cochlear duct), contains endolymphatic fluid. In the conventional cross-sectional view of the cochlea (fig. 2), the scala media is triangular in shape. Its upper boundary, the vestibular or Reissner's membrane, attaches to the osseous spiral lamina and to the outer wall of the co-

chlea, separating it from the scala vestibuli. The lower boundary, the basilar membrane, also attaches to the osseous spiral lamina and the outer wall of the cochlea. The basilar membrane supports the hearing organ, the organ of Corti, and separates the scala media from the scala tympani. The lateral wall of the scala media is the highly vascular stria vascularis. At the base of the cochlea, the perilymph of the scala vestibuli contacts the oval window and the perilymph of the scala tympani contacts the round window (figs. 1 and 3).

Arterial blood is supplied to the inner ear by the internal auditory artery, which arises from either the basilar or the inferior anterior cerebellar artery and passes through the internal auditory meatus with cranial nerve VIII. The arterial supply to the cochlea is *via* end vessels, with no collateral circulation. The afferent special somatic exteroceptive cochlear nerve provides innervation. The peripheral processes arise in the organ of Corti, their cell bodies forming the spiral ganglion of the cochlea in the osseous spiral lamina. The processes converge, traversing the modiolus to form the cochlear nerve, which passes through the internal acoustic meatus with the vestibular nerve. A smaller number of efferent nerve fibers arise from the olivary complexes and terminate axodendritically on the afferent dendrites that innervate the inner hair cells.

The cochlear fluid system is made up of two fluids, perilymph and endolymph (fig. 2). Perilymph fills the scala vestibuli and scala tympani and has an ionic composition, low in potassium and high in sodium, similar to interstitial fluid and nearly identical to CSF.¹⁰ There are two theories regarding its origin: (1) it is filtrate produced by capillaries in the spiral ligament; and (2) it is CSF communicated to cochlea through the cochlear aqueduct, a small channel in the temporal bone near the round window, which connects the scala tympani directly to the subarachnoid space. The endolymphatic fluid is unique to the inner ear. It has a high concentration of potassium and a low concentration of sodium, similar to intracellular fluid. The stria vascularis is believed to be the source of the unique endolymph ionic composition. Although there is slow longitudinal flow of endolymph, maintenance of the critical ionic environment for the organ of Corti is due to local radial flow between the stria vascularis and nearby cells. Endolym-

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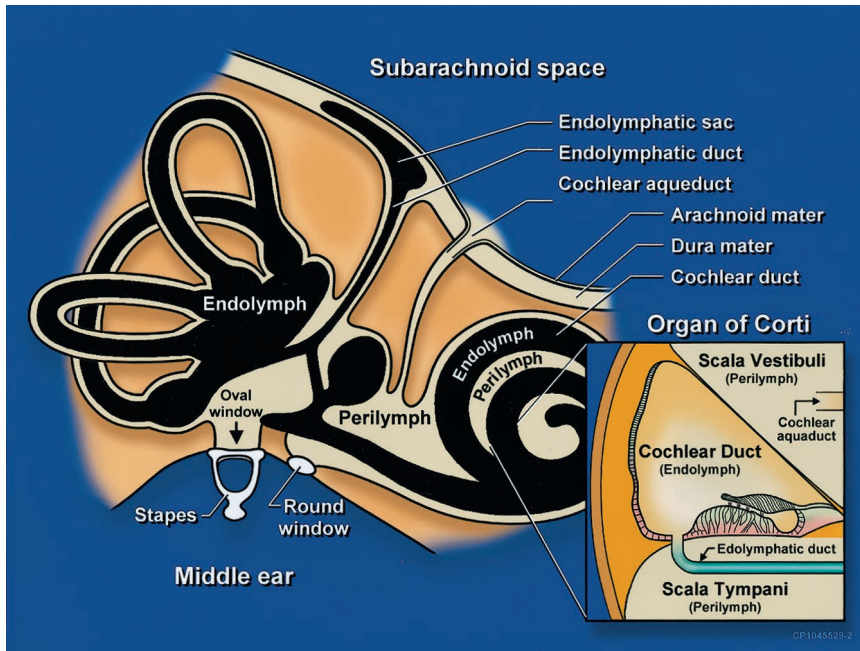


Fig. 1. Diagram of the inner ear. (Bottom) Middle ear with the stapes at the oval window; (top) subarachnoid space; (left) semicircular canals; (right) cochlea; (inset) cross-section of the cochlea. Reprinted from Lybecker H, Andersen T, Helbo-Hansen HS: The effect of epidural blood patch on hearing loss in patients with severe postdural puncture headache. *J Clin Anesth* 1995; 7:457-64, with permission from Elsevier Science.

phatic absorption takes place in the endolymphatic sac, a specialized structure of the membranous labyrinth. The endolymphatic sac is located in the subdural space and communicates with the membranous labyrinth *via* the endolymphatic duct (fig. 1). It is thought to participate in balancing endolymphatic pressures with changes in CSF pressure. Two-way flow through a patent cochlear aqueduct provides relatively rapid perilymphatic pressure equilibration with the CSF. Endolymphatic pressure regulation is considerably slower, being primarily a function of production and absorption. Only minor and temporary pressure adjustments are made by the endolymphatic sac.

Organ of Corti and Excitation of Hair Cells

The organ of Corti, the hearing transduction mechanism, is located in the cochlear duct (scala media). The sensory cells of the organ of Corti are mechanoreceptors with stereocilia projecting from the top of the cells into the endolymph.¹¹ There are two types of hair cells, outer (OHC) and inner (IHC) hair cells (fig. 2). They differ in morphology, microanatomical location, and function. The IHCs and OHCs initiate the transduction process by transforming the acoustic signal into neural activity. Activation of the hair cells causes neurotransmitter release that depolarizes afferent dendrites causing an all-or-none

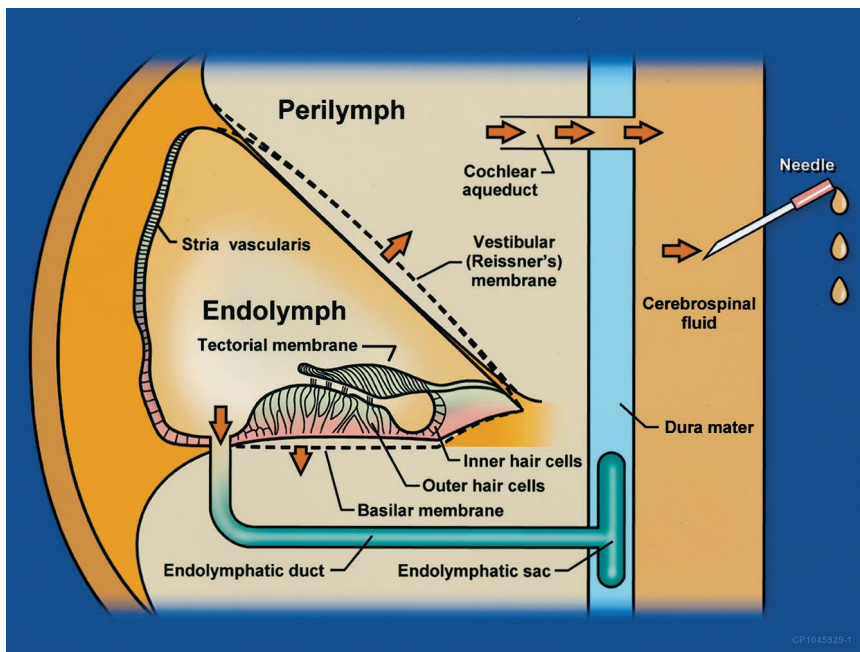
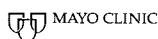
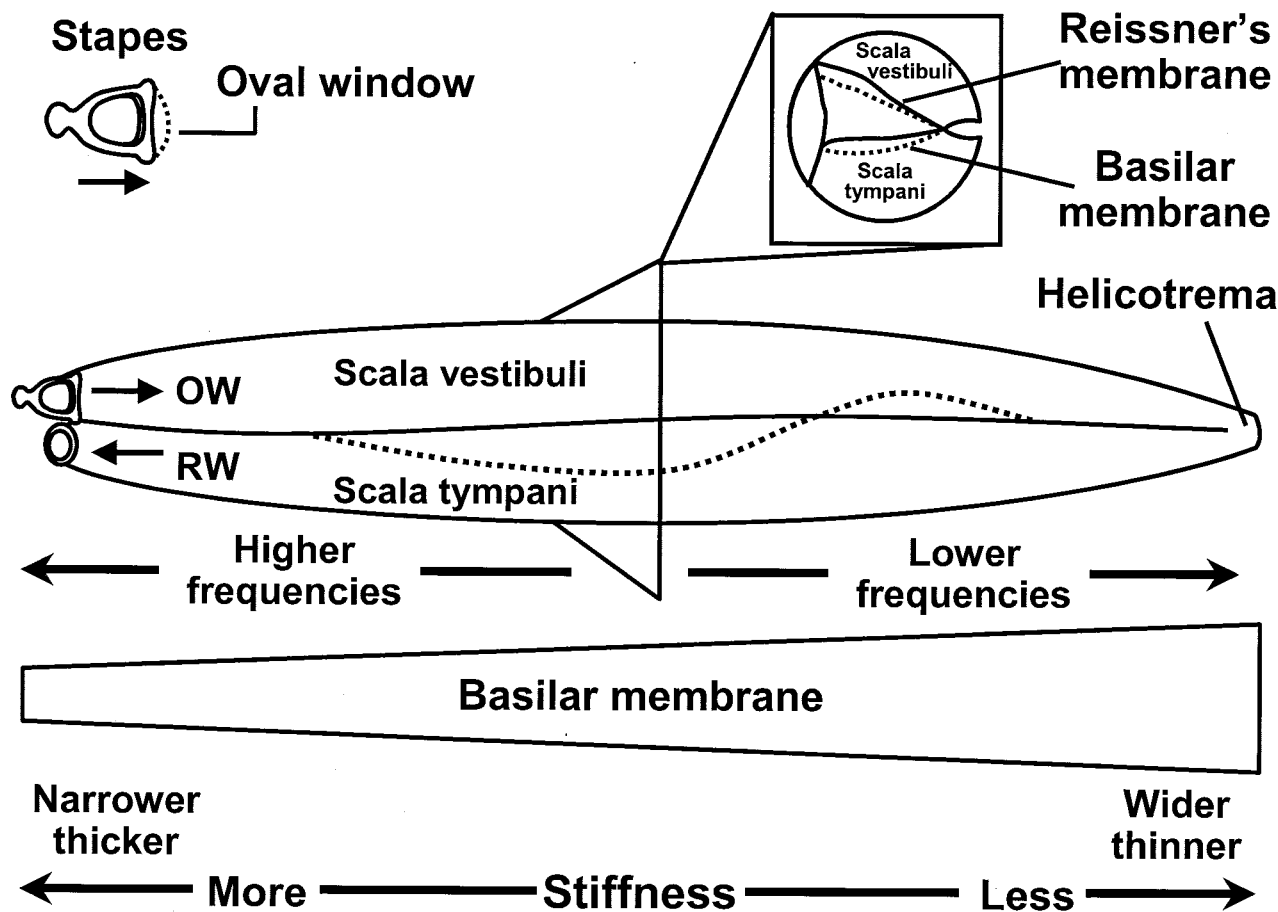


Fig. 2. Cross-sectional diagram of the cochlea. Perilymph and cerebrospinal fluid (CSF) communicate freely through a patent cochlear aqueduct. A decrease in CSF pressure from a dural leak (lumbar puncture) causes a decrease in perilymphatic pressure. Endolymphatic pressure-volume changes are much slower. As a result, endolymphatic pressure exceeds perilymphatic pressure, and both the vestibular and basilar membranes are distorted (hashed lines). This distortion disrupts hair cell function, which causes hearing loss. This is the postulated mechanism of hearing loss associated with spinal anesthesia.



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Fig. 3. Schematic diagram of the uncoiled cochlea. The scala media is represented as a line separating the scala vestibuli and the scala tympani. The hashed lines (at the stapes/oval window, inset, and main diagram) represent the pressure wave generated by sound entering the ear. Below, the representation of the basilar membrane indicates that it is narrower and thicker, more stiff, and tuned to higher frequencies on the left at the base of the cochlea, and wider and thinner, more compliant, and tuned to lower frequencies on the right at the apex of the cochlea. OW = oval window; RW = round window.

spike discharge in individual auditory nerve fibers. Overlying the organ of Corti is a gelatinous flap, the tectorial membrane. The tectorial membrane is attached on its medial edge to the spiral limbus and laterally is in contact with the stereocilia of the OHCs. Since the tectorial membrane and basilar membrane have different medial attachment locations, upward or downward displacement of basilar membrane induces shearing displacements of the hairs in contact with the tectorial membrane. These various bending movements of the OHC stereocilia can cause excitation or inhibition. Excitation initiates the transduction of acoustic energy into neural signals. In contrast to OHCs, the stereocilia of the IHC are not embedded in the tectorial membrane but rest in a groove near the undersurface of the tectorial membrane. The IHC do not respond to displacement of the basilar membrane, but they do respond to the velocity of displacement of the basilar membrane. The neural signals initiated by the IHCs and OHCs are transmitted

through the acoustic division of cranial nerve VIII ultimately to the auditory cortex of the temporal lobe.

A simplified diagram of the cochlea uncoiled is usually used to describe cochlear mechanics (fig. 3). On the left is the base of the cochlea with the stapes and oval window. Below these structures and adjoining the scala vestibuli is the round window of the scala tympani. In the diagram, the uncoiled cochlea is divided horizontally by a line representing the scala media, Reissner's membrane, the organ of Corti, and the basilar membrane. However, this line, for purposes of understanding cochlear mechanics, can be thought of as simply the basilar membrane. Above the dividing line is the scala vestibuli, and below is the scala tympani. Because of the inertia of the fluid mass and frictional resistance to fluid flow in the narrow channels of the scala, inward or outward motion of the stapes against the oval window causes a pressure wave to travel down the scala; there is no fluid flow. Since the fluids are incompressible within the bony

cochlea, the pressure wave traveling down the scala vestibuli causes the basilar membrane to deflect downward toward the scala tympani for positive pressures and upward for negative pressures. The flexible round window compensates for pressure changes transmitted to the scala tympani. For the acute changes caused by pressure waves in the audible frequencies, the connection between the scala vestibuli and the scala tympani, the helicotrema, functions as though it were closed. The helicotrema is only involved in very slow equalization of pressures between the two scalae.

The most important property of the basilar membrane is its ability to separate various frequencies. The compliance of the basilar membrane changes by a factor of greater than 100 from base to apex. At the base, near the oval window, the basilar membrane is narrower and thicker, making it less compliant (fig. 3). It is wider and thinner at the apex, resulting in greater compliance. Since a system's resonant frequency is directly proportional to stiffness and inversely proportional to mass, the stiff base responds maximally to high frequencies ($> 4,000$ Hz), and the compliant apex responds maximally to low frequencies ($< 1,000$ Hz). In short, the basilar membrane functions as a sharply tuned bandpass filter, performing a spectral analysis on the incoming sound (pressure) waves. The effect is to translate the frequency of incoming sound into distance along the basilar membrane, a frequency-to-place or tonotopical transformation. Therefore, both IHCs and OHCs at a specific point along the basilar membrane respond to a very specific, narrow frequency range. Excited hair cells depolarize specific acoustic neurons, and the information is finally interpreted in the auditory centers.

Anesthesia and Hearing Loss

Hearing Loss and Neuraxial Anesthesia

Clinical or subclinical hearing loss following spinal anesthesia or lumbar puncture has been reported frequently (table 1).^{4,6-8,12-24} Despite many reports, few anesthesiologists appear to be aware of the possibility of this complication. Since there has been no large-scale audiometric study of hearing loss associated with spinal anesthesia, the precise incidence of clinical or subclinical hearing loss is unknown but may occur more frequently than appreciated. Further, the specific presentation and onset of the hearing deficit after spinal anesthesia can vary widely.

The first case of hearing loss associated with spinal anesthesia²⁵ was reported in 1914. In 1956, Vandam and Dripps²⁶ reported that of 9,277 patients who had spinal anesthesia, 0.4% experienced auditory difficulties, such as impaired hearing, tinnitus, buzzing, or roaring. Few specific data were gathered, and no audiometry of these patients was performed. Michel *et al.*²⁷ described nine cases of hearing loss following myelography, diagnostic

lumbar puncture, and spinal anesthesia. The hearing deficits were almost uniformly in the lower frequencies (125–1,000 Hz). The deficits were bilateral in six of the nine patients. Six of the nine patients had full recoveries in less than 1 month without treatment.

In addition to these case reports, there have been several studies of hearing loss related to spinal anesthesia. In one study, 6 of 14 patients had audiometrically detectable mild hearing deficits in the low-frequency range that resolved spontaneously in 1–7 months.²⁸ Walsted *et al.*²⁹ tested 34 patients with audiometry before and after spinal anesthesia. Most of the patients had a small but significant threshold shift at 500 Hz. However, one patient developed a considerable unilateral hearing loss in the low-frequency range, which persisted until an epidural blood patch was performed. Gultekin *et al.*³⁰ used pure tone audiometry to test hearing before and after spinal anesthesia for hernia repair. Fourteen patients (32%), despite being unaware of it, had audiometrically measurable low-frequency hearing losses. All spinal anesthetics were performed with 22-gauge Quincke needles (needle gauge and design are discussed in the next section), and the authors suggested that hearing loss was less frequent when the anesthetic agent was bupivacaine rather than prilocaine. In 35 patients undergoing elective cesarean section under spinal anesthesia (24-gauge Sprotte needle), pure tone audiometry revealed that five patients had developed low-frequency hearing loss on the first postoperative day.³¹ The deficits were unilateral in three patients and bilateral in the other two. All patients had full recovery without treatment by the fifth postoperative day. In contrast, Finegold *et al.*³² found no audiometric hearing decrement postoperatively in 44 patients undergoing cesarean section under spinal anesthesia. Half of the patients' spinals were performed with 25-gauge Quincke needles; 24-gauge Sprotte needles were used for the other half. This is the only study of 13 found that failed to show a relationship between spinal anesthesia and hearing loss. Hearing acuity is affected by hormonal changes,^{33,34} but peripartum changes have not been documented. The explanation for this singular result may be in the study's methodology.

In a study of 100 patients undergoing elective urologic procedures under spinal anesthesia, eight patients experienced noticeable hearing impairment.¹⁸ Audiometry was performed on three of the patients and revealed a 30-dB loss in the low-frequency range. Hearing deficits resolved within a short time without treatment. In another study of 100 patients undergoing elective general or urologic surgery under spinal anesthesia, 16 had audiometrically documented hearing losses.³ The hearing loss typically affected only the frequencies between 125 and 2,000 Hz. The deficits were usually detected on the second day after the spinal anesthetic and in all cases resolved within 3 days without treatment. The incidence of hearing loss after spinal anesthesia for men under 30 yr of age was compared to that in men over the age

Table 1. Hearing Impairment and Spinal Anesthesia

Source	Patients, n	Onset	Hearing Loss Frequency	Affected Side	Needle Gauge	Recovery†
Vandam <i>et al.</i> ²⁶	35	NR	NR	Unilateral and bilateral	NR	NR
Panning <i>et al.</i> ¹⁷	8	NR	Low (A3)	Bilateral	22	F, 1 or 2 days
Wang ²²	1	1 day	Low (A)	Bilateral	22	F, 5 days
Lee <i>et al.</i> ⁴	1	2 days	Low (A)	Unilateral	25	F, 4 days
Hardy ⁵³	3	<1 h	NR	Bilateral	17 E	F, < 10 min
Wang <i>et al.</i> ²⁸	6	2 days	Low (A)	Unilateral and bilateral	22 & 25	F, 1–7 mo
Hardy ²⁴	2	1 day	NR	Unilateral and bilateral	18 & 22	F, 2–5 days
Dreyer <i>et al.</i> ³	16	1 day	Low (A)	Unilateral and bilateral	NR	F, 3 days
Fog <i>et al.</i> ²	17	2 days	Low (A)	Unilateral and bilateral	22 & 26	NR
Michel <i>et al.</i> ²⁷	3	2 days	Low (A)	Bilateral	NR	F, 7 days (2); N (1)
Walsted <i>et al.</i> ⁴⁷	1	2 days	Low (A)	Unilateral	NR	F, 5 days
Oncel <i>et al.</i> ¹	30	3 or 4 days	Low (A)	Unilateral and bilateral	22 & 25	NR
Sundberg <i>et al.</i> ³⁹	8	1 or 2 days	Low (A)	Unilateral	22	NR
Wang <i>et al.</i> ³⁷	1	2 days	Low (A)	Bilateral	26	F, 5 days
Hussain <i>et al.</i> ³¹	5	1 day	Low (A)	Unilateral and bilateral	NR	F, 2–5 days
Wemama <i>et al.</i> ⁸	1	1 day	High (A)	Unilateral	22	NR
Lamberg <i>et al.</i> ⁴⁰	16	<1 h	Mid (A)	Unilateral and bilateral	*	F, 3 days
Johkura <i>et al.</i> ¹²	1	2 days	Low (A)	Bilateral	22	F, 3 weeks
Schaffartzik <i>et al.</i> ³⁶	2	<1 h	Low (A)	Unilateral and bilateral	26	F, 2 days
Gultekin <i>et al.</i> ³⁵	14	2 days	Low (A)	Unilateral and bilateral	22	NR

* Nine patients with single-shot spinal using 25- or 27-gauge needle and seven patients with continuous-spinal using a 22-gauge needle with a 29-gauge catheter and a 19-gauge needle with a 22-gauge catheter. † Numbers in this column indicate the number of patients with full or no recovery of hearing impairment.

A = audiometric testing performed; F = full recovery; N = no recovery; NR = not reported.

of 60.³⁵ Fifty-two percent (13 of 25) of the younger and 16% of the older patients had significant hearing losses (≤ 10 dB) confined to the low-frequency range (125–500 Hz). There was no hearing impairment in the speech frequencies (500–2,000 Hz) and higher frequencies for either group. No patient was aware of a hearing deficit, and no patient developed a post-dural puncture headache (PDPH). In a recent study of hearing loss following spinal *versus* general anesthesia, there were audiometric losses in both groups, although the spinal group had significantly greater hearing losses, particularly in the low-frequency range.³⁶ Interestingly, in the spinal group, increases in the low-frequency audible threshold were inversely related to the preoperative low-frequency audible threshold.

Overall, it appears that 10–50% of patients receiving spinal anesthesia experience an audiometrically measurable low-frequency hearing deficit. Less than one fourth of these patients have a clinical, or noticeable, hearing loss.

Hearing Loss after Neuraxial Anesthesia: Needle Gauge and Design

The size of the needle used for dural puncture appears to play a role in postspinal anesthetic hearing impairment. The hearing loss may be a result of CSF leakage even when the CSF loss is insufficient to cause a PDPH. Fog *et al.*² found that 13 of 14 patients whose spinal anesthetics were performed with a 22-gauge needle had a 10-dB or greater hearing loss across the audible frequency range with significantly greater losses in the low-frequency range. Only 4 of the 14 patients in whom a 26-gauge needle was used had similar hearing impairment, and none had significant (> 10 dB) losses. When

spinal anesthesia was performed with a 26-gauge needle,³⁷ only one of nine patients was found to have a low-frequency hearing loss, which was severe but transient. Interestingly, this patient also experienced a PDPH. In another study of 100 patients, 50 had spinal anesthesia using either a 20- or a 22-gauge needle. Of these, 10% had significant decreases in audiometric values in the 250- to 500-Hz frequency range on the first postoperative day that resolved spontaneously within 5 days.³⁸ In the 50 patients in whom 24- and 25-gauge needles were used, no decrement in audiometric values occurred. Oncel *et al.*¹ used pure tone audiometry at 125 Hz preoperatively and postoperatively to assess hearing loss. Three groups of 15 patients were studied. One group received epidural anesthesia. In the other two groups, spinal anesthesia was performed with either a 22- or 25-gauge needle. No hearing impairments were detected in the epidural group. There was significantly greater hearing loss in the 22-gauge group *versus* the 25-gauge spinal group. None of the study patients developed a PDPH. The authors suggested that pure tone audiometry may be a more sensitive indicator of CSF leakage than the presence of PDPH.

Needle design may also play a role in hearing impairment following lumbar puncture. Using audiometry, Sundberg *et al.*³⁹ compared the effect of 22-gauge cutting-tip (Quincke) to 22-gauge pencil-point (Whitacre) needles on postspinal hearing loss. A hearing loss of at least 10 dB at two or more frequencies below 1,000 Hz was observed in 6 of 25 (24%) patients in the Quincke group as compared to only 2 of 23 (9%) in the Whitacre group. In addition, of those patients with hearing deficits, the mean hearing level was significantly worse in

the Quincke group. This study suggests an association between the design of the spinal needle tip and postoperative hearing dysfunction that is similar to the relationship between the needle types and the incidence of PDPH, implying a similar pathogenesis. Lamberg *et al.*,⁴⁰ using audiometric testing, compared hearing losses after a single-shot spinal with a 25-gauge Quincke needle with those following a continuous spinal anesthetic (19-gauge Touhy-type needle and 22-gauge catheter). None of the patients developed a PDPH or were aware of a hearing loss. However, nine (43%) of the single-shot patients and seven (37%) of continuous spinal patients had hearing deficits in excess of 10 dB when tested postoperatively. This result also seems to parallel experience with the cause and incidence of PDPH.

In summary, hearing impairment following neuraxial anesthesia seems to be related to the same factors (age, needle gauge, and needle type) that are implicated in CSF leakage and PDPH.⁴¹ Two of the studies cited above included a total of 27 patients who received epidural anesthesia. Audiometric testing did not reveal measurable postoperative auditory impairment in any of these patients. On the other hand, the consensus of the studies indicates a consistent rate (10–50%) of SNHL in the low frequencies after lumbar puncture. Also, postspinal hearing impairment resolved without treatment, usually within several days, again suggesting an etiology common with that of PDPH. Interestingly, in two reports, patients who received epidural blood patches for PDPH had simultaneous reversal of their postspinal, low-frequency hearing loss.^{4,29}

Cerebrospinal Fluid Pressure and Hearing Loss

Cerebrospinal fluid leakage can cause a decrease in the CSF pressure that may be transmitted to the inner ear. A relative balance in the endolymphatic and perilymphatic pressures maintains the normal structural conformation in the inner ear. Disruption of this pressure balance can cause hearing impairment as well as impairment of semicircular canal function. A change in CSF pressure is promptly transmitted through a patent cochlear aqueduct to the inner ear perilymph (fig. 2).^{6,42–45} A decrease in CSF pressure following a dural puncture and CSF leak would cause a rapid and similar decrease in perilymph pressure. The endolymphatic system, however, responds much more slowly. Endolymphatic pressure-volume adjustments are primarily the result of altered endolymph production at the stria vascularis or altered absorption at the endolymphatic sac. Therefore, an acute drop in CSF pressure could result in endolymphatic pressure sufficiently exceeding that of the perilymph to cause distortions of both Reissner's membrane and the basilar membrane. The consequent disruption in the position of the hair cells results in hearing impairment.¹⁰ This mechanism has been postulated in several reports.^{3,6,8,46} Walsted *et al.*⁴⁷ proposed such a mechanism

in 1991. Later, in controlled prospective studies of neurosurgical patients and in animal studies, she showed the relationship between CSF loss and low-frequency hearing impairment.^{48–52} The finding that the degree of hearing impairment is correlated with the size of the spinal needle further supports the theory that postspinal hearing loss and PDPH share a common etiology: leakage of CSF from the subarachnoid space.^{1,2}

Distortion of the basilar membrane and hearing loss could also be caused by acute increases in CSF pressure transmitted to the perilymph. In a case report, Hardy⁵³ postulated that injection into the epidural space caused an acute increase in CSF pressure that was immediately transmitted through the cochlear aqueduct to the perilymph, thus distorting the basilar membrane and causing an acute low-frequency hypoacusis.

Finally, if the mechanism of postspinal hearing loss is the same as for PDPH, several questions arise: why do we not see as many patients with vestibular symptoms; why does not every patient with a severe PDPH experience hearing impairment; and why are some hearing losses unilateral? In response to the first question, not only are the changes in CSF pressure transmitted less rapidly to the vestibular apparatus, but the vestibular apparatus is somewhat less sensitive than the cochlea to perilymph–endolymph pressure imbalances. Further, the hearing deficit may be subclinical, not obvious without audiometric testing. In answer to the second question, as many as 7% of adults have an anatomically obstructed cochlear aqueduct, and as many as 30% have a functionally obstructed aqueduct.^{44,45,54} For these patients, CSF pressure changes are not transmitted to the perilymph, and, therefore, a severe PDPH could exist without a hearing deficit. This mechanism may also explain the cases of unilateral hearing deficit. In addition, restricted cochlear aqueduct flow and the mechanical compliance of the cochlear windows can limit the stress of CSF pressure changes.

The reports cited above indicate that the great majority of hearing deficits that result from lumbar puncture or CSF loss occur in the low frequencies and, in most cases, bilaterally. We propose that this is the result of the physical characteristics of the basilar membrane. At the cochlear base, where higher frequencies are transduced, the basilar membrane is narrow, thick, and stiff, and therefore resistant to pressure changes that are not in its resonant frequency range (fig. 3). At the cochlear apex, where low frequencies are transduced, the basilar membrane is much more compliant. Changes in CSF pressure transmitted to the perilymph can cause significant static displacement of the basilar membrane, disrupting the normal OHC relationship to the tectorial membrane and resulting in low-frequency hearing loss. Hearing loss confined to the low frequencies is unlikely due to CSF pressure-mediated stretch or pressure injury to cranial nerve VIII.

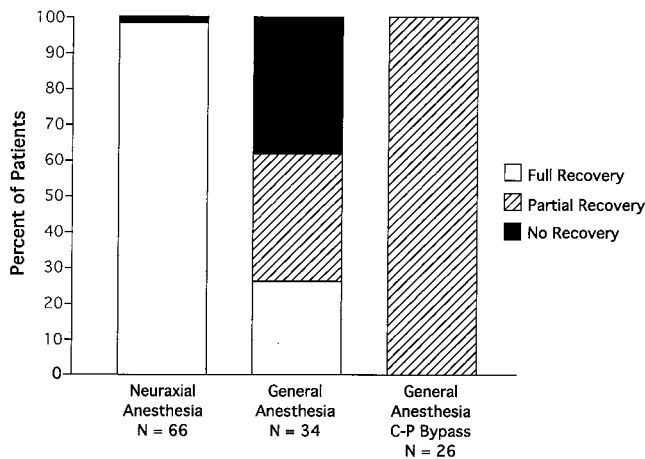


Fig. 4. Histogram showing hearing loss outcome following hearing loss associated with anesthesia (from tables 1, 3, and 4). Sixty-five of 66 patients had full recovery of hearing loss with neuraxial anesthesia. All patients with hearing loss following cardiopulmonary bypass under general anesthesia had partial recovery with some permanent loss. Recovery of hearing loss after general anesthesia without cardiopulmonary bypass is less uniform and is a function of the specific etiology.

It is interesting to note that the low-frequency hearing loss observed in Ménière's syndrome involves a similar mechanism. In Ménière's syndrome, instead of a decrease in perilymphatic pressure relative to endolymphatic pressure, there is an increase in endolymphatic pressure (endolymphatic hydrops). This imbalance in pressures would cause a similar distortion of the basilar membrane with the greatest effect at the cochlear apex and resulting, at least in the early stages, in a hearing loss (low frequency) similar to that which can occur after spinal anesthesia.

Neuraxial Anesthesia: Treatment and Prognosis

Presuming that hearing impairment following spinal anesthesia or dural puncture has the same etiology as PDPH, *i.e.*, loss of CSF or decreased CSF pressure, then the treatment and prognosis should be similar. Figure 4 indicates that untreated hearing impairments following dural puncture recover fully greater than 95% of the time. Several authors have noted improvement in auditory symptoms after an epidural blood patch.^{4,29,55} Lybecker *et al.*⁴⁶ prospectively studied the effects of an epidural blood patch on hearing in patients with severe PDPH caused by dural puncture. They performed audiometry at several frequencies 1 h before and 1 h after an epidural blood patch. Twelve of the 16 patients had significant audiometric improvement in hearing. Simply changing position from sitting to supine improved hearing in half of the patients in the absence of the blood patch. This study offers clear evidence of the potential efficacy of epidural blood patch for post-dural puncture

hearing loss. In another case report in which conservative measures followed by two epidural blood patches failed to relieve a severe PDPH and bilateral low-frequency hearing loss, an epidural dextran 40 infusion relieved the headache and reversed the hearing loss.⁵⁶

Hearing Loss after Other Types of Regional Anesthesia

Rosenberg *et al.*⁵⁷ performed an audiometric study of 20 patients undergoing surgery with interscalene brachial plexus block anesthesia. Four of the patients (20%) experienced hearing impairment at various frequencies on the side of the block. The low frequencies (250–500 Hz) were affected in only one patient who also had hearing threshold decrements on the opposite side of 15–20 dB measured in the 6,000- to 10,000-Hz range (table 2). Farrell *et al.*⁵⁸ described four patients with sudden permanent deafness following anesthesia for dental surgery. Anesthesia for three of the patients was local infiltration or nerve block, and the fourth had general anesthesia. The patient who had general anesthesia had bilateral SNHL, while the three other patients had ipsilateral SNHL. Similarly, Shenkman *et al.*⁵⁹ reported a case in which permanent neurologic symptoms, ipsilateral hearing impairment, a facial nerve palsy, and ataxia occurred following an inferior alveolar nerve block. These findings are summarized in table 2.

Regional Anesthesia: Mechanisms, Treatment, and Prognosis

Within 24 h after the interscalene blocks, all four of the patients had spontaneous resolution of their hearing deficits.⁵⁷ The authors postulated that the mechanism of hearing loss involved an effect due to sympathetic blockade. Vasodilation caused by the sympathetic block may have developed, resulting in edema of the mucosal membranes of both the Eustachian tube and the middle ear and producing a hearing decrement on that side. The hearing loss resolved within 24 h without treatment in all four patients.

With regard to the hearing deficits following dental procedures, the authors^{58,59} could not identify a specific etiology for any of the cases. Several possibilities were considered. Noise-induced hearing loss from dental drills is known to result in temporary, noise-induced, bilateral hearing deficits.⁶⁰ Dental extractions are known to cause microemboli that could reach the cochlear vasculature and cause temporary or permanent hearing impairment.⁵⁸ Vasoconstrictors are usually used with local anesthesia for dental procedures. Intravascular injection could cause vasospasm of the cochlear division of the internal auditory artery.⁵⁸ Finally, nociceptive afferents or autonomic nerve fibers could initiate a reflex vasocon-

Table 2. Hearing Impairment and Other Nerve Blocks

Source	Patients, n	Onset	Hearing Loss Frequency	Affected Side	Block Type	Recovery
Farrell <i>et al.</i> ⁵⁸	3	6 h to 7 days	NR	Unilateral and bilateral	Infiltration (2) Alveolar nerve block (1)	N
Rosenberg <i>et al.</i> ⁵⁷	4	10 min to 24 h	Low and high (A)	Unilateral	Interscalene brachial plexus block	24 h
Shenkman <i>et al.</i> ⁵⁹	1	20 min	NR	Unilateral	Alveolar nerve block	N

A = audiometric testing performed; N = no recovery; NR = not reported.

striction of critical vessels to the hearing structures. Hearing loss during dental procedures appears to be quite rare and usually temporary.

Hearing Loss and General Anesthesia

Hearing impairment after general anesthesia for non-cardiopulmonary bypass surgery is rare. Our review of the English language literature through 2001 revealed only 35 reported (29 documented audiometrically) cases of hearing loss after general anesthesia (table 3). Evan *et al.*⁶¹ reported three cases of SNHL following general anesthesia; one patient recovered, and the other two sustained permanent hearing loss. The authors were unable to elucidate the etiology in any of the patients. Cox and Sargent⁶² reported three cases of hearing loss following nonotologic surgery. Two patients had permanent, profound unilateral hearing loss, and one patient had moderate bilateral hearing loss. Velazquez⁶³ described a case of acute unilateral hearing deficit following abdominal surgery under general anesthesia. Other authors have described bilateral hearing impairment immediately following minor abdominal surgeries.^{64,65} It should also be noted that some patients who receive general anesthesia come to the operating room from an

intensive care unit having already sustained an unrecognized hearing loss. Halpern *et al.*⁶⁶ have discussed the myriad causes of hearing loss in the critical care setting.

General Anesthesia: Mechanisms of Hearing Loss

Although the etiology of hearing loss associated with general anesthesia often cannot be ascertained, there are a number of potential etiologies: changes in middle ear pressure, vascular pathology, CSF pressure changes, embolism, ototoxic drugs, and other miscellaneous causes.

Middle Ear Pressure

Excessive or sudden changes in middle ear pressure can disrupt the tympanic membrane, the round window, and the conducting structures. Excessive middle ear pressure can cause the round window to rupture, resulting in significant hearing loss.^{67,68} This type of injury has been described as occurring during cardiopulmonary resuscitation.⁶⁹ Two patients who were successfully resuscitated developed partial deafness, one from disarticulation of the incomallear joint and the other from an oval window perilymph fistula. It was postulated that

Table 3. Hearing Impairment and General Anesthesia for Non-Bypass Surgery

Source	Patients, n	Onset	Hearing Loss Frequency	Affected Side	N ₂ O Used	Recovery*
Jaffe ⁸²	4	NR	NR	Unilateral	NR	N
Tonkin <i>et al.</i> ⁷⁷	2	Immediate	Low and high (A)	Unilateral	NR	N (1), F (1)
Patterson <i>et al.</i> ⁷⁶	1	Immediate	NR	Unilateral	Yes	P
Davis <i>et al.</i> ⁷⁵	1	Immediate	Low and high (A)	Unilateral	Yes	N
Man <i>et al.</i> ⁷³	2	Immediate	Low and high (A)	Unilateral	Yes	F (1), P (1)
Millen <i>et al.</i> ⁵	3	Immediate to 5 days	Low and high (A)	Unilateral	NR	P
Segal <i>et al.</i> ⁷²	3	Immediate	Low and high (A)	Unilateral	Yes	P
Hochermann <i>et al.</i> ⁶⁴	1	Immediate	Low (A)	Bilateral	Yes	P
Journeaux <i>et al.</i> ¹³	1	6 days	NR (A)	Unilateral	Yes	N
Farrell <i>et al.</i> ⁵⁸	1	3 days	NR	NR	Yes	N
Velazquez ⁶³	1	24 h	Low and high (A)	Unilateral	No	P
Rosenberg <i>et al.</i> ⁵⁷	2	Immediate to 24 h	Low and high (A)	Unilateral	NR	F
Cox <i>et al.</i> ⁶²	3	Immediate	Low and high (A)	Unilateral and bilateral	Yes (2)	N
Evan <i>et al.</i> ⁶¹	3	Immediate to 2 days	Low and high (A)	Unilateral	Yes	F (1), N (2)
Gilbert <i>et al.</i> ¹¹⁰	1	6 days	High (A)	Bilateral	Yes	NR
Schaffartzik <i>et al.</i> ³⁶	4	Immediate	Low and high (A)	Unilateral and bilateral	Yes	F
Girardi <i>et al.</i> ⁵²	2	2 days	Low and high (A)	Unilateral and bilateral	Yes	P

* Numbers indicate the number of patients with full, partial, or no recovery of hearing impairment.

A = audiometric testing performed; F = full recovery; N = no recovery; NR = not reported; P = partial recovery.

Table 4. Hearing Impairment and Cardiac Surgery

Source	Patients (n)	CABG Valve	Onset	Hearing Loss Frequency	Affected Side	Recovery
Arenberg <i>et al.</i> ⁸³	1	V	Immediate	Low and high (A)	Unilateral	P
Wright <i>et al.</i> ⁹⁰	1	V	24 h	NR	Unilateral	P
Plasse <i>et al.</i> ⁹¹	7	C and V	Immediate	Low and high (A)	Unilateral	P
Shapiro <i>et al.</i> ⁸⁸	11	C	Immediate to 96 h	Low and high (A)	Unilateral	P
Millen <i>et al.</i> ⁵	2	C	Immediate	Low and high (A)	Unilateral	P
Cervantes Escarcega, <i>et al.</i> ⁸⁵	11	C and V	Immediate	NR	Unilateral	NR
Walsted <i>et al.</i> ⁸⁹	4	C and V	Immediate to 11 days	Low and high (A)	Unilateral	P

A = audiometric testing performed; C = coronary artery bypass graft surgery; CABG = coronary artery bypass graft; NR = not reported; P = partial recovery; U = unilateral; V = valve surgery.

during vigorous mask ventilation, excessive pressure was suddenly transmitted through the Eustachian tube causing the middle ear injuries.

Nitrous oxide (N₂O) anesthesia was shown in the late 1960s to be capable of causing oscillations of middle ear pressures that could result in tympanic perforation and cause hearing loss.^{70,71} Depending on the uptake or elimination phase, N₂O can displace tympanic membrane grafts outward or inward and disrupt reconstructed middle ear conducting structures. Segal *et al.*⁷² described a case of labyrinthine membrane rupture and sustained SNHL due to elevated intratympanic pressure during general anesthesia with N₂O. The degree of intratympanic pressure fluctuation is a function of N₂O concentration and its rate of change. Several cases of middle ear injury have been described as resulting from sudden changes in intratympanic pressure during general anesthesia with N₂O.⁷³⁻⁷⁷ Further, the patency of the Eustachian tube may play a role in potential injury during N₂O anesthesia. During N₂O elimination, Eustachian tube obstruction can result in significant negative middle ear pressure and tympanic perforation.⁷⁸

Vascular Pathology

A vascular pathogenesis is another possible mechanism for hearing impairment after general anesthesia. Umemura *et al.*⁷⁹ employed a photochemical method to induce epithelial damage in the inner ear's microcirculation in a rat model. Soon after the experimental injury, cochlear action potentials (measured by electrocochleogram) diminished. The damaged vascular epithelium caused adhesion and aggregation of platelets in the microcirculation of the inner ear, and disintegration of the inner ear hair cells was evident within 24 h. These findings demonstrate that injury to the inner ear's microcirculation, either in the stria vascularis or the vessels of the spiral ligament, leads to ischemic damage of the hair cells and hearing loss.

General Anesthesia: Treatment and Prognosis

Hearing loss under general anesthesia for nonbypass surgery has a relatively good prognosis despite some

uncertainty in many cases as to the precise etiology. Table 3 indicates that, of those reported, approximately 50% of the patients had at least a partial recovery following hearing loss. This figure is in concert with the report of Mattox and Simmons⁸⁰ on hearing loss not associated with anesthesia. They found that, overall, 65% of acute idiopathic hearing dysfunctions recover completely and independently of any treatment, most within 14 days and many within just a few days. Likewise, an 8-yr prospective study of 225 patients with SNHL found that complete recovery occurred in 45% of the patients.⁸¹ Important prognostic indicators were the severity of the initial hearing deficit and the presence of vertigo. In some cases of injury to the conductive system of the middle ear, surgical treatment is indicated. However, in most cases of hearing impairment after general anesthesia, it is unlikely that any treatment would significantly alter the outcome.⁸²

Hearing Loss and Cardiopulmonary Bypass

Hearing loss after general anesthesia is more frequently associated with surgery in which cardiopulmonary bypass (CPB) is used *versus* other types of surgery (table 4).^{5,83-90} The incidence of permanent hearing impairment following CPB has been estimated to be less than 0.1%.⁸⁷ Arenberg *et al.*⁸³ published the first report of SNHL following CPB in 1972. Within the next decade, a number of other authors reported hearing impairment following CPB.^{5,88,90,91} Millen *et al.*⁵ described five cases of sudden hearing loss after general anesthesia. Two occurred after surgery involving CPB. Shapiro *et al.*⁸⁸ studied hearing acuity in 68 CPB surgery patients and found that 11 (16%), two of whom also developed tinnitus, experienced a mild, less-than-10-dB, high-frequency hearing impairment. A review of 5,975 patients who underwent open heart surgery revealed 11 cases (0.18%) of sudden unilateral hearing loss.⁸⁵ Most recently, Walsted *et al.*⁸⁹ reported four cases of hearing impairment following extracorporeal circulation. The hearing loss was evident immediately after emergence from anesthesia in three of the patients and was noted several days later in the fourth patient.

Cardiopulmonary Bypass: Mechanisms of Hearing Loss

Mechanisms discussed under general anesthesia and/or ototoxic drugs can be the etiology of hearing loss during procedures in which CPB is used, but particulate emboli generated during CPB have been considered the likely cause of most SNHLs after cardiovascular surgery.⁵ The number of arterial microemboli is significantly increased during extracorporeal perfusion and aortic cross-clamping, and microembolism to the basilar artery and the branches to the inner ear is believed to be the most likely cause of hearing loss following the use of pump oxygenator systems.^{90,92} The fact that the vast majority of these cases result in unilateral hearing loss tends to eliminate ototoxic drugs or globally decreased cerebral blood flow as likely etiologies. Further, the higher incidence of hearing deficits associated with valve procedures as opposed to coronary bypass graft procedures tends to support the embolic etiology. The inner ear is particularly susceptible since it is supplied by end arteries, and there is no collateral vascular supply. Sources of emboli include air, antifoam, fat, and particulate matter from aortic plaques or calcified valves. Hearing loss after CPB is still occasionally reported despite the improvement in CPB technology.⁸⁹

Cardiopulmonary Bypass: Treatment and Prognosis

The prognosis for hearing loss occurring during anesthesia for procedures using CPB is poor. Table 4 indicates that all of the 26 patients had partial recovery from hearing loss, but no patient had complete recovery. If the embolic etiology is, in fact, the case, this result is not surprising. Embolism to the microcirculation of the inner ear could cause ischemic damage to the stria vascularis and hair cells, resulting in some irreversible hearing loss. The loss of hearing across the auditory frequency range tends to support the vascular embolism etiology. Any treatment is unlikely to be beneficial.

Hearing Loss Associated with Otolgic Surgery

For completeness in this review, several of the more common causes of hearing loss during otologic surgery are discussed. Damage to the ossicular chain with a high-speed burr during mastoid or other surgeries causes immediate hearing loss. The loss is most profound at 4,000 Hz, fanning out to both higher and lower frequencies. If spontaneous recovery does not occur within 2 weeks, further recovery is unlikely. An iatrogenic violation of the semicircular canal causes immediate hearing loss across the audible frequency range.

Depending on the ability to patch the fistula, recovery ranges from rapid and complete to partial or none. Following stapes surgery, approximately 1% of the patients will have permanent hearing loss over the audible frequency range that is secondary to unknown causes. Procedures involving manipulations at the cerebellopontine angle can damage the cochlear nerve or the internal auditory artery. Injury to these structures can cause either immediate or delayed hearing loss that is usually permanent.

Drug-induced Ototoxicity

Over 130 drugs in clinical use can cause temporary or permanent damage to the human auditory and vestibular systems.⁹³ Commonly used drugs that are ototoxic include diuretics, antiinflammatory agents, aminoglycoside antibiotics, and antineoplastic agents.⁹³⁻⁹⁷ In general, the ototoxicity of a drug is related to the speed of administration, and the dosage, and, most importantly, to the duration of administration. Since most anesthesiologists interact with patients on a relatively short-term basis, it might seem that drug ototoxicity would have little impact on our practice. However, many of our patients are receiving or have recently received drugs with ototoxic potential. Virtually all ototoxic drugs have at least additive ototoxic interactions. It is important, therefore, to be aware of the potential for ototoxicity, identify additional intraoperative risk factors, and be cognizant of possible ototoxic drug interactions. Further, most ototoxic drugs are also nephrotoxic (aminoglycosides, NSAIDs, furosemide, vancomycin, cisplatin), and impaired renal function can increase the ototoxicity of these drugs. It is important for anesthesiologists to be aware of drugs that can cause ototoxicity following a brief course of administration or even a single dose (furosemide, ketorolac) as well as those combinations of drugs whose ototoxicity is synergistic/additive (diuretics and aminoglycosides).^{94,97} Table 5 lists the most commonly used drugs that are associated with ototoxicity.

Salicylates

Salicylates are virtually never administered during anesthesia and are usually discontinued several days before elective surgery. However, emergency surgery may be required for patients who have been taking large doses of salicylates. High-dose aspirin is associated with transient (rarely permanent) symmetrical hearing impairment and tinnitus.^{94,98} The hearing loss and tinnitus increase slowly over days with both the increased dosage and plasma concentrations.⁹⁹ The site of action appears to be at the level of basic cochlear mechanics with audiometric findings consistent with either flat hearing loss or hearing loss in high frequencies.^{98,100} Salicylates

Table 5. Common Drugs Associated with Ototoxicity

Ototoxic Drug	Types	Characteristics of Hearing Loss	Mechanism	Audiometry
Aminoglycosides	Streptomycin, kanamycin, neomycin, amikacin, gentamicin, tobramycin, netilmicin	Hearing loss is delayed, unilateral or bilateral and partially reversible weeks to months after exposure. Neomycin toxicity is usually rapid and profound with more lasting loss.	Hair cells are targeted in an energy-dependent process resulting in hair cell death. Dead hair cells are replaced with scar tissue.	Initially high-frequency SNHL
Erythromycin		Usually only ototoxic following IV administration due to high blood levels. Hearing loss is reversible on discontinuation.	Unknown. Animal studies, cochlear hair cell damage, or effects on central auditory pathways, particularly of the cochlear nerve and cochlear nuclei (causing latency in the V wave of the auditory brain stem response).	Initially high-frequency SNHL.
Vancomycin		Ototoxicity seen only after IV administration.	It has not been proven that vancomycin causes ototoxicity; probably potentiates the ototoxic effect of other drugs.	Initially high-frequency SNHL.
Loop diuretics	Furosemide, bumetanide, ethacrynic acid	Usually sudden onset, bilateral, symmetric SNHL that is usually temporary but occasionally permanent. Usually occurs when high doses are given rapidly. Especially ototoxic in combination with aminoglycosides.	Altered metabolism in stria vascularis, causing swelling and structural changes that result in an alteration of endolymphatic ions and endocochlear potential.	Middle- or high-frequency SNHL.
Salicylates	Aspirin	Reversible (within 72 h), dose-dependent SNHL and tinnitus (toxic when given in doses of > 6 g/d).	Alteration in turgidity and motility of outer hair cells; decreased cochlear blood flow.	Flat or high-frequency SNHL.
NSAIDs	Ketorolac, naproxen, piroxicam	Reversible or irreversible SNHL, less common than with salicylates.	Reversible physiologic changes, no known morphologic changes. Imbalance of vasodilatory prostaglandins and vasoconstricting leukotrienes decreases cochlear blood flow causing tissue ischemia altering sensory cell function.	Flat or high-frequency SNHL.
Antineoplastic agents	Cisplatin, nitrogen mustards, vincristine, vinblastine	Dose-related SNHL, initially high-frequency, bilateral, irreversible. Occasionally following the first dose. Ultra high frequency affected in 100% of patients.	Cisplatin: progressive outer hair cell loss. With high dose inner hair cells, neural structures, and stria vascularis also affected. Nitrogen mustards: only mechlorethamine is ototoxic. Vincristine: loss of hair cells and primary auditory neurons. Vinblastine: loss of inner and outer hair cells only, shrinkage of the organ of Corti.	High-frequency SNHL.

NSAID = nonsteroidal antiinflammatory drug; SNHL = sensorineural hearing loss.

are at least additive with other ototoxic drugs that may be administered during surgery and in such cases can result in permanent impairment of hearing.⁹⁶

Other Nonsteroidal Antiinflammatory Agents

Ototoxicity from nonsteroidal antiinflammatory agents other than the salicylates is rare, and there are only

isolated reports of hearing loss with ketorolac,^{101,102} naproxen,¹⁰³ and piroxicam.¹⁰⁴ Unlike salicylates, when hearing loss occurs with these nonsteroidal antiinflammatory agents, it may be irreversible. Ketorolac is of most interest to the anesthesiologist as it can be given intravenously, resulting in high serum levels. Ketorolac may have both a direct and an indirect ototoxic mechanism. It is thought to be concentrated in the basilar IHC of the organ of Corti, accounting for the high-frequency

losses.^{94,95} There is also an indirect action due to local inhibition of prostaglandin production, which disrupts cochlear blood flow autoregulation.^{103,105,106} It should be remembered that ketorolac has at least additive effects with other ototoxic drugs.

Diuretics

Furosemide, the most frequently used loop diuretic, can cause reversible hearing loss especially when administered concomitantly with aminoglycosides.¹⁰⁷ Furosemide-induced hearing loss may be sudden, especially after large doses (> 240 mg/day) or even a single rapidly administered intravenous dose.^{95,108} To minimize ototoxic blood concentrations (> 50 µg/ml), furosemide should be given slowly, at a rate of less than 15 mg/min, rather than by a rapid bolus injection.^{95,107} If a diuretic response cannot be obtained using these guidelines, substitution of another diuretic, such as bumetanide, can achieve the therapeutic response while minimizing ototoxicity.¹⁰⁷ On a milligram-per-milligram basis, bumetanide is approximately 5 to 6 times more ototoxic than furosemide but 40 times more potent than a diuretic. Therefore, bumetanide can achieve the same diuretic effect with much lower ototoxicity.^{93,106}

Antibiotics

Aminoglycosides

The aminoglycosides are the best known ototoxic antibiotics. They are typically associated with delayed and irreversible loss of hearing, but the characteristics of the hearing loss and the onset can vary among the agents. Hearing impairment caused by the aminoglycosides is due to lesions in the organ of Corti, including the destruction of auditory sensory cells.^{93,94} Because different aminoglycosides have affinities for different groups of hair cells, variable patterns of hearing deficit are encountered. Likewise, the time of onset of toxicity varies widely among the aminoglycosides. For example, ototoxicity due to neomycin may be rapid in onset and profound, while other aminoglycosides have a slower onset of toxicity. Several authors have reported permanent ototoxicity following peritoneal irrigation with solutions containing neomycin.¹⁰⁹⁻¹¹¹ Ototoxic symptoms following administration of amikacin are always delayed a minimum of 4 days.¹¹² Jin and Sheng¹¹³ demonstrated in an animal model that a single dose of gentamicin prolongs the latency and diminishes the amplitude of compound action potentials (the firing mechanism) while hardly affecting the conductive function of the nerve fibers. An examination of 3,506 patients treated with tobramycin identified 21 patients (0.6%) with ototoxicity.¹¹⁴ Nine patients had vestibular symptoms only. The remaining patients had auditory or auditory and

vestibular symptoms. Symptoms subsided within a month for 14 of the 18 patients available for follow-up. Of the remaining four, one had a mild pan-frequency hearing deficit, and the other three had high-frequency losses, one of which exceeded 40 decibels. In the four patients with permanent hearing losses, the authors identified several contributing factors: preexisting renal impairment, prior or concomitant therapy with other ototoxic drugs, or a tobramycin dose of greater than $3 \text{ mg} \cdot \text{kg}^{-1} \cdot \text{day}^{-1}$ for 10 or more days.

Once-a-day dosing can reduce the ototoxicity of the aminoglycosides. There is also evidence that *N*-methyl-D-aspartate antagonists and iron chelators can attenuate aminoglycoside-induced toxicity.^{115,116}

Vancomycin

Intravenous vancomycin has been described as ototoxic, but there is no experimental animal confirmation of vancomycin ototoxicity. In most of the clinical reports of vancomycin ototoxicity, the patients were treated simultaneously with an aminoglycoside drug and/or furosemide. Vancomycin is excreted by the kidneys and is nephrotoxic. This is important because renal impairment can increase the half-life of vancomycin and therefore the possibility of ototoxicity. In one animal study, no evidence of vancomycin ototoxicity was found. However, vancomycin was shown to greatly enhance the ototoxicity of gentamicin.¹¹⁷

Erythromycin

Sensorineural hearing loss after intravenous administration of erythromycin is usually bilateral and reversible. The hearing loss may be sudden and usually affects higher frequencies.^{118,119} Using audiometry, Swanson *et al.*¹²⁰ demonstrated that 30% or more of patients receiving 4 g/day erythromycin will develop symptomatic hearing deficits and tinnitus. Further, as with many other ototoxic drugs, ototoxicity increases with renal disease, hepatic disease, and age.¹²¹

Azithromycin

Azithromycin is frequently used in treating pneumonias, particularly in HIV-infected patients. Little is known about its mechanism of ototoxicity. However, this antibiotic can cause reversible or permanent hearing damage.¹²²⁻¹²⁴

Antineoplastic Agents

Approximately 70% of patients treated with cisplatin sustain permanent hearing loss, usually in the higher frequencies (4,000–8,000 Hz), that is commonly accompanied by tinnitus.¹²⁵ The hearing loss is dose related, and it can occur after a single dose. Cisplatin ototoxicity may be inversely related to the patient's age. Cisplatin

impairs auditory function by preferentially destroying the OHC in the basal turn of the cochlea.¹²⁶ Hair cell loss in the vestibular labyrinth has also been observed. The IHC, neural structures, and the stria vascularis are affected at very high doses.¹²⁷ As noted with other medications, recent exposure to cisplatin can greatly enhance the ototoxicity of other ototoxic drugs administered during anesthesia.

Audiometric Testing of Ototoxic Drugs

Early detection of ototoxicity can be important to the anesthesiologist. Medications given during anesthesia can exacerbate the ototoxic effects of drugs administered prior to anesthesia or can initiate ototoxicity themselves. Audiometry is the most useful tool for early detection of drug-induced ototoxic effects on the cochlea. Drug-induced ototoxicity appears to be most pronounced in the higher frequencies (8,000–20,000 Hz) with progression to the lower frequencies in more advanced toxicity.^{100,127,128} High-frequency audiometry permits earlier detection of the effect of ototoxic drugs. Early recognition is especially important with drugs that can cause irreversible hearing loss (aminoglycosides, cisplatin). Fausti *et al.*¹²⁹ performed conventional (250–8,000 Hz) and high-frequency (9,000–20,000 Hz) serial hearing audiometric threshold monitoring in 123 patients who were treated with aminoglycosides or cisplatin. Of the patients who sustained hearing loss, 63% initially had losses confined to the high-frequency range, 13% had losses only in the conventional-frequency range, and 24% had losses in both frequency ranges.¹²⁹ Thus, by monitoring high frequencies only, auditory deficits were detected early in 87% of the patients. These investigators demonstrated the importance of serial monitoring of high-frequency auditory thresholds for patients receiving ototoxic medications to prevent profound irreversible hearing loss. Finally, Tange *et al.*¹⁰⁰ demonstrated that 100% of patients treated with cisplatin have detectable hearing loss in the ultrahigh-frequency range (9,000–20,000 Hz).

Ototoxic Drugs: Prevention

The anesthesiologist can take an active role in preventing or limiting drug-induced hearing deficits. Anesthesiologists should be aware of patients who are at high risk for developing ototoxicity: (1) patients with impaired renal function; (2) patients with preexisting ototoxic drug serum levels; (3) patients with preexisting SNHL; and (4) patients who could receive a synergistic combination of ototoxic drugs.⁶⁶ Prevention of drug-induced ototoxicity is based on awareness of risk factors, continuing assessment of renal function, avoidance of excessive serum concentrations of potentially ototoxic drugs,

and, when indicated, monitoring auditory function before and during drug therapy. Ototoxicity may cause considerable distress and, in some cases, necessitate discontinuing the offending drug or selecting an alternate to prevent permanent damage.

Miscellaneous Causes of Hearing Loss

Despite evidence that general anesthesia may increase the threshold for noise-induced hearing loss,⁹ excessive noise from surgical power equipment or other sources may produce an SNHL, particularly if the patient is exposed to ototoxic drugs. Damage to the external ear from prolonged or excessive pressure or from direct trauma can obstruct the external auditory canal and cause a unilateral conductive hearing loss. Blockage of the external auditory canal by protective wax or cotton balls, clotted blood, cleansing solutions, or other foreign bodies, such as syringe caps, can cause a similar loss. Inadvertent spillage of certain medications into the canal can cause edema and obstruction of the canal.

Patients brought to the operating room after prolonged intubation in an intensive care unit may develop hearing loss due to middle ear effusion or infection.¹³⁰ This well-known complication may emerge shortly after a surgical procedure. Intraoperative head trauma from positioning, being struck accidentally with a heavy device, or other mishaps can result in either a unilateral or bilateral hearing loss that is conductive, sensorineural, or mixed.

Various phenomena associated with anesthesia and emergence from anesthesia may mimic a hearing loss. Residual muscle relaxant may permit adequate ventilation but prevent the patient from responding to verbal instructions, simulating a hearing loss. Neuroleptic states, such as those occurring after administration of droperidol and certain other drugs,¹³¹ may lead the anesthesiologist to suspect a hearing loss. The central anticholinergic syndrome,¹³² occasionally observed after scopolamine, may give the impression that the patient has sustained a hearing loss. Other nonspecific emergence syndromes causing lack of response to auditory stimuli can be misinterpreted as intraoperative hearing deficits.¹³³

Examination for Hearing Loss

Postoperative hearing loss, whether noticed by the patient, family, or caregivers, in most cases ultimately results in the patient being referred to an otolaryngologist. There are preliminary steps the anesthesiologist can take. Historical information about prior hearing acuity or episodes of hearing problems, hepatic or renal disease, recent medications, anesthetic technique and agents used, type of surgery performed, allergies, and recent

upper respiratory tract infections should be gathered. The patient may be able to relate whether both or only one ear is affected and indicate generally what frequency range is lost. A visual examination of the external ear may reveal evidence of trauma. Foreign material may be visible in the external canal. An otoscopic examination can reveal foreign material (cerumen, blood, prep solutions, cotton balls), inflammation and/or edema, or occasionally a perforated tympanic membrane.

Preliminary testing with a 512-Hz tuning fork can be completed. The Weber test is performed by placing the stem of the vibrating tuning fork at midforehead. If the vibration is louder in the ear with unilateral hearing loss, the loss is most likely conductive. If it is heard better in the normal ear, the loss is sensorineural. In the Rinne test, the vibrating tines of the fork are held several inches from the suspect ear for a few seconds. Then the stem is placed in contact with the mastoid bone. If the vibration is heard better *via* air conduction than by bone conduction, the loss is most likely sensorineural. The loss is conductive if bone conduction is better than air. If the vibration is heard in the other ear, the hearing loss in the ear being tested is severe, and the other ear should also be tested.

Audiometric testing provides more sophisticated insights into hearing pathology. The most basic testing is pure tone audiometry. Continuous tones at frequencies from 125 to 8,000 Hz are presented to the ear. The intensity is increased until the subject indicates that the tone is perceptible. Tone intensity can be varied from -10 to 110 dB. Threshold values for 500, 1,000, and 2,000 Hz are particularly important for speech recognition. Bone conduction testing is done similarly, using either the mastoid bones or the forehead for locating the vibrating apparatus. In general, similar hearing by both air and bone conduction occurs in those with normal hearing and those with sensorineural losses. Conductive or mixed hearing losses result in better bone than air conduction.

Speech tests augment the results of pure tone audiometry. Speech detection threshold, speech recognition threshold, and word recognition scores can be determined monaurally or binaurally. A masking sound in the nontest ear is frequently used. These results are helpful in diagnosing the site of the lesion in the auditory system as well as in managing and evaluating the success of audiological rehabilitation.

The term *acoustic immittance* is used to describe the various measurements made at the plane of the eardrum that provide information regarding the mechanical transfer of sound in the outer and middle ear, cochlear function, and neural integrity of the acoustic nerve, facial nerve, and areas of the brainstem. The tympanogram measures the compliance of the tympanic membrane as a function of pressure applied in the outer ear. It can help identify disruptions in the bony chain or middle ear

fluid. Acoustic reflexes are the bilateral contraction of the stapedius muscles in response to a 70-dB pure tone signal into one ear. The resulting decrease in tympanic membrane compliance is measured. The test checks the integrity of the ipsilateral and contralateral acoustic reflex arc, which includes the outer ear, middle ear, auditory nerve, cochlear nucleus, superior olivary complex, and facial nerve. The acoustic reflex delay is a measurement of the period for which the acoustic reflex can be sustained and assesses acoustic nerve, facial nerve, and cochlear function.

Other audiometric tests include otoacoustic emission testing, which measures, using a small microphone in the external ear canal, the sound waves produced by the cochlea, either spontaneously or in response to a brief acoustic stimulus. Auditory evoked potentials include electrocochleographic, auditory brainstem, and audiometric electroencephalic responses to acoustic stimuli. These tests are beyond the scope of this review, but the interested reader is referred to *Introduction to Audiology* by F. N. Martin.¹³⁴

Summary: Treatment of and Prognosis for Perioperative Hearing Loss

Hearing loss after dural puncture in the low-frequency range is almost certainly due to CSF leak and should resolve completely within days to, at most, weeks (table 1 and fig. 4). If necessary, an epidural blood patch can be effective in hastening recovery.^{4,29,46,55}

Unilateral hearing loss following CPB almost uniformly results in some permanent hearing deficit probably due to embolism and subsequent ischemic injury to areas of the organ of Corti (table 4 and fig. 4). Although there is usually some recovery, there does not appear to be any effective treatment. It is unlikely that any of the proposed treatments achieve a better result than that which occurs spontaneously.¹³⁵ Bilateral hearing loss following CPB is more likely due to one of the causes discussed under general anesthesia.

Hearing loss following general anesthesia for nonbypass surgery does not appear to have a uniform prognosis (table 3 and fig. 4). The outcome is more a function of the specific etiology: CSF leak (ENT and neurosurgery), middle ear barotrauma, embolism, vascular, and pharmacologic. If the specific etiology can be identified, treatment may be effective in certain cases.

Figure 4 summarizes the outcomes following anesthesia-associated hearing losses described in the studies and case reports available in the English literature (tables 1, 3, and 4). It is clear from these data and figure 4 that there are significant differences in outcome as a function of anesthetic technique or etiology. Virtually all hearing impairments resulting from spinal anesthesia (or dural puncture) resolve completely, whereas those occurring

after CPB result in some permanent hearing impairment. The prognosis for hearing loss occurring during general anesthesia without extracorporeal circulation is less certain and may depend on the specific etiology. However, in all instances, any recovery appears to be independent of any type of treatment.^{80,81,135}

Some treatments other than those already mentioned have been advocated, but their effectiveness is highly variable. There is some evidence that hearing deficits resulting from cochlear ischemia may benefit from calcium channel blockers, scavengers of reactive oxygen species, or glutamate antagonists.^{136,137} Sensorineural hearing losses from vascular causes or labyrinthine membrane rupture have been reported to respond to carbon dioxide inhalation or systemic corticosteroids.¹³⁸ These treatments have mixed results, and prevention, whenever possible, is preferable.

Conclusion

Transient subclinical forms of SNHL after anesthesia, especially after subarachnoid puncture, usually go unnoticed by the patient and clinician but probably occur more often than is generally assumed. The true incidence of perioperative hearing impairments, regardless of anesthetic technique, will continue to be unknown unless specifically studied on a large scale using audiometry. However, the anesthesiologist should be increasingly aware of hearing impairment as a perioperative complication, especially in relation to neuraxial anesthesia, where the incidence may be as high as 50%. An awareness of the potential for and the causes of hearing loss during anesthesia may permit the anesthesiologist to prevent or minimize the risk of significant hearing deficits. The suggestion that this risk be discussed in the preoperative period with patients who are at high risk for perioperative hearing loss may be good medical-legal advice.³ A better understanding of the incidence, causes, and prognoses for perioperative hearing loss is essential for the anesthesiologist.

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