Hearing loss after general anesthesia for cecectomy and small bowel resection

Acute hearing deficit following nonotologic surgery and general anesthesia is a rare phenomenon. Hearing loss following anesthesia has more commonly been associated with spinal anesthesia or following cardiopulmonary bypass surgical procedures. This case study describes a patient who developed a left-sided hearing loss after cecectomy and small bowel resection. The postoperative management was directed toward reversing idiopathic hearing loss secondary to a microvascular hypoperfusion etiology. Possible causative factors and the management of sensorineural hearing loss following anesthesia are discussed.

Key words: General anesthesia, nonotologic surgery, sensorineural hearing loss.

Introduction
Sensorineural hearing loss (SNHL) has an incidence of 0.4% after spinal anesthesia, a 0.1% incidence after general anesthesia for coronary artery surgery where cardiopulmonary bypass (CPB) is employed, and even less after general anesthesia for nonototic, noncardiac surgery. This compares with an estimated incidence of 0.02% in the general population. Nitrous oxide has been implicated in hearing loss following general anesthesia, secondary to pressure changes in the middle ear. Frequently, the etiology of sensorineural hearing loss cannot be identified, which leads the clinician to consider a vascular or viral origin.

This report describes an unusual postoperative complication of sudden SNHL after general anesthesia for cecectomy and small bowel resection. Although the surgical procedure and anesthetic course appeared uneventful, the day following surgery the patient reported a left-sided hearing loss. The patient had no history of hearing disorder or otologic disease, nor could any exposure to ototoxic medications be implicated.

Case summary
A 39-year-old male with a 6-hour history of abdominal pain was admitted to the hospital for observation. He had experienced several previous episodes of similar discomfort, all of which had lasted several days. His past medical history was otherwise unremarkable, and his hematologic studies were normal except for an elevated white blood cell count of 12,400. He appeared to improve over the next few hours, but then developed a fever of 103°F with persistent abdominal symptoms. A decision was made to proceed with an exploratory laparotomy under general endotracheal anesthesia.
Following preoxygenation, general anesthesia was induced with fentanyl 100 μg, thiopental 4.5 mg/kg, and atracurium 0.5 mg/kg to facilitate endotracheal intubation. Cricoid pressure was applied to guard against aspiration. Anesthesia was maintained with isoflurane 0.5%-3% and 100% oxygen. Nitrous oxide was not used to avoid further distension of the bowel.

Patient monitoring included automated noninvasive blood pressure, electrocardiogram, pulse oximetry, precordial stethoscope, and peripheral nerve stimulator. Blood loss during the procedure was estimated at 100 mL, and crystalloid replacement consisted of Ringer's solution 2,600 mL.

The course of the anesthetic was uneventful, and hemodynamic parameters remained within normal limits throughout the perioperative period. His blood pressure in the operating room was never lower than 98/64 nor higher than 136/88. His oxyhemoglobin saturation values via pulse oximetry (SpO₂) were from 99% to 100%, except upon his arrival to the operating room, when it was 95% while breathing room air.

At the conclusion of the surgical procedure, the patient was breathing spontaneously without reversal of the neuromuscular blockade. He was extubated and taken to the postanesthesia care unit in satisfactory condition. His vital signs were blood pressure 126/90, pulse rate 80, respiratory rate 16, and SpO₂ 100% on 3 L/min of oxygen by nasal cannula.

A diagnosis of Crohn's disease involving primarily the distal ilium and cecum was made, and surgical excision of the affected areas was performed. Postoperatively, the patient did well, and his recovery was uneventful, except that on the day following surgery he reported complete hearing loss in the left ear. This was a new occurrence, and he had no history of prior otologic disease or hearing disorder. He was evaluated by his primary care physician, and an otologic examination revealed no visible abnormalities.

Two weeks after surgery the patient was seen by an otolaryngologist who, after conducting evaluative studies, including audiograms, made the tentative diagnosis of left sudden sensorineural hearing loss attributable either to general anesthesia or surgery. The hearing loss was presumed to be of microvascular perfusion etiology, and the patient was placed on papaverine 300 mg per day orally and acetylsalicylic acid 325 mg orally twice daily for 1 month. The audiograms were then repeated. No improvement in hearing was noted after 1 month, and on a follow-up visit 6 months later, only a mild improvement had taken place.

Discussion

The vestibulocochlear nerve (cranial nerve VIII) consists of two sensory branches: the vestibular nerve, concerned with equilibrium, and the cochlear nerve or auditory nerve. These two branches of the vestibulocochlear nerve are joined into a common trunk which enters the internal acoustic meatus along with the facial nerve. Blood supply to the internal ear (labyrinth) arises from the basilar artery and the posterior auricular artery.

Sudden sensorineural hearing loss is a common problem encountered by the otolaryngologist; however, when associated with general anesthesia, it is a rare occurrence. Possible nonsurgical etiologies include head trauma, ototoxic medications, meningitis, ear tumors, and cerebellopontine tumors.

Hearing loss related to anesthesia has more often been associated with spinal anesthesia, where the acute hearing deficit is temporary and usually resolves without treatment within a few days. The causative mechanism appears to be related to cerebrospinal fluid (CSF) escaping into the epidural space through the dural puncture and subsequent decrease in CSF pressure.

Lee and Peachman reported one case of complete unilateral loss of hearing on the second postoperative day after spinal anesthesia for cesarean section which persisted until an epidural blood patch was performed on the fourth postoperative day.

Shortly after the blood patch was applied, the patient's hearing began to improve, and it was completely and permanently restored within 1 hour. Treatment was directed toward restoring the CSF pressure to normal levels. Panning and associates treated one patient with an infusion of low molecular weight dextran and pentoxifylline in an attempt to improve circulation to the ear.

Nitrous oxide anesthesia has also been implicated in sensorineural hearing loss and related to the pressure changes that take place in the middle and inner ear when nitrous oxide is administered. Hockerman and Reimer in their review of the literature found only four cases of SNHL with no prior history of ear disease, documented with hearing loss after nitrous oxide anesthesia, in which neither otological nor cardiac surgery was performed.

The first report of SNHL following CPB was made by Arenberg et al in 1972. Other cases of hearing loss following CPB have since been reported.

There is a possible direct correlation between surgical procedures requiring CPB and periopera-
tive SNHL. There are at least two possible explanations for the hearing loss associated with CPB surgery: microembolic phenomena and general perfusion failure.

It is theorized that atherosclerosis in the basilar artery system, in combination with less than normal perfusion pressures, results in a localized perfusion deficit. Millen and colleagues suggest that the probability of an embolic phenomenon appears stronger than hypoperfusion, because microemboli have been demonstrated at autopsy in the lung, kidney, and brain of more than 50% of patients who died following surgery requiring CPB.

The management of sudden SNHL has been varied. Some of the modalities have included anticoagulation therapy, corticosteroids, vitamins, low molecular weight dextran, vasodilators, and stellate ganglion block.

Three cases of acute hearing loss in which the surgery was noncardiac and nonotologic have been reported more recently. Two of these patients received no specific therapy for their hearing disorders and realized almost complete recovery of their hearing. The third patient was treated initially with amoxicillin (the erroneous diagnosis of otitis media was made) and later treated with carbogen inhalation. Carbogen, a mixture of carbon dioxide and oxygen, was used in an attempt to increase cerebral blood flow, but the patient's hearing did not improve significantly. No specific cause was determined for the hearing loss in any of these patients.

This patient's hearing was ostensibly normal prior to surgery. A review of the surgical procedure and anesthesia record revealed no predisposing events which might account for the hearing loss. No known ototoxic medications were used during or after surgery nor taken preoperatively.

The SNHL which occurred in the patient in this case report probably represents a coincidence, not unlike idiopathic sudden hearing loss. After approximately 18 months, the patient continues to demonstrate the same degree of hearing deficit.

REFERENCES

AUTHOR
Frank A. Velazquez, CRNA, BS, staff anesthetist at McKee Medical Center, Loveland, Colorado, earned a BS degree in Medical Technology from Madison College, Madison, Tennessee, in 1963; an AD degree in Nursing from Western Kentucky University, Bowling Green, in 1967; and a diploma in Anesthesia from Middle Tennessee School of Anesthesia in 1969. He is currently an MS candidate in the University of Kansas Medical Center's Nurse Anesthesia Education Program, Kansas City, Kansas.

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