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Unilateral sensorineural hearing loss after arthroscopic shoulder surgery in the beach-chair position: a case report

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Sudden hearing loss after nonotologic surgery is rare and is defined as a loss of greater than 30 decibels (dB) in three contiguous frequencies that occurs within three days of surgery.[1,5,32] Most reports of hearing loss after surgery involve patients undergoing cardiopulmonary bypass for open heart surgery and are attributed to either a hypoperfusion "watershed" infarct or embolic phenomenon to the vasculature supplying the vestibulocochlear nerve.[1,22,24] However, there are also numerous reports of acute sensorineural hearing loss after nonotologic, non-cardiopulmonary bypass surgery.[5] The etiology is unclear and may be multifactorial, but hypotheses include transient cerebral hypoperfusion, labyrinthine membrane rupture (LMD) secondary to nitrous oxide (NO) anesthetic use, cerebrospinal fluid (CSF) leak after spinal anesthesia or dural tear in spine surgery leading to traction on the vestibulocochlear nerve, recent viral illness, and air or tissue embolus leading to an acute infarct.[28] Most of these cases in the orthopedic literature include patients with sudden hearing loss after spine surgery in the prone position.[5,12,13,18,19,23,28] However, reports also exist of hearing loss after surgery in patients in the supine position.[8,27,30]

In shoulder surgery, the beach-chair position is commonly utilized for arthroscopy. This position presents with it the concern for cerebral hypoperfusion, especially in elderly frail patients with an impaired cerebral vasculature autoregulation mechanism. Although there have been reports of hypoglossal nerve palsy (CN XII) and unilateral vision loss/opthalmoplegia (CNII) after arthroscopic shoulder surgery in the beach-chair position, there are no reports of sudden sensorineural hearing loss (CNVIII).[2,20] We describe the case of a patient with unilateral sensorineural hearing loss after arthroscopic rotator cuff repair, performed in the beach-chair position. The patient consented for the publication of this case report.

Case

A 57-year-old right-hand-dominant woman presented with six months of worsening right shoulder pain and weakness which began after a motor vehicle collision. She has a past medical history significant for arrhythmogenic right ventricular dysplasia with an automatic implantable cardiac defibrillator, patent foramen ovale with closure, and history of two previous transient ischemic attacks (TIAs) with unclear etiology. Computed tomography angiography demonstrated no obvious carotid artery disease, and a 2-mm aneurysm from the left distal anterior cerebral artery. Her TIAs were managed with Plavix. The patient's pain and shoulder dysfunction persisted despite formal physical therapy and nonsteroidal anti-inflammatories. She had significant night pain and her subjective shoulder value was 30%.

On physical examination, she was tender to palpation over the acromioclavicular joint, greater tuberosity, and biceps tendon in the groove. Her baseline range of motion with forward elevation of 150 degrees, external rotation 45 degrees, and internal rotation to T10. Special testing revealed a positive Speed's test, positive empty can test, and positive cross-body adduction test. She had 4/5 weakness in forward flexion and 4/5 in external rotation testing. Advanced imaging (MRI) shows full-thickness rotator cuff tear measuring ~2.5cm with retraction to the mid humeral head involving both the supraspinatus and infraspinatus tendon. There was no fatty infiltration involving the rotator cuff musculature. She was indicated for right shoulder arthroscopic surgery. Given her multiple medical comorbidities, her primary care physician and cardiologist

Institutional review board approval was not required for this case report.

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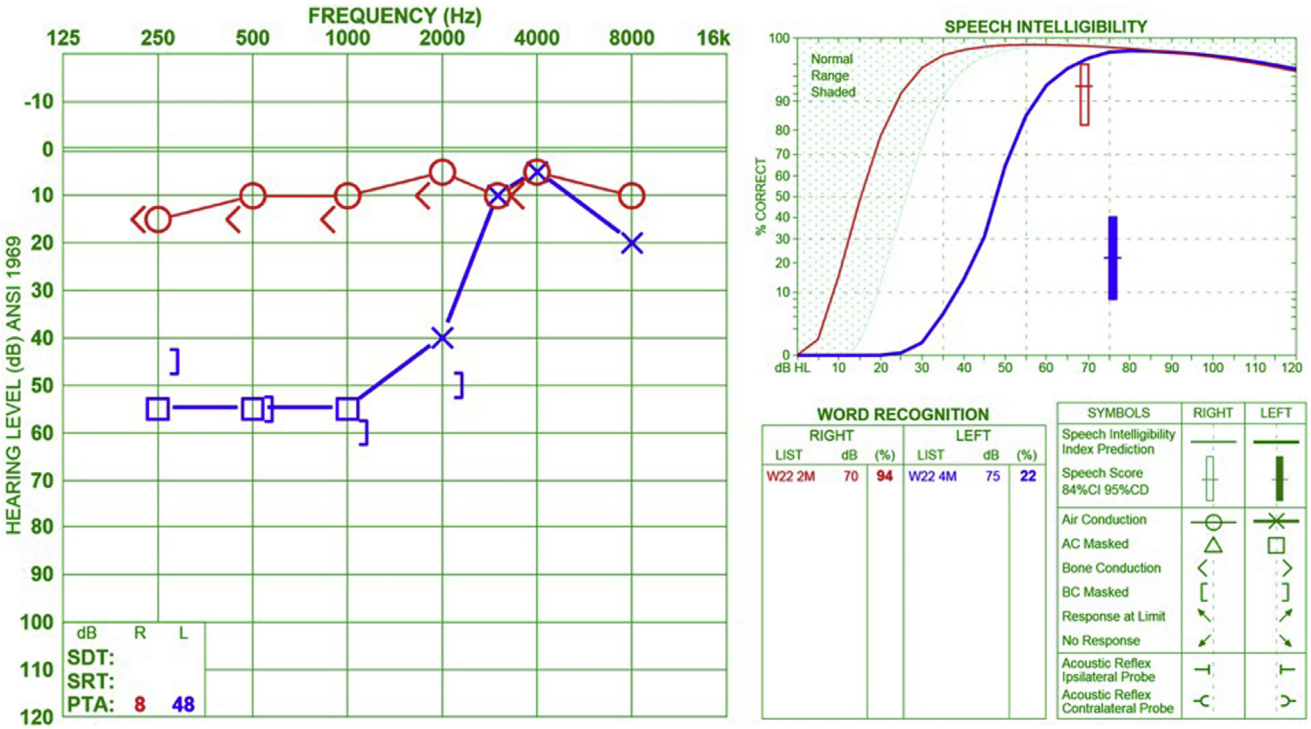

Figure 1 Audiology report of our patient showing significant decrease in hearing levels in the left ear with low dB and also at lower frequencies as compared with the right ear. Furthermore, word recognition was 22% on her left ear compared with 94% in her right ear.

provided preoperative clearance and recommended stopping aspirin seven days before surgery and Plavix the day before surgery, and then restarting both medications the morning after surgery.

She underwent general anesthesia with a preoperative interscalene nerve block and was placed in the beach chair position (60 to 70 degrees of inclination) with the right upper extremity secured in an articulating arm holder. General anesthesia was maintained with both sevoflurane and NO. The arthroscopic-assisted acromioclavicular joint resection, biceps tenodesis, subacromial decompression, and rotator cuff repair was performed without complication. Review of the anesthesia records revealed an operative time of 76 minutes, average mean arterial pressure of 77.86 mmHg, and that the lowest blood pressure during the operative case was 95/59 mmHg.

On the morning of the first postoperative day, the patient reported left-sided hearing loss which persisted until her first postoperative visit, at which point she notified the surgical team. She was then evaluated by an otolaryngologist about ten days after surgery with a formal audiology examination. Inner ear examination demonstrated no abnormalities and no signs of infection. The patient was diagnosed with sudden sensory auditory deficit of the left ear. Her word recognition was 22 percent of normal. Figure 1 summarizes the results of the audiology report. Per the otolaryngologist, she was given prednisone 60 mg for 14 days, with a taper dosing regimen for 5 days after. Despite this medical management, her hearing did not improve. At her final follow-up, her hearing loss persisted.

Discussion

Hearing loss after surgery is a rare complication with most nonotologic cases occurring in patients undergoing cardiopulmonary bypass. Plasse et al[24] reported 7 of 7000 (0.1%) patients who underwent cardiopulmonary bypass developed hearing loss postoperatively. The authors attributed the mechanism of hearing loss to an embolus of either air, antifoam, fat, or particulate matter from calcified valves and the aorta. In the orthopedic literature, most of sensorineural hearing loss cases have occurred after spine surgeries in the prone position.[5,12,28] One case includes a patient with a history of migraines and sinus bradycardia who underwent lumbar surgery and experienced unilateral severe sensorineural hearing loss postoperatively which remained unchanged one year later. Another patient undergoing lumbar surgery for spinal stenosis under general anesthesia had a unilateral decrease in speech reception threshold postoperatively. This patient's medical history included lymphoma treated with chemotherapy, atrial fibrillation, and peripheral neuropathy. No improvement with hearing was reported at final follow-up.[5] Finally, a patient underwent a L2-L5 laminectomy for spinal stenosis under general anesthesia with a noncontributory medical history and experienced a unilateral decrease in speech recognition threshold which persisted to 14 weeks with no improvement.[12] The etiology for these sudden sensorineural hearing loss in orthopedic surgery has been attributed to a number of mechanisms including CSF loss as occurs in dural tears or spinal anesthesia leading to brain sagging and traction on the vestibulocochlear nerve, NO anesthetic use resulting in LMD, valsalva maneuvers or high airway pressures during the surgery leading to high pressures in the middle ear, hypotension, or microemboli. Table I summarizes all reports of hearing loss after orthopedic surgery and their respective proposed etiologies. To our knowledge, there are no published reports of sensorineural loss after shoulder arthroscopy surgery.

Arthroscopic shoulder surgery can be performed in either the lateral decubitus or beach-chair position.[16,17] Although there are advantages inherent to each position, the benefits of the beach-chair position include an upright anatomic position, ease of conversion to an open approach, and the ability to manipulate the operative arm in space to allow optimal positioning for surgery.[17,29] By contrast, the main disadvantage of the beach-chair position, especially important in elderly patients with comorbidities, include

Table I
Review of cases of sensorineural hearing loss after orthopedic surgery.

Author	Year	Surgery performed	Use of nitrous oxide	Hearing loss (left/right/bilateral)	Proposed etiology for hearing loss	Outcome
Mallepally et al.[19]	2019	Posterior stabilization of T10-L1 for ankylosing spondylitis	Yes	Left	Unknown	No improvement
		L4-5 transforaminal lumbar interbody fusion	Yes	Left	Unknown	No improvement (IV steroids)
		Decompression and stabilization for tuberculous spondylodiskitis	Yes	Right	Unknown	No improvement
Goodrich et al.[13]	2018	Lumbar spine decompression (L2-5) and fusion (L3-5)	Yes	Right	Hypotension, positioning, microemboli from the cell-saver, and nitrous oxide	Hyperbaric O2 (mild right side improvement)
Vilhena et al.[30]	2016	Total knee arthroplasty	No	Bilateral (L>R)	**Spinal and epidural anesthesia**: loss of CSF leads to drop in intracranial pressure transmitted to the inner ear perilymph via the cochlear aqueduct	Hyperbaric O2 therapy: audiogram to 90% normal on left at follow-up
Sahin et al.[27]	2015	Hallux valgus surgery	No	Bilateral	**Spinal anesthesia:** CSF leads to drop in intracranial pressure transmitted to the inner ear perilymph via the cochlear aqueduct	Prednisone therapy & Hyperbaric O2: Resolved by 3rd postoperative day
Park et al.[23]	2006	Lumbar spine decompression with L2-S1 instrumented fusion	Yes	Left	Elevated airway pressures or excessive straining	Mild improvement
Mak et al.[18]	2003	L3-S1 posterior decompression and fusion	Yes	Right	Dural tear leading to loss of CSF and drop in intracranial pressure	Saline syringing to external auditory canal; No improvement at 18 months follow up
Girardi et al.[12]	2001	L2-L5 laminectomy	Yes	Bilateral	Nitrous oxide use leads to elevated pressures in the middle ear and labyrinthine membrane rupture (LMR)	Bilateral cerumen impactions were removed; Mild improvement but required hearing aids
		L3-L5 laminectomy with L3-S1 fusion	Yes	Right		Significant improvement
De La Cruz et al.[8]	1998	Open reduction and pinning of metatarsal	Yes	Bilateral	Unknown	Oral steroids and bedrest; No improvement
Cox et al.[5]	1997	Lumbar spine surgery	No	Right	Unknown	No improvement (salt restricted diet, hydrochlorothiazide, triamterene, prednisone)
		Lumbar spine decompression	Yes	Right		Unknown
Segal et al.[28]	1984	Lumbar disc herniation surgery	Yes	Left	Nitrous oxide use leads to elevated pressures in the middle ear and labyrinthine membrane rupture (LMR)	No improvement

an increased risk of hypotension and bradycardia leading to cerebral hypoperfusion and potential "watershed" infarct.[17] Deliberate hypotensive anesthesia is an effective technique to reduce blood loss and optimize arthroscopic visualization during subacromial decompression and rotator cuff repair. However, potential cerebral hypoperfusion and complications related to cerebral or brainstem infarct have been attributed to this technique.[26] In our case, the patient's lowest blood pressure was 95/59 mmHg with a heart rate of 65, and this was corrected by phenylephrine. Her mean arterial pressure was 77 and maintained throughout the case. Hypotensive bradycardic episodes are defined as a 1) decrease in heart rate of at least 30 beats per min within a 5-minute interval, 2) any heart rate less than 50 beats per min, and/or a 3) decrease in SBP of greater than 30 mm Hg within a 5-minute interval or any systolic pressure less than 90 mm Hg.[7] The patient did not meet any of these criteria; therefore, it is unlikely that her hearing loss was secondary to hypotension during the arthroscopic surgery. However, when operating on a patient with an extensive past medical history, the choice of surgical position should be considered. It is possible that the lateral decubitus position would have maintained better perfusion than the beach chair position and thus prevented the brief hypotensive state that our patient experienced. However, the hypotensive state that our patient experienced did not meet the criteria for hypotensive bradycardic episode and unlikely to have contributed to her hearing loss.

Another proposed etiology for hearing loss after orthopedic surgery is due to loss of CSF, either from a dural tear in posterior spine surgery or spinal anesthesia.[18,27,30] An alteration in CSF levels is hypothesized to lead to hearing loss due to either an imbalance in intracochlear fluid levels or a stretch injury of the vestibulocochlear nerve resulting from brain sagging.[18] However, our patient did not receive spinal anesthesia and has no clear reason to have had her CSF levels perturbed during the arthroscopic shoulder surgery, so this is unlikely for our discussed scenario. Other authors have conjectured that NO use as a component of the general anesthetic may lead to a pressure imbalance in the middle ear and subsequent hearing loss.[12,13,28] NO dissolves more quickly into blood than oxygen and quickly displaces nitrogen in enclosed cavities such as the middle ear. This can lead to a rapid increase in pressure causing LMD and subsequent hearing loss. However, this mechanism is controversial as there are a number of cases of hearing loss in patients without NO use.[5,27,30]

In our patient, it is possible that the most likely etiology of the unilateral sensorineural hearing loss was a thromboembolic phenomenon related to her multiple comorbidities. The patient's medical history was significant for arrhythmogenic right

ventricular dysplasia with implanted automatic implantable cardiac defibrillator, patent foramen ovale with closure one year prior, and two TIAs 2 years prior. As per the cardiology recommendations, she discontinued her Plavix regimen immediately before the surgery. In addition, she stopped aspirin for 7 days before the surgery. Although the effect of these medications lasts for a period beyond their termination, the patient may have been at a slightly increased risk of coagulation intraoperatively and may have developed an embolism, a possibility which has been documented in those receiving shoulder arthroscopy in the beach chair position.[3,4,6] Furthermore, the patient had a patent foramen ovale closure 1-year before her shoulder surgery. Despite the repair, the existence of an incompletely closed patent foramen ovale should be considered. Emboli may have passed from the venous circulation and into the cerebral circulation via the patent foramen ovale, causing ischemia or an infarction which could contribute to the patient's hearing loss. However, without advanced imaging or formal thromboembolic workup from that day, we can only conclude that the cause of her hearing loss is likely attributable to a thromboembolus. In addition, an air embolism passing through the foramen ovale is another consideration. Air emboli arising from arthroscopic shoulder surgery in the beach chair position have also been documented and are worthy of consideration.[11,15] Despite taking all of the preoperative precautions and obtaining consultation with cardiology, it is very difficult to prevent this complication given the patients preoperative extensive medical history.

The management of sudden sensorineural hearing loss has not been standardized in the literature. Many treatment options have been reported for otologic etiology which includes oral steroids, intratympanic steroids, antiviral agents, vasodilators, and hyperbaric oxygen therapy.[9,10,21] Oral or IV corticosteroids in high doses administered during a 10- to 14-day period is the classic treatment of patients with sudden hearing loss. However, only 61% patients will recover their hearing after the initial systematic steroid therapy.[31] Intratympanic steroid injections directly to the inner ear have been used in patients who did not improve with systematic steroids; however, Haynes et al reported only 27% of patients showed improvement after treatment.[14,25] In addition, complications have been reported, including tympanic membrane perforations and infections. Hyperbaric oxygen therapy is another option for treatment. In nineteen patients with idiopathic sudden hearing loss, Muzzi et al[21] found that hyperbaric oxygen therapy with pure oxygen at 2.5 atmospheres absolute pressure administered for 90 minutes and for 30 sessions appeared to improve these patients' pure tone hearing thresholds particularly at the low frequencies. In our patient, given her etiology is likely a thromboembolic phenomenon, a course of 60 mg prednisone for 14 days followed by a 5-day taper was recommended by the otolaryngologist as the best treatment option.

Conclusion

Although there are many proposed mechanisms of hearing loss after surgery, the etiology for this patient likely is related to her cardiac-related morbidities. These preexisting conditions along with the temporary discontinuance of her Plavix and aspirin may have led to a hypercoagulable state during surgery and created a paradoxical embolus due to her patent foramen ovale. Although hearing loss after orthopedic surgery is rare, it is important to consider as a possible complication and conduct appropriate preoperative counseling. Depending on the etiology for the sudden hearing loss after surgery, treatment options include systemic steroids (oral or IV), direct intratympanic steroid injection, vasodilators, or hyperbaric oxygen treatment.

Conflicts of interest

The authors, their immediate families, and any research foundations with which they are affiliated have not received any financial payments or other benefits from any commercial entity related to the subject of this case report.

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Patient consent

Obtained.

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