PERILYMPHATIC FISTULA INDUCED BY BAROTRAUMA

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ABSTRACT

The association between diving, barotrauma, and the production of perilymphatic fistula has been known for almost 20 years. Forty-eight cases of round and oval window fistulas following diving have been reviewed and essentially corroborate previous findings. Any patient with a history of diving and subsequent sensorineural hearing loss within 72 hours should be suspected of having a round or oval window perilymphatic fistula and surgical exploration and closure of the fistula should be undertaken. Patients who have a loss of hearing, vertigo, nausea, or vomiting following a decompression dive should be re-compressed and if symptoms do not clear, exploration should be performed. Surgical treatment should be executed as soon as possible after the diagnosis is suspected for the best possible results.

Since the advent of the self-contained underwater breathing apparatus (SCUBA), more than 2 million divers have been trained and certified in the United States. Many problems associated with diving have been encountered by otolaryngologists in the recent past. Major problems such as decompression sickness (BENDS) and high-pressure nervous syndrome (HPNS) fortunately are rare.

The general gas law states that the initial pressure, times the initial volume, divided by the initial temperature, equals the final pressure, times volume, divided by temperature.[1] This is important in the sinus cavity and in the middle ear, because as pressure increases as the diver goes deeper, volume decreases. As pressure decreases as the diver ascends to the surface, the volume expands producing a change within tissues caused by the expansion or contraction of air. This is called barotrauma.

Barotrauma is the most common medical condition arising from diving and it is directly attributable to change in the pressure and volume of air within the body. To maintain an airspace at the original volume when the individual descends deeper in the water and pressure increases, air must be added to the closed airspace. The amount of air required is directly proportional to the pressure. If the volume of air exceeds the capacity of the container, the container may be ruptured by the expansion of air. During descent, pressure increases and pushes inward on body surfaces such as the tympanic membrane. If an airspace is not equalized, negative pressure is established and barotrauma results. This is commonly called an ear *squeeze.* When a squeeze affects an airspace in the body, pain results until increased air volume is added to that airspace to make the air pressure equal to the total pressure outside. Figure 1[2] illustrates the otologic barotrauma of descent showing the theoretic sequence of changes in an ear of a diver. Figure 1A shows the surface condition of the ear with equal pressures within the middle ear and the external ear canal of 760 mm Hg. Figure 1B shows a depth of approximately 2.6 feet after the diver has failed to open the eustachian tube as he descended. The middle ear pressure is still 760 mm Hg and the external auditory canal has a pressure of 820 mm Hg. There is a pressure differential of 60 mm Hg, which pushes the tympanic membrane and the stapes into the inner ear and bulges the round window membrane into the middle ear because of the decreased pressure compared to the pressure of perilymph. Figure 1C shows a depth of approximately 3.9 feet with 90 mm Hg pressure differential with a blocked and locked eustachian tube. With a forceful Valsalva maneuver, such as lifting something under water or increasing the pressure within the perilymphatic space, the round window will rupture as in Figure 1D. Studies in cats have shown that the round window may rupture at an increased pressure of 120 to 300 mm Hg.[3] With continued descent, tympanic membrane rupture or even round window rupture may be produced.

Ascending from depth causes a relative increase in pressure within the middle ear and eustachian tube that, unless equalized, also can cause a rupture of the round window membrane. Goodhill[4] has called the rupture caused by an increase in perilymphatic pressures an "explosive" round window rupture, and the rupture of the round window as a result of increased pressure within the middle ear, an "implosive" rupture.

The first documented occurrence of round window membrane rupture in a diver was presented at the American Academy of Ophthalmology and Otolaryngology in 1971 by this author.[5] Rupture of the round window attributable to barotrauma and subsequent perilymphatic fistula (PLF) has since been documented by many other authors. Patients with

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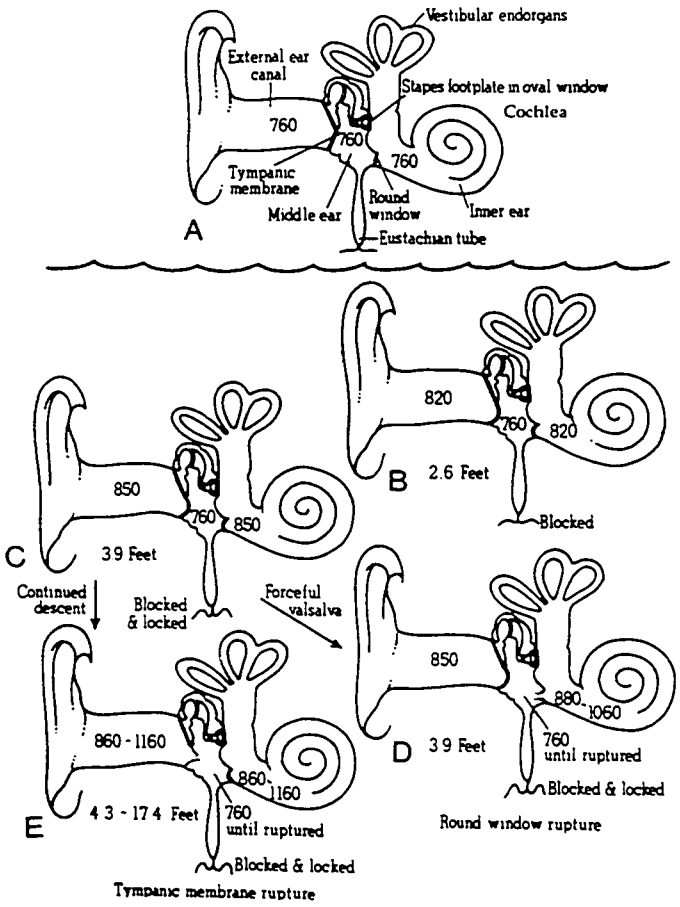

Figure 1. Otologic barotrauma of descent. Theoretic sequence of changes in the right ear of a diver who does not equalize middle ear pressure during descent. Pressures are shown in mm Hg. *A*, Surface condition with equal pressures. *B*, Depth of approximately 2.6 feet after diver failed to open the eustachian tube. Pressure differential of 60 mm Hg exists. Tympanic membrane and round window are bulging into the middle ear. Pain, pressure, and possible vertigo. *C*, Depth 3.9 feet with 90 mm Hg pressure differential and blocked eustachian tube. *D*, Forceful Valsalva with possible rupture of round window and leak of perilymph into the middle ear. *E*, Continued descent may lead to tympanic membrane rupture at pressure differentials of as low as 4.3 feet. (From Farmer JC Jr: Ear and sinus problems in diving, In: Bove A, Davis J, eds. Diving medicine. Philadelphia: WB Saunders 1990 with permission.)

PLF have been found to have varying degrees of sensorineural hearing loss (SNHL). These include total SNHL or only a mild high-frequency loss or any degree between. Vertigo or dizziness is not a major problem in the diver, although it does present as a symptom in approximately 30 percent of patients.

METHODS

Between 1971 and 1989 sixty-two patients with acute barotrauma of the ear and suspected round or oval window fistula were examined and treated. Of these, 48 underwent surgery for PLF. Of this group only two cases were caused by pressure changes as a result of flying and the rest were attributable to diving.

Surgical correction of the PLF was based primarily on the history of a nondecompression diving injury with continued hearing loss or vertigo. Of the 14 patients who did not have surgery, most of them were beginning to have a return of hearing at the time of our examination, or had a hearing loss limited to the high frequencies. We have shown no improvement in these patients by treatment.

Audiologic studies showed three patients to have a low-frequency SNHL, 17 a high-frequency loss, and the most a flat 50-dB loss or greater. Fourteen of the 48 had some complaint of dizziness that ranged from true vertigo to a mild instability. After closure of the PLF that was visualized by the surgeon by using Gelfoam pledgets and Valsalva maneuver or was easily seen with no increased pressure, 29 of the 48 patients were left with a mild to moderate high-frequency SNHL. Recognition of the PLF may be made easier in the future by using intravenous or intrathecal fluorescein. Our theory is that trauma to the cochlea may be caused by an air-bubble within the cochlea and that the higher frequencies are more susceptible to this damage, as in noise-induced hearing loss. Thirty-one patients presented with profound SNHL and after closure of PLF only two remained with severe SNHL. Twelve of these continued to have a mild high-frequency SNHL, but with good discrimination ability.

CASE REPORTS

Case 1

A 40-year-old diver had been diving to approximately 30 feet; he noted no ear symptoms at the time of the dive. The day following the dive he began noticing a feeling of blockage in the ear. He had had some difficulty in clearing the ear while diving, but did not feel this was serious enough to seek treatment until 3 days after the dive. At that time, he noted almost total sensorineural hearing loss in the left ear.

Physical examination was normal. The tympanic membrane and eustachian tube function were intact. No dizziness was encountered. Fistula test was negative. Electronystagmography and posturography were not performed. Surgical exploration of the middle ear was performed and a round window fistula was found in the center of the round window. There was no true niche found in this case or in most of the divers operated on by the author. The patient had a round window perilymphatic fistula repaired with Gelfoam and his hearing returned to normal. He has subsequently been diving for the past 19 years (against medical advice) but has had no further loss of hearing.

Case 2

A 15-year-old was diving to 25 feet and noted a sudden hearing loss when he was unable to clear the

ear on ascent. He was found to have a total SNHL on examination. After 5 days of bedrest, there was no improvement in hearing so the ear was explored. Round and oval window PLF were found. Fibrous fatty tissue was used to close these and his hearing returned to normal. This patient's brother had a similar episode 3 years later, which was also corrected.

DISCUSSION

In our original series, 17 patients with sudden sensorineural hearing loss secondary to PLF associated with barotrauma were treated.[6] Sixteen of those cases underwent surgery, and of those cases with flat audiometric sensory losses, nine of eleven showed improvement of the hearing to within 10 dB of the normal ears within 2 weeks of surgery. Six of the 17 cases with SNHL limited to high frequency did not improve.

In our present series we have added an additional 31 patients, making a total of 48 patients with perilymphatic fistula attributable to barotrauma who have been surgically treated. The findings of the original paper have been corroborated in the additional 31 cases. Those findings may be summarized as follows.

In divers with total or near total SNHL following a nondecompression dive, immediate surgery is indicated with closure of the perilymphatic fistula. If closure is delayed more than 2 weeks, the possibility of return of hearing in the affected ear is limited.

In patients whose hearing loss has been limited to the high frequencies, closure of perilymphatic fistula does not improve hearing, and therefore this is a relative contraindication to surgery, unless vertigo is an issue.

Approximately 30 percent of our patients (14 of 48 patients) had vestibular symptoms as well as hearing loss and these individuals had an improvement of their vertiginous episodes after surgery. One patient has had occasional episodes of benign positional vertigo, but has refused further exploration of the ear. Only one patient with a total SNHL did not have any return of function of the ear.

The findings at operation have almost invariably included a round window membrane that was at a more lateral angle than the normal, making the visualization of the round window membrane easy.

Electronystagmographic testing as well as posturography have been of little value in these cases. The best diagnostic criteria is a history of barotrauma and a suspicion of fistula with total or near total SNHL. Patients with hearing loss limited to high frequencies should not be explored unless they have continued vertigo or unsteadiness with vestibular signs.

If a diver presents with signs of inner ear injury such as SNHL and has not been subjected to a dive requiring recompression, a round window perilymphatic fistula should be suspected. Exploration for a possible PLF and, if found, closure with a solid tissue graft should be performed. Divers with vertigo or dizziness should be questioned carefully about their dizziness. If a PLF is a possibility, they should be placed at bedrest for 5 days, and explored if symptoms do not clear.

In our experience, sudden sensorineural deafness after diving has not been a sign of decompression sickness. Although the disease of decompression sickness of the ear has been reported previously,[7] it is theorized that many cases of inner ear decompression sickness are actually cases of round window membrane rupture in which air-bubbles enter the inner ear through the round or oval windows and are not true nitrogen bubbles within the inner ear vascular system. Recompression would help these cases by decreasing the size of the bubble in the inner ear, be it air or nitrogen. Obviously, further research is necessary to differentiate between the two injuries.

Any diver with ringing in the ears, loss of hearing, vertigo, dizziness, nausea, and vomiting following or during a dive in which decompression sickness is possible, according to the standard diving decompression tables, should be considered as having decompression sickness and treated appropriately in a recompression chamber. The same symptoms following a nondecompression dive should suggest a round or oval window PLF and surgical intervention should be undertaken immediately.

SUMMARY

The diagnosis of round or oval window membrane rupture and subsequent perilymphatic fistula caused by barotrauma, such as diving or flying, is a known and accepted clinical entity. It has been found that immediate closure of the fistula secures the best clinical result.

Any patient with a history of diving and sudden sensorineural deafness within 72 hours should be suspected of having a round or oval window perilymphatic fistula attributable to barotrauma and surgical exploration and closure of the fistula should be undertaken immediately. Those with continued vertigo or dizziness and no hearing loss also should be suspected of having a PLF and explored.

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