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Intranasal forces and labyrinthine deformations and fistulae

Victor Goodhill, Los Angeles, California, U.S.A.

The labyrinth with its delicately balanced perilymph-endolymph system, intimately compartmented by fine membranes, is (1) hydrostatically related to somewhat remote hydrodynamic forces in the carotid arterial system, the intracranial venous-sinus system, and the CSF pressure gradients in the subarachnoid space. It is (2) aerodynamically related to air forces which are in direct contact with the round window membrane and the oval window stapedial footplate ligament. The aerodynamic routes are either nasal-nasopharyngeal via eustachian tube or tympanic via external auditory canal.

BASIC MECHANISMS

Hydrodynamic (CSF) or aerodynamic (tubotympanic) routes can be responsible for membrane breaks and they can co-exist in some cases. I reported these in my 1970 Triological Presidential Address (Goodhill, 1971) (Figure 1). Surgically visible perilymph fistulae as seen in the middle ear via oval window and/or round window membrane breaks are accompanied in many cases by surgically invisible intralabyrinthine mixed perilymph-endolymph fistulae due to internal membrane lesions. The latter can occur alone.

Hydrodynamic explosive mechanisms

The *cochlear aqueduct route* to scala tympani can cause breaks in round window membrane, and/or internal breaks in Reissner's basilar, saccular, utricular, and/or semicircular canal membranes (Figure 2).

The *internal auditory canal route* to scala vestibuli can cause breaks in oval window ligament and/or similar internal labyrinthine membrane breaks. Either or both of the above routes can cause explosive fistulae resulting in hearing loss, vertigo and tinnitus. Membrane deformations with or without fistulae can also occur.

Aerodynamic implosive mechanisms (Figures 3 and 4)

Aerodynamic pressure surges via the tubotympanic system or the external audi-

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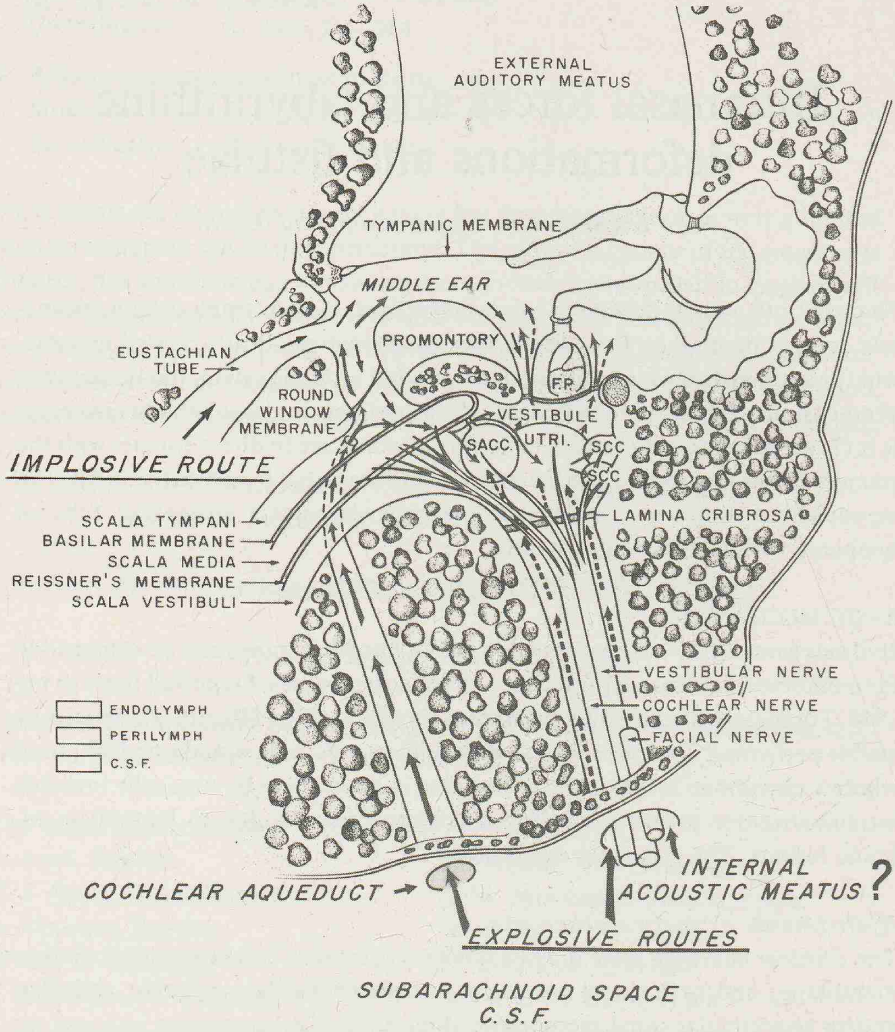

Figure 1. Possible pathways of implosive and explosive forces sketched in inner ear. (Laryngoscope 81: 1462–1474, 1971).

tory canal communicate with round window membrane and oval window footplate ligament areas as a route for implosive fistulae. Self-induced forceful eustachian tube inflation forces or tubotympanic environmental valsalva forces in flying and diving can cause implosive round window and/or oval window fistulae, with chain reaction internal labyrinth membrane sequelae (Figure 5) producing intralabyrinthine membrane deformations with or without fistulae with resulting hearing loss, vertigo and tinnitus.

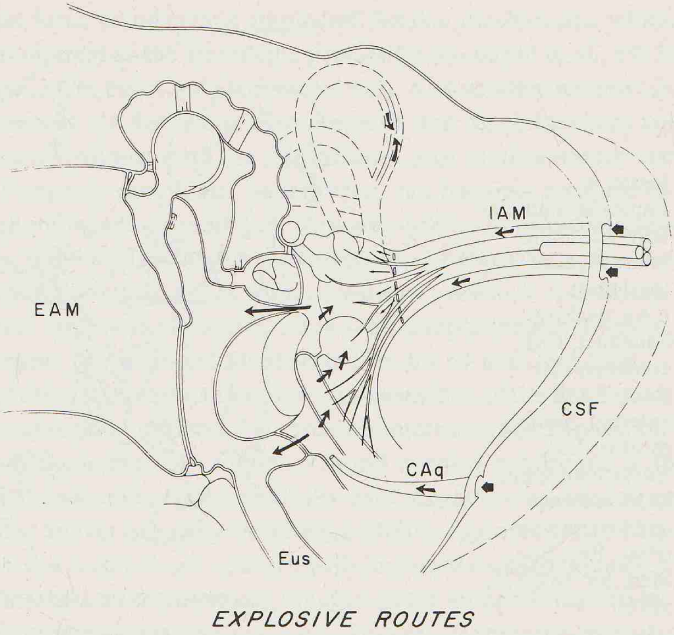

Figure 2. Explosive route for labyrinth membrane ruptures (in detail). CAq, cochlear aqueduct; CSF, cerebrospinal fluid; EAM, external auditory meatus; Eus, Eustachian tube; IAM, internal auditory meatus. (Proc. Roy. Soc. Med. 69: 565–572, 1976).

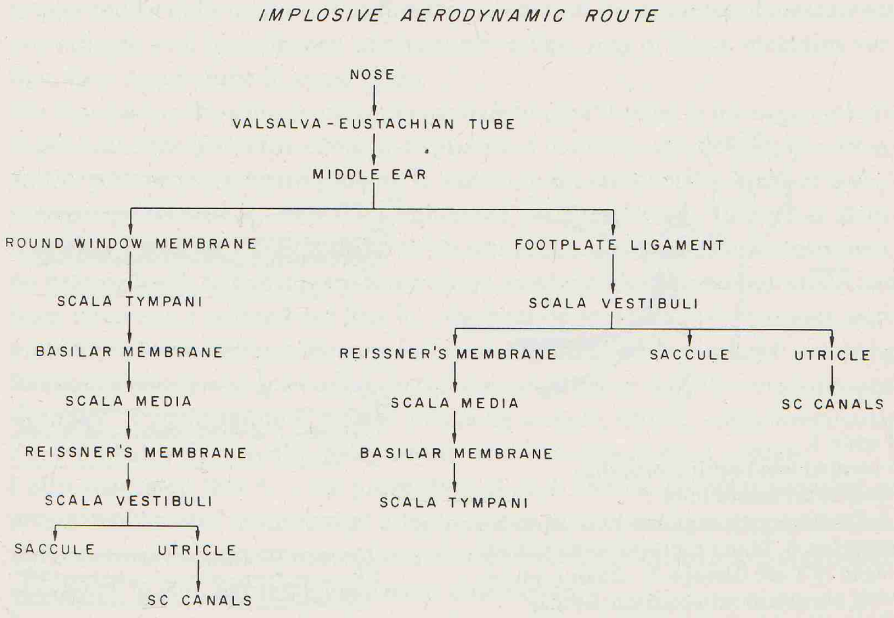

Figure 3. Potential implosive routes for labyrinth membrane ruptures.

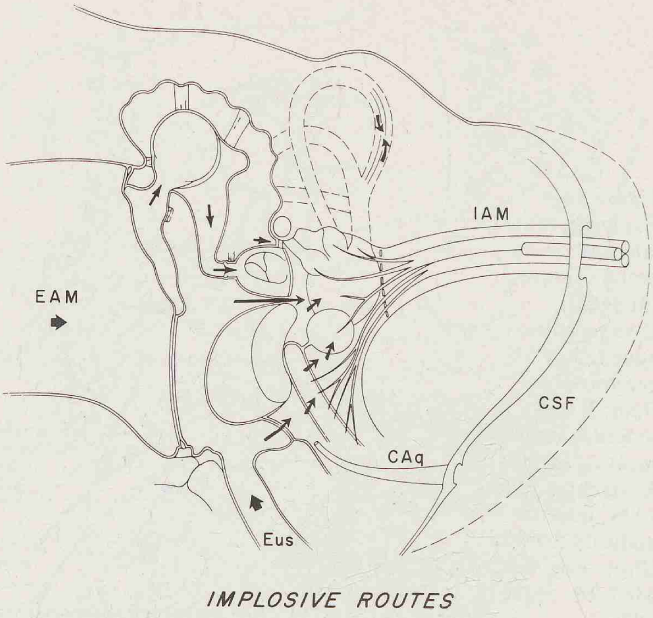

Figure 4.
Implosive routes for labyrinth membrane ruptures (in detail).
CAq, cochlear aqueduct; CSF, cerebrospinal fluid; EAM, external auditory meatus; Eus, Eustachian tube; IAM, internal auditory meatus.
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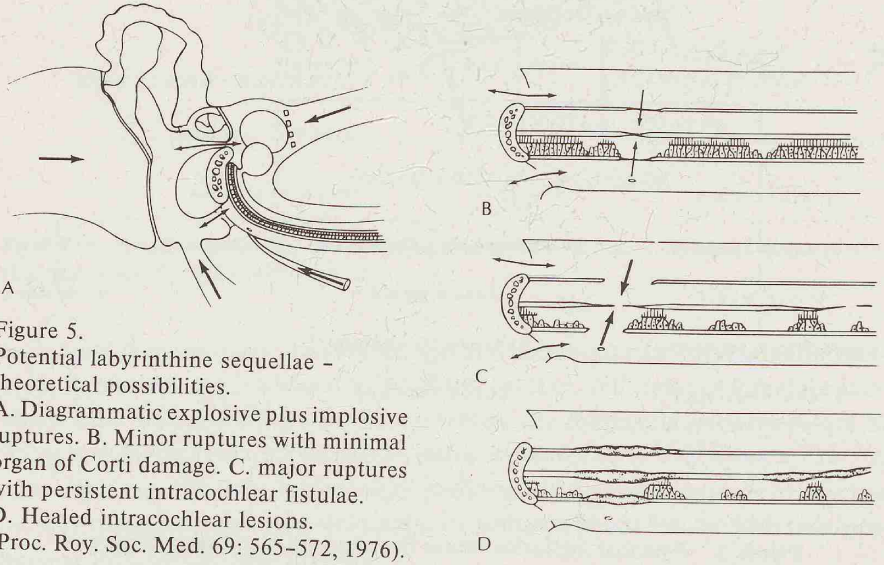

Figure 5.
Potential labyrinthine sequellae – theoretical possibilities.
A. Diagrammatic explosive plus implosive ruptures. B. Minor ruptures with minimal organ of Corti damage. C. major ruptures with persistent intracochlear fistulae.
D. Healed intracochlear lesions.
(Proc. Roy. Soc. Med. 69: 565–572, 1976).

It is these intranasal force aerodynamic implosive fistulae mechanisms which should be of special interest to the rhinologic profession (Goodhill et al., 1973; Goodhill, 1976; Palva, 1970; Palva and Dammert, 1969). Also sudden increases in external ear air pressures via the tympanic membrane and ossicular chain are transmissible to the oval window, with the possibility of oval window membrane break and fistula. Tympanic membrane aerial forces can transmit pressure increases also to the round window membrane with possibilities of round window membrane ruptures, if the eustachian tube is closed. Such external ear pressure trauma", (Freeman and Edmonds, 1972), which described persistent sensorineu-
When eustachian tube air flow is blocked by impaired rhinopharyngeal function, middle ear air pressures can be forcefully directed to one or both windows.
The primary issue of this presentation, therefore, is recognition of the force transmissibility potential of sudden intranasal air pressure increases due to nasal obstruction with aerodynamic pressure surges to round window membrane or to oval window ligament producing either surgically demonstrable oval window or round window fistulae and/or intralabyrinthine membrane ruptures or deformations. A secondary issue is rhinologic concern with impaired eustachian orifice-tube ventilation factors related to poor nasal function in the vulnerability of labyrinthine windows to trauma via external ear canal-tympanic membrane-ossicular chain to oval window and intratympanic air pressure sudden force trauma to round window membrane. Sequelae in the inner ear are due to intralabyrinthine breaks and/or deformations. The functional labyrinthine sequelae of these forces are variable with hearing loss, tinnitus and vertigo. Any of these, including vertigo, may occur alone in some cases.
For many years the concept of "inner ear barotrauma" linked to nitrogen embolization was considered the etiologic explanation in divers who developed vertigo and/or sensorineural hearing losses. In a letter to the editor of the *Archives of Otolaryngology* (Goodhill, 1972), I commented on a paper on "Inner Ear Barotrauma", (Freeman and Edmonds, 1972),which described persistent sensorineural hearing losses in five trained Navy divers, in whom nitrogen embolization had been ruled out. I pointed out that ". . . the hearing losses in these patients were probably due to intracochlear membrane ruptures. Such ruptures involving Reissner's membrane and basilar membrane could occur via either oval or round windows. A chain reaction rupture involving saccule, utricle, and semi-circular canal probably occurred in those divers who also experienced vertigo."
I also suggested that ". . . the prevention of such serious cochlear sequelae in divers may involve more critical attention to nasal and nasopharyngeal factors, not only from the point of view of selection of divers, but also in terms of pre-dive check ups to rule out temporary nasal obstruction."
In a paper on ruptures of the round window membrane in inner ear barotrauma, Freeman, Tonkin and Edmonds (1974) from the School of Underwater Medicine,

Royal Australian Navy, reported two cases in experienced professional divers. Both had difficulty in auto-inflation while diving, one suffered from increasing deafness in both ears over several days with mild vertigo and the other had pronounced vertigo without any cochlear symptoms or signs. The first diver had been aware of increasing nasal congestion due to a gross septal deformity narrowing the right side of the nose. The second diver suffered from recurrent nasal congestion due to allergic rhinitis.

The authors concluded that normal nasal function and ability to auto-inflate were crucial factors in diving. Attention is given to normal nasal function in the field of aviation medicine, where the maximum pressure gradient is 1 atmosphere. They correctly commented on the fact that even greater emphasis should have been made on the necessity for normal nasal function when diving, and they strongly recommended that all divers should have normal nasal function.

King (1979) pointed out the crucial relationship between nasal-eustachian tube function and potentials for labyrinthine membrane ruptures, and stated that ". . . as long ago as 1933 Hughson and Crose showed that the round window membrane bulged into the tympanic cavity during a rise in pressure of the cerebrospinal fluid. It was not until 1971 that Goodhill drew attention to spontaneous rupture of the round window membrane, following a rise in intracranial pressure associated with coughing, sneezing or straining."

Other reports relating to fistulae were published by Fraser (1975), Morrison (1975), Tingley and MacDougall (1977) and Farmer (1977) which suggested that such lesions are more common in divers than aviators. As King commented "This is not surprising when one remembers that the ambient environmental pressure at a depth of 17 feet below sea level is 1160 mmHg, and with the tube blocked from the surface downwards there will be a pressure differential of 400 mmHg on the tympanic and window membranes."

Sasaki et al. (1975) reported the case of a physician diver whose left oval window ligament and round window membrane ruptured on a 12 ft. dive. They concluded that concomitant nasal disease and impaired eustachian function was a common factor in the pathogenesis of the disease. They stressed the importance of avoiding forceful auto-inflation under hyperbaric conditions. In addition, the significance of normal nasal function with regard to ease of auto-inflation in divers is emphasized in prevention of inner ear barotrauma.

Forced tubal clearing in flyers, voluntarily produced by violent closed nasal valsalva inflation can produce perilymph fistula. At the University of Birmingham Fifth Academic Congress in England I reported the case of a professional pilot with a long history of nasal allergy who attempted to clear a blocked right ear during flight (Goodhill, 1980). He forcibly attempted closed nasal valsalva inflation and noted sudden right tinnitus, hearing loss and dizziness. Surgical explora-

tion eight weeks later showed partial incudostapedial joint dislocation and oval window fistula, both of which were repaired. The vertigo disappeared but there was only moderate gain in hearing.

CONCLUSION

We are now at a point in the expanded conception of relationships between the aerodynamic nasal route and labyrinthine implosive sequellae where there is need for more research in problems relating labyrinthine membranous integrity to the sequellae of intranasal forces. It is at this point in time that a closer correlation between research methodologies of rhinologists and otologists becomes necessary.

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Victor Goodhill, M.D., F.A.C.S.
Adjunct Professor of Surgery/Head and Neck
UCLA Medical School
Los Angeles, California 90024, U.S.A.