Otology seminar
Diving-related otologic disorders

I. Introduction

1. underwater environment: rapidly changing ambient pressures
2. SCUBA (self-contained underwater breathing apparatus)
3. at sea level: $100\text{kPa} = 1 \text{ atm.} = 760\text{mmHg}$
   every additional 10m (33ft) of descent adds 100kPa (1atm.)
4. Boyle’s law: the volume of gas inversely with the pressure applied
   Henry’s law: the amount of a given gas that is dissolved in a liquid is directly proportional to the partial pressure of that gas

II. Barotraumas: during descent from a drive

1. tissue injury resulting from the failure of a gas-filled body space to equalize its internal pressure to correspond to changes in ambient pressure
2. the risk of barotraumas is more pronounced near the surface of the water
3. barotraumas of the EAC: any obstructions that prevent water replacing the air in EAC (cerumen, stenosis, atresia, exostoses, foreign bodies) will result in petechial hemorrhages and/or blebs of the EAC/TM similar to bullous myringitis
4. the most common disorder in divers: barotraumas of the middle ear
5. symptoms: vary from a sensation of pressure, followed by pain and conductive hearing loss, to rupture of the tympanic membrane, usually with an acute relief of pain, followed by whirling vertigo due to uneven caloric stimulation.
6. gradings: grade 0 (symptoms only) to grade 5 (TM perforation) (Edmonds, 1981)
7. mechanisms: an inability to equalize the pressure in the middle ear because of faulty clearing techniques, upper respiratory infection, or anatomical variations in the nasal skeleton (figures)
8. managements: topical nasal and systemic decongestants. If purulent otorrhea (+), give antibiotics. Most TM perforations heal spontaneously. (R/O IEB)

9. **Inner-ear barotraumas** (IEB): less frequent, 0.5% of divers. Due to a consequence of forceful efforts to equalize middle-ear pressure when the E-tube is locked and bloked. (elevated ICP, cochlear aqueduct, basement membrane rupture or fistula)
   a. cochleovestibular (CV) symptoms
   b. definition: an injury to the IE due to diving that cause SNHL, with or without coexistent CV symptoms
   c. the Bay Memorial Medical Center, Panama City, FL (Parell GJ, 1993)
      twenty patients, all male, 21~50 years (35 years), follow-up: 62 months
d. otologic history, statement of difficulty equalizing the ears (swallowing, jaw thrust, or yawning maneuver) during the dive, documentation of the dive profile, physical examination, and serial audiogram; ENG
e. results: three types as intracochlear hemorrhage, PLF, or ICMT (tear)
  deep diving is not a factor in barotraumas (most were within 9m, i.e. 30ft)
f. during the follow-up period, no diver experienced a further deterioration of hearing, recurrence of vestibular symptoms, or deterioration of tinnitus
g. conclusion: recommending no further diving after IEB may be unnecessarily restrictive
10. managements of IEB: complete bed rest (head elevated), vestibular tranquilizers, decongestants, eventually surgical repair of the PLF
11. alternobaric vertigo: an asymmetric increase in pressure in the right and left middle ears that exceeds a threshold difference of 45mmHg (N/V, disorientation)
12. facial baroparesis: a dehiscent intratympanic segment of the facial nerve compressed because of overpressure (transient ischemic neurapraxia)

III. Decompression sickness: during ascent from a drive, 0.0034% of divers
1. breathes air under increased pressure, tissue loaded with increased quantities of oxygen and nitrogen (Henry’s law)
2. When the ambient pressure decreased as the diver returns to the surface, a state of supersaturation is created that may lead to the liberation of free gas.
3. avoided by decompression table (control ascent rate with stops)
4. risk factors: advanced age, female sex, obesity, elevated PaCO\(_2\), low water temperature, poor physical fitness and activity during the dive, repeated dives
5. classifications: Type I (mild insult), Type II (more severe variety, CNS)
6. Inner-ear decompression sickness (IEDCS): occurs mainly in deep prof. diving
7. pathophysiology: the formation and growth of inert gas bubbles within microvessels (venous circulation of the stria vascularis, spiral ligament, and SCC) and otic fluids (cochlea hemorrhage, fibro-osseous labyrinthitis in the SCC).
8. symptoms: severe SNHL, tinnitus, and vertigo
9. managements: administration of inhaled oxygen at the highest possible conc. and proper hydration; transported to a recompression chamber (figures)
10. the Israel Naval Medical Institute, from 1988 to 2003 (Shupak A, 2003)
  11 out of 24 (46%) of the patients with IEDCS, regularly re-examined all men, 24~69 years (mean: 40 years); bed-side testing, PTA, ENG, etc.
11. the long-term follow-up of divers with IEDCS had demonstrated residual CV deficits in more than 90% (10/11) of the patients, despite maximal recompression therapy. IEDCS carries a high risk for residual inner ear damage than IEB.
IV. Differentiation from IEB and IEDCS

Much has been written regarding the differential diagnosis between IEDCS and IEB, but the most important factors include:

1. In IEB, the patient will report having had difficulty clearing his ears during pressure changes. In contrast, in IEDCS there will have been no such problems.
2. Symptoms of IEB appear during the dive, whereas those of IEDCS appear after the dive.
3. Other forms of DCS may accompany IEDCS.
4. Signs of middle ear barotrauma will often accompany IEB. No such signs will accompany IEDCS.
5. In contrast to the improvement observed in IEDCS during hyperbaric treatment, IEB will be aggravated by treatment in the hyperbaric chamber.

References:
