Facial Nerve Palsy Aboard a Commercial Aircraft

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CASE REPORT

Facial nerve palsy resulting from barotrauma is a phenomenon that is frequently seen in divers, but is under-reported in aviators or passengers aboard commercial aircraft. This case describes a 24-yr-old healthy aviator who experienced an episode of facial nerve palsy during ascent while traveling as a passenger aboard a commercial flight. The probable pathogenesis of this phenomenon in this case is described.

Keywords: facial nerve palsy, aviators, aerospace medicine, divers.

DISCUSSION

The volume of air in the middle ear varies inversely with pressure according to Boyle's law. During diving, one additional atmosphere is experienced every 10 m. This pressure change is much less dramatic in the atmosphere, where 5500 m elevation represents a reduction of one-half atmosphere. Aural barotrauma is caused by the inability to equalize middle ear and ambient pressures. During ascent, expanding middle ear air vents through the Eustachian tube. The pressure difference required to force air out of the Eustachian tube during ascent is approximately 52 cm H2O (7). If the middle ears vent unequally and a pressure gradient of greater than 60 cm H2O exists between the two ears, than increased labyrinthine discharge may result in irritative nystagmus and vertigo (3).

The cabin altitude in a commercial aircraft cruising at 35,000 ft (the usual flight altitude) is approximately 8000 ft. If the flight starts at sea level, then the theoretical maximum pressure difference between the middle ear and the cabin atmosphere at cruising level would be 266 cm H2O, which is much higher than the mean capillary BP (4). Under normal conditions, equilibrium would be achieved by gas escaping through the Eustachian tube to the nasopharynx because of the large pressure gradient, but any delay in this equilibration may lead to elevated pressure within the middle ear.

Facial nerve palsy resulting from barotrauma is termed facial baroparesis. The facial nerve is most vulnerable during its course along the medial wall of the...
middle ear, where only the thin bony covering of the Fallopian canal protects it (9). Reduced conductivity and neuropaxia as a result of extremely elevated hydrostatic pressure in the middle ear were demonstrated in isolated nerve preparations (2), yet the probable mechanism for facial baroparesis is ischemic neuropaxia. In studies carried out in guinea pigs, increased middle ear pressure resulted in decreased blood flow to the middle ear but not to the inner ear (6). Blood flow to the facial nerve also decreases if middle ear pressure is transmitted through a dehiscent facial nerve canal, this being present in 55% of the population (10).

Facial nerve injury in divers or aviators may occur by an additional mechanism. Farmer proposed that gas bubbles might enter a non-dehiscent fallopian canal through the fenestrum of the chorda tympani nerve and result in decompression sickness (1). This mechanism is unlikely in our case as the flight profile did not result in significant pressure changes, but it should be kept in mind particularly in divers or in aviators exposed to flight conditions which predispose them to decompression sickness, such as those flying following diving.

Facial baroparesis is certainly under-reported because of its transient nature, yet most cases described in the literature occurred in a single episode, in spite of recurrent exposure to similar flight or diving conditions. This led to the hypothesis that certain conditions must coexist to precipitate the palsy. These include transient Eustachian tube dysfunction, hypotension, or subclinical infection with one of the neurotropic viruses (5). In our case, no apparent precipitating cause for the facial baroparesis was revealed by the history taking or the physical examination, yet the fact that the aviator flew hundreds of hours in similar flight conditions, with this episode occurring only once, supports the hypothesis that a precipitating factor was present. We believe this factor to be a subclinical infection with one of the neurotropic viruses.

Most cases of facial paralysis associated with flight resolve spontaneously. The occurrence of this phenomenon in our pilot after exposure to hundreds of hours in similar conditions and his occupation, in which recurrence may be particularly hazardous, prompted us to perform an MRI examination, which was found to be normal. Most cases of facial baroparesis do not need to undergo imaging studies and patients should be reassured regarding the benign nature of this condition. Brain and auditory canal imaging should be considered only in limited cases. If recurrent, the facial nerve palsy can be prevented by adequate self-inflation and, in selected cases, by ventilation tube insertion.

CONCLUSIONS

Facial baroparesis is a benign process that occurs in divers or aviators during ascent. It usually occurs in the presence of co-existing conditions that contribute to increased middle ear pressure. The occurrence of transient facial nerve palsy during ascent is the typical presentation. It is important to be aware of this phenomenon in order to avoid unnecessary work-up and treatment as is required in decompression sickness or suspected air embolism. Reassurance of the patient is crucial, and in recurrent cases, ventilation tube insertion should be considered.

REFERENCES