Clinical Reports

Facial nerve palsy associated with underwater barotrauma

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Summary: This report describes a case of facial nerve palsy following barotitis media sustained at shallow depth. The neuropraxia is likely to have been due to the direct effect of pressure, facilitated by a congenital hiatus in the bony canal protecting the facial nerve in the middle ear.

Introduction

Underwater diving is an increasingly popular activity for industrial, military, and recreational purposes, but it has inherent dangers. Middle ear injury due to barotrauma is caused by inability to equalize the pressures across the tympanic membrane during descent underwater. It is the rate of pressure change which creates the problem, and this is greatest at shallow depth: by 10 metres, the pressure is already twice that at sea level. The ability to increase middle ear pressure by the Valsalva manoeuvre is essential, and failure is usually due to acute or chronic infections of the nose, or a severely deviated nasal septum.

Facial nerve palsy is a dramatic complication of otitic barotrauma sustained at shallow depth, and highlights the degree of risk.

Case report

A fit 22 year old soldier was forced to abandon a sea dive at a depth of only 5 metres due to increasing pain in his ears, despite repeated Valsalva manoeuvres. Soon afterwards, he noticed that his hearing had deteriorated, and the diagnosis of otitic barotrauma was confirmed by the observation of congested tympanic membranes; there was no perforation present.

Five days later, he noticed slight left-sided facial weakness, which gradually worsened until the lower motor neurone facial nerve palsy became complete. Lacrimation was normal (Schirmer's test) but there was subjective loss of taste sensation on the left side of his tongue, anteriorly. This localized the facial nerve lesion to its middle ear section, between the geniculate ganglion and the junction of the chorda tympani.

He was treated with a course of dexamethasone and antibiotics. His conductive hearing loss returned to normal within 2 weeks, but the facial palsy took 6 weeks to recover completely.

Discussion

All parts of the ear may be damaged by underwater barotrauma. Mild injury may produce congestion of the tympanic membrane from vascular engorgement. The membrane may rupture at only 3–5 metres' depth without clearance, or at even shallower depth if the membrane is thin or scarred.¹ Relatively negative pressure in the middle ear promotes formation of a fluid transudate there, causing conductive deafness. Round window rupture causes a perilymph leak and inner ear damage, promoting sensorineural deafness and vertigo; it may result from high external pressure or from too powerful attempts at Valsalva.²

The facial nerve is most vulnerable in its course along the medial wall of the middle ear,³ where it is protected only by the thin bony covering of the Fallopian canal. In less than 10% of the population there is a congenital absence of part of this bony covering; in others there is probably a physiological hiatus, if not an anatomical one.⁴

Facial nerve palsy is a recognized, but uncommon, complication of suppurative otitis media, but it has not been reported previously in association with barotrauma. As this palsy recovered completely, it is likely that the direct effect of pressure produced oedema in the nerve sheath and a neuropraxia, in addition to the fluid transudate in the middle ear. Treatment with oral steroids is aimed at limiting the inflammatory oedema, and speeding recovery of nerve function.

Diving underwater can be a hazardous activity even at shallow depth, and divers must be well

trained. All doctors must be alert to ensure that potential divers are generally fit, have a normal upper respiratory tract and, in particular, are able to Valsalva.

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