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Facial baroparesis secondary to middle-ear over-pressure: a rare complication of scuba diving

A. F. HYAMS*, S. C. TOYNTON, F.R.C.S., M. JARAMILLO, L. R. STONE*, P. J. BRYSON†

Abstract
A facial nerve palsy, as a result of middle-ear high pressure, is a rare complication of sub-aqua diving. It may occur as a result of an acute pressure change in the middle ear during ascent in those patients who have experienced difficulty equalizing their middle-ear pressure during the prior descent. We present the case history of this occurring in a 21-year-old diver and discuss the pathophysiology, management and the previous literature. The correct diagnosis of this condition is important if unnecessary, and potentially hazardous, recompression treatment is to be avoided.

Key words: Facial Paralysis; Barotrauma; Ear Middle; Diving

Introduction

Sub-aqua (Scuba) diving is becoming increasingly popular for both occupational and recreational purposes. A rapid pressure change in air-filled organs makes them liable to problems during descent or ascent.

Ear related injuries are common and are grouped under the term 'otitic barotrauma'.[1] This occurs as a result of eustachian (pharyngo-tympanic) tube dysfunction resulting in the inability to equalize the pressure differential across the tympanic membrane.

The rare condition of a transient facial nerve palsy associated with ipsilateral middle-ear over-pressure during ascent has been described in both divers and aviators.[2,3] It may be associated with a vertiginous episode due to asymmetrical middle-ear pressures also occuring during ascent (alternobaric vertigo).[4] Excessive pressure exerted on the facial nerve through a dehiscence of the horizontal portion of the fallopian canal is believed to compress the vasa nervorum. This may cause relative hypoxia and subsequent temporary neuropraxia.[1,3,5-10]

We report a case of this condition that occurred in a healthy young sports diver, and discuss the possible mechanisms of its cause, and its presentation, differential diagnosis and management.

Case report

A 21-year-old female sports diver performed two successive sea water dives, each of 17 minutes duration. The maximum depth was 13 m, with an interval of nine minutes between dives. British Sub Aqua Club – 1988 decompression tables were followed,[11] and no decompression stops were required. There were no rapid ascents during either dive.

The first dive was uneventful. During descent on the second dive there was difficulty equalizing the left middle ear. Several forced Valsalva manoeuvres were performed, which relieved the otalgia and allowed further descent. No further symptoms were experienced to a depth of 11.9 m. There was severe left-sided otalgia on ascent to 0.2 m, which was relieved by descending back to a depth of 5 m. This depth was maintained for three minutes, followed by a slow re-ascent to the surface (Figure 1). On surfacing she experienced left ear fullness and pain with hearing loss and 10 minutes later she noticed a twitch in her left upper lip. This was soon followed by paralysis of the whole of the left side of her face and forehead. She also noticed an altered sensation on the left side of her tongue. Following a boat journey of 15 minutes back to the shore, her face was noted to have some increase in tone. Once on shore 100 per cent oxygen was administered as a first aid procedure. There was a rapid resolution of all her symptoms accompanied by the return of normal facial movements.

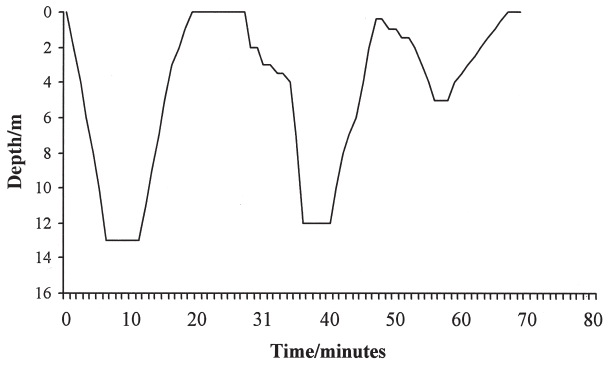

FIG. 1
Dive profile.

From the Department of Otolaryngology – Head and Neck Surgery, Derriford Hospital, Plymouth, the University of Bristol*, School of Medical Sciences, University Walk, Bristol, and the Diving Diseases Research Centre†, Tamar Science Park, Davy Road, Plymouth, UK.
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On arrival at the nearest diving medical centre a diagnosis of decompression illness (DCI) was made. Examination of the left tympanic membrane was reported to reveal marked congestion and oedema with bulging. Despite having no symptoms she received precautionary recompression in a hyperbaric chamber for four hours 40 minutes, following a US Navy treatment table.[12] The patient remained asymptomatic.

No further facial weakness has been noted after a follow-up period of over two years.

- **Facial nerve palsy is a rare complication of sub aqua diving**
- **It may occur as a result of an acute pressure change in the middle ear during ascent**
- **The correct diagnosis is important if unnecessary, and potentially hazardous, recompression treatment is to be avoided**

Discussion

Whilst possible, decompression illness following dives with profiles such as those undertaken by this patient is very unlikely. Both dives were short and shallow with controlled ascents. The facial nerve palsy was associated with difficulty equalizing the left middle-ear pressure, there being subsequent otoscopic evidence of middle-ear barotrauma. This course of events has been described in previous, sporadic case-reports of facial baroparesis (FB)(Table I).

When diving underwater there is an increase in ambient pressure on descent of 1 atmosphere (101.3 kPascals) for every 10 m depth. If there were no eustachian tube function, then the pressure differential across the tympanic membrane would double in the first 10 m of descent, the pressure in the middle ear being relatively negative. Active opening of the eustachian tube allows air into the middle ear thereby equalizing the trans-tympanic pressures.[13] Without eustachian tube opening, descent to a depth of only 0.8 m will cause a pressure differential of 8 kPa (60mmHg), which is sufficient to cause mucosal congestion and oedema. With further descent to a depth of 1.2 m a 12 kPa (90mmHg) pressure differential is achieved. It is then usually impossible to open the eustachian tube voluntarily ('eustachian tube locking') and intense ear pressure and pain are experienced.

As the diver ascends the pressure change is reversed and the middle-ear pressure becomes positive relative to the ambient pressure. The gas contained in the middle ear expands and is normally passively vented through the eustachian tube. This process may, however, be hindered by any mucosal oedema and haemorrhage in the middle-ear cleft which may have occurred during descent as a result of eustachian tube dysfunction. With yet further ascent there will be an increasing pressure differential. This causes the tympanic membrane to bulge outwards causing pain, hearing loss and even rupture. Inner ear barotrauma is rarely associated.

The most widely accepted explanation for a transient unilateral facial palsy occurring during, or soon after, ascent is that of a pressure-induced neuropraxia. Dehiscence of the facial nerve in its intra-mastoid portion occurs in 0.5 per cent to 57 per cent of temporal bones, a lower incidence being found intra-operatively, as compared to histological studies.[14] Experiments in a guinea pig animal model showed that blood flow in the vasa nervora to the facial nerve decreases if increased middle-ear pressure is transmitted through a dehiscence of the facial canal.[10] A neuropraxia has also been induced by applying increasing pressures to an exposed part of the facial nerve in cats.[15] An alternative hypothesis has been postulated that in a non-dehiscent facial canal, pressure may be transmitted through the fenestra of the chorda tympani.[14,16] Either of these mechanisms could also explain the transient nature of the condition as the blood flow would rapidly return to normal when the middle-ear pressure becomes less than the capillary closing pressure.

The incidence of FB is difficult to determine, the transient nature of the condition makes it likely that it is under-reported.[3,6-9] It is important to rule out other causes of facial palsy and a meticulous description of the type, and profile, of the dive is essential. The course of the symptoms should be carefully documented, with particular attention given to the time and rapidity of onset. Clinical evaluation should include complete general neurological, and neuro-otological examinations.

TABLE I

DOCUMENTED CASES OF BAROTRAUMATIC FACIAL PALSY IN THE MEDICAL LITERATURE

Reference	Number of cases	Modality	Outcome
Anonymous report[23]	1	Diving	Resolved
Bannery[19]	3	Diving	Resolved
Becker[6]	3	Diving	1 Resolved, 2 Unknown
Eidsvik and Molvaer[7]	5	Diving	Resolved
Fortes-Rêgo[20]	1	Diving	Unresolved
Hudson and Tscharke[8]	1	Diving	Unresolved
McCleve[22]	1	Diving	Unknown
Molvear[5]	2	Diving	1 Resolved, 1 Unknown
Renon *et al.*[24]	1	Diving	Resolved
Shepherd *et al.*[17]	1	Diving	Resolved
Vincey and Renon[18]	2	Diving	Resolved
Whelan[1]	1	Diving	Resolved
This report (2004)	1	Diving	Resolved
Total no. of divers reported	23	Diving	17 Resolved, 2 Unresolved, 4 Unknown
Bennett and Liske[2]	2	Flying	Resolved
Anonymous report[21]	1	Flying	Resolved
Motamed *et al.*[9]	1	Flying	Resolved
Total reported	27	Diving 23, Flying 4	21 Resolved, 2 Unresolved, Unknown

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DCI is more common following deep dives, especially when the diver has ascended rapidly. A facial palsy due to a DCI would be exceptionally unlikely to occur in isolation without any other symptoms or signs. Conversely a patient with an FB would not be expected to have symptoms and signs of a DCI. The FB patients would be likely to have associated symptoms of otitic barotrauma, which include ipsilateral otalgia, a pressure sensation, and hearing loss. The tympanic membrane may appear infected and haemorrhagic with a middle-ear serous effusion. A haemotympanum or even perforation may occur. FB is typically of rapid onset and resolves after a few minutes. This would be unlikely to be the case if a DCI were the cause. There is a conceivable situation where both pathologies could co-exist if, perhaps due to some emergency, rapid surfacing was required. To our knowledge this combination has not been reported.

The initial emergency treatment of DCI is inspired 100 per cent oxygen with the administering of intravenous or oral fluids. These should always be given if there is initial doubt as to the true diagnosis. It is likely, however, that this treatment did not contribute significantly to the resolution of the facial paresis in this case. If the patient presents with a painful bulging membrane then the treatment of choice is a myringotomy. The patient should be assessed by a clinician competent in the diagnosis of DCI and an early referral to an otolaryngologist is strongly advised. Given the likely aetiology of an FB, if rapid resolution does not occur then steroid treatment should be considered. A persisting palsy should be investigated and treated as for a DCI.

Recompression treatment may worsen symptoms when eustachian tube dysfunction is still present and is clearly not indicated when a diagnosis of isolated FB has been made. If a DCI is also suspected, or if uncertainty as to the true diagnosis persists, then a myringotomy should be performed and a grommet inserted, prior to proceeding with recompression treatment.

Conclusion

Facial baroparesis is a rare cause of a transient facial palsy occurring during, or very soon after, ascent when diving. It is usually of short duration and a full recovery is expected. The palsy usually spontaneously recovers rapidly prior to medical attention being received, however, if it has not, then an urgent myringotomy should be performed. It is important to distinguish the condition from an intracerebral DCI which would require immediate recompression treatment.

Following an episode of FB, divers should be advised to avoid diving until the middle-ear changes have recovered fully and normal eustachian tube function has returned. They should take special care to descend slowly, performing frequent middle-ear equalization manoeuvres. They should not dive if they have upper respiratory infections and dives should be aborted if middle-ear pressure equalization manoeuvres are unusually difficult to perform on descent.

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Address for correspondence:
Mr S. C. Toynton, F.R.C.S. (ORL),
ENT Department,
Level 7,
Derriford Hospital,
Plymouth,
Devon PL6 8DH,
UK.

Fax: 01752 763185
E-mail: Steve.Toynton@phnt.swest.nhs.uk

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