The Journal of Laryngology and Otology
March 1991, Vol. **105**, pp. 217–219

Maxillary sinus barotrauma with fifth cranial nerve involvement

A. W. Murrison, M.B., Ch.B.,* D. J. Smith, M.D., M.Sc.,* T. J. R. Francis, Ph.D.,* R. T. Counter, F.R.C.S., Ed.† (Gosport)

Abstract
A case of neurapraxia of the infraorbital nerve occurring as a result of maxillary sinus barotrauma in a diver is presented. Existing reports of a similar nature are reviewed and the pathogenesis of cranial nerve involvement in barotrauma is discussed. Guidelines for treatment are suggested.

Introduction

The maxillary sinus is at risk of damage from the expansion of gas within it in the event of free flow of gas through the antrum being compromised such as might occur in a diver ascending from depth. The infraorbital nerve, as it courses through the sinus, may, rarely, be damaged as a result.

Case report

A healthy 40-year-old male undertook a recreational dive to a maximum depth of 20 metres. The total dive time was 39 minutes. His ascent was gradual along the contour of a fresh water lake and occupied most of the dive. During the ascent, at a depth of 18 metres, the patient experienced sudden onset of pain in the right upper teeth and mandible which was exacerbated on ascending further. When close to the surface he noticed pain in his upper neck and occiput radiating to the vertex. Upon reaching the surface the diver became aware of right sided numbness of the upper lip, teeth and gums together with numbness of the internal and external surfaces of the right cheek. In all other respects the patient felt well, went home and retired to bed. The lip numbness disappeared 30 minutes after surfacing. Forty-five minutes later he experienced a 'hissing noise' in his head on rolling over in bed and found that the headache and tooth pain were instantaneously relieved. Medical advice was sought 72 hours ater the causative dive by which time much of the numbness had resolved spontaneously. The patient reported that three companions had performed the same dive with him without difficulty either during the dive or subsequently.

The patient had experienced two similar episodes two years previously. On both occasions he had been diving in a swimming pool to a maximum depth of six feet and had noticed numbness as described above, on surfacing which resolved within two or three hours. On the second occasion, he also had a headache and both were abruptly relieved following a 'hissing' sensation in his head.

The patient gave an account of how, 30 years previously, a playground accident had left him with several teeth missing and with damage to his upper jaw as a result of dental impaction. Subsequently, the patient had competed as an amateur boxer and played rugby in the course of which he was rendered unconscious for a few minutes on two occasions.

On examination, the patient was found to be relaxed with normal vital signs. Systemic examination revealed a nasal septum deviated to the right. There was no evidence of recent head injury.

Examination of the cranial nerves was unremarkable with the exception of the fifth (trigeminal) nerve. There was a circular area, 4 cm in diameter, of reduced sensation to pin-prick over the right cheek. Light touch, two point and hot-cold discrimination appeared to be unaffected. A partial denture was worn to replace the missing pre-molar and incisor teeth of the right upper jaw. There was anaesthesia of the right upper canine, remaining pre-molar and first molar with reduced sensation both anteriorly and posteriorly of the corresponding gum and contiguous area of the right cheek. Sensation over the right side of the nasal septum was reduced. The remainder of the neurological examination was normal.

Examination by a Dental Surgeon including orthopantomography failed to reveal structural abnormality of the existing teeth. Sinus X-ray views revealed moderate mucosal congestion of both maxillary and frontal sinuses.

A diagnosis of maxillary sinus barotrauma with neurapraxia of the infraorbital nerve and/or its intra-sinus branches was made. The patient was advised to return home and rest.

Follow-up three days after discharge revealed that there had been a gradual improvement in symptoms with only a slight residual sensory deficit in the affected teeth and contiguous gum remaining. This, in turn, resolved over the next 11 days. In view of the risk of cumulative damage, particularly to dental innervation, the patient was advised to discontinue diving.

Discussion

The volume of gas within the paranasal sinuses decreases with increasing environmental pressure and expands during pressure reduction in accordance with Boyle's Law. Normally equilibration with the external environment is achieved by venting through the sinus openings. If there is an obstruction to the free flow of gas, however, a relative vacuum or state of increased gas pressure occurs within the sinus. Both of these eventualities may result in trauma to the sinus mucosa and submucosa as gas attempts to contract or expand against resistance—sinus barotrauma of descent and ascent respectively. In both cases the predominant symptom is pain over the affected

*Institute of Naval Medicine, Alverstoke, Gosport, Hants. PO12 2DL.
†Royal Naval Hospital Haslar, Gosport, Hants. PO12 2AA.
Accepted for publication: 8 November 1990.

sinus. The condition, introduced as 'aero-sinusitis' in relation to aviators by Campbell (1942, 1944, 1945), is comprehensively described in Fagan *et al's* review of 50 cases of paranasal sinus barotrauma in divers (1976). Analogous situations may occur in other areas of the body, such as in the middle ear, carious teeth and the lungs, where gas is unable to equilibrate barometrically with the environment.

Delicate structures in intimate anatomical relationship to gas-filled spaces which sustain barotrauma are at risk of injury. Seventh nerve palsy following middle ear barotrauma, although uncommon, is well described (Fortes-Rêgo, 1974; Molvaer and Eidsvik, 1987). The facial nerve classically runs in a bony canal separating it from the cavity of the middle ear. In 57 per cent of people, however, as a result of dehiscences in the canal wall, the facial nerve lies unprotected from pressure in the middle ear (Dietzel, 1961). Alternatively, direct exposure to middle ear pressure may be the result of nerve bifurcation and prolapse (Durcan *et al.*, 1967). If the middle ear pressure exceeds that of the venules draining an exposed nerve then venous outflow will become obstructed and an ischaemic neurapraxia will eventually ensue. The process will be rectified by equilibrating the pressure in the middle ear with the environment either by restoring the patency of the Eustachian tube or by spontaneous or prompt surgical tympanostomy (Devriese, 1972). Eventually excess gas in the cavity of the middle ear may be absorbed by the lining mucosa; however this may not be before irreversible damage is caused to the facial nerve.

Whilst the situation with regard to neurapraxia of the infraorbital branch of the fifth cranial nerve as a result of barotrauma to the maxillary sinus, with which the nerve has a close anatomical relationship, is less well described, its mechanism is likely to be similar. Computerized Axial or Roentogenic Tomography might have been used to detect a dehiscence of the infraorbital nerve in the case reported. Negative results would not, however, have excluded a small dehiscence and neither investigation would have materially affected the management of the case.

Damage to the branches of the infraorbital nerve is a well recognized complication of maxillary fractures and surgery. The pattern of the ensuing sensory loss generally allows determination of the branches affected since there is little overlap between the territories that they serve. In the case reported, the facial trauma sustained by the patient as a child may have predisposed him to ischaemic neurapraxia by anatomically exposing the infraorbital nerve. Alternatively, structural damage to the maxillary sinus may have made barotrauma more likely by impeding the free exchange of gas with the atmosphere.

There are no reported cases of infraorbital nerve barotrauma occurring in divers in which the neural damage was likely to have been sustained during descent, although Wright and Boyd (1945) detailed such a case in an aviator. In this isolated case, maxillary sinus pressure would have been negative with respect to the environment, in contrast to the relative sinus over-pressure present in divers developing infraorbital neuropraxia whilst making their ascent (Idicula, 1972; Neuman *et al.*, 1975; Shepherd *et al.*, 1983; Garges, 1985).

Idicula (1972) describes the onset of numbness in the distribution of the infraorbital nerve occurring on ascent from a 680 ft. neon-oxygen dive accompanied by symptoms suggestive of maxillary sinus over-pressure. Concomitant appearance of joint pain suggestive of decompression sickness raises the possibility that the neurological symptoms were the result of decompression sickness rather than ischaemic neurapraxia. A more convincing case was recorded by Neuman *et al.* (1975) in which a diver, on surfacing from a dive so shallow that the diagnosis of decompression sickness was untenable, presented with symptoms and signs suggestive of infraorbital nerve neurapraxia which resolved spontaneously. Garges (1985) described a case of infraorbital nerve neurapraxia occurring during ascent from a chamber dive to 112 ft. In this case, a mucous retention cyst, completely obliterating the right maxillary sinus, had expanded on ascent pressing on the intra-sinus nerves. The case was treated by recompression with resolution of symptoms. Shepherd *et al.* (1983) reported the appearance of symptoms and signs of infraorbital nerve damage in a breath-hold diver recently known to have had bilateral maxillary sinusitis. Again, it was possible to exclude a diagnosis of DCS from the dive history alone.

Diagnoses excluded in the case reported here were:

1. *Non-diving related illness.* The likelihood of the onset, coincidentally, of a non-diving related condition is remote and is made more unlikely in this case by the spontaneous resolution of the condition.
2. *Dental barotrauma.* Air trapped in carious or inadequately filled teeth may expand on ascent or contract on descent causing pain. This may be distinguished from pain referred to the upper teeth as a result of damage, from maxillary sinus barotrauma, to the anterior and middle superior alveolar nerves running through the maxillary sinus by its more discrete nature and by the exacerbation of pain by pressing on the affected tooth.
3. *Cerebral Arterial Gas Embolism (CAGE).* The introduction of gas bubbles into arterial blood as a result of pulmonary barotrauma ('burst lung') is unlikely to be the cause of symptoms and signs referrable to a single, well defined nerve lesion such as the one described here. Additionally, there was no suggestion in this case of an uncontrolled or panic ascent of the sort associated with the development of pulmonary barotrauma and subsequent CAGE.
4. *Decompression sickness (DCS).* Following inadequate decompression, gas bubbles may form in situ in central nervous tissue or may migrate to it by way of the blood stream from elsewhere in the body. However, decompression sickness presenting with discrete peripheral nerve palsies is not well recognized. Additionally, the dive profile in this case, being relatively shallow and short, is unlikely to have provoked DCS.

None of these differential diagnoses would account for the two previous similar, although less severe incidents.

It is important to recognize that nerve lesions occurring in relation to changes in environmental pressure may not necessarily be the result of decompression sickness or cerebral arterial gas embolism. An accurate diagnosis is important as these two conditions require the expeditious administration of hyperbaric oxygen whereas ischaemic neurapraxia, occurring during the course of sinus barotrauma, will resolve spontaneously as demonstrated by the reported cases. Recompression may, in fact, be contraindicated since it may merely result in further sinus barotrauma. If there is any doubt about the diagnosis, recompression should be attempted in view of the potentially dire consequences of failure to institute prompt treatment for DCS or CAGE.

The principles of treatment of neural damage caused by middle ear and maxillary sinus over-pressure are the same. It may be that pressure within the enclosed space has already equilibrated with the environment by the time medical advice is sought. Resolution may be gradual or dramatic as in the case reported here. If, however, there is evidence of continuing neural compression, remedial steps must be taken without delay. Initially, decongestants may be used. If this is unrewarding, it may be helpful to raise ambient pressure to the level of that in the sinus by means of recompression and, subsequently, resurface very gradually. Alternatively, neural ischaemia may be alleviated by placing the patient on 100 per cent oxygen at atmospheric pressure which will promote the resorption of trapped gas and may improve the oxygenation of ischaemic tissue. As a last resort, antrostomy may be considered.

Long-term management should be aimed at treating remediable sinus pathology and outlet obstruction. Intranasal antrostomy may be employed electively to enable those with a history of maxillary sinus barotrauma to return to diving.

Acknowledgement

The authors would like to thank the Medical Director General (Navy), for permission to publish this article.

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Address for correspondence:
Surgeon Lieutenant Commander A. W. Murrison R.N.,
Institute of Naval Medicine,
Alverstoke,
Gosport,
Hants PO12 2DL.

Key words: Barotrauma; Maxillary sinus; Trigeminal nerve