Facial baroparesis: A report of five cases

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Eidsvik S, Molvær OI. Facial baroparesis. A report of five cases. Undersea Biomed Res 1985; 12(4):459–463.—Six episodes of facial baroparesis in 5 divers are reported. Three of the divers experienced left-sided paresis and 2 right-sided. In 2 of the subjects (3 episodes) the paresis disappeared spontaneously when the relative middle ear overpressure cleared, and in the remaining 3 subjects the paresis disappeared on recompression to less than 2 m. No residual effects were found, but the subject who experienced 2 episodes stopped diving. Available evidence favors the theory of ischemic compression neurapraxia.

facial baroparesis
facial palsy
neurapraxia

Alternobaric activities add yet another variant to the variety of causes for peripheral facial palsies already established (1, 2). Only 16 cases of facial baroparesis have been mentioned or described in the available literature (3–13), but we know from personal communication that more cases have been observed. In addition to the only Norwegian case published previously (8), we have collected 5 more cases from Norwegian waters which have previously been reported briefly in congress abstracts (14) and proceedings (15).

CASE REPORTS

1. A 17-yr-old sport diver with 1 yr diving experience performed an open sea scuba dive to 20 m (msw) for 30 min, which is well within accepted limits for decompression stop air diving according to standard dive tables (16, 17). He had experienced acute otitis media several times as a child. Pressure equalization to his left middle ear was usually more sluggish than to his right and he had caught a slight common cold before the described dive. During ascent he felt pressure in his left ear, and approximately 2 min after surfacing the left side of his face became paralyzed. He observed his face in a mirror and discovered that he was unable to close his left eye and could not move any part of the left side of his face. When
he smiled his mouth was pulled to the right. He had no other symptoms of neurological injury. After 15–20 min the pressure in his left ear was gradually relieved and the paralysis simultaneously disappeared. He dived infrequently after this episode, without ill effects. Approximately 0.5 yr after the first incident, he made a scuba dive for 30–45 min in a 4-m deep swimming pool despite a slight common cold. He again experienced pressure in his left ear during ascent and developed a transitory paralysis of the left side of his face with exactly the same extent and course as before. He saw a general physician who found nothing wrong, but was referred to an ENT specialist who found no abnormality on the clinical ENT examination, which included a pure tone audiogram (except for a dip to 25 dB at 6 kHz in the right ear), speech audiometry, tympanometry, and stapedius reflexes. This subject ceased diving due to the described incidents.

2. A 19-yr-old Navy diving student performed a scuba dive to 9-m in the sea (msw) for 63 min, despite a slight common cold. During the course of the dive he surfaced 3–4 times and experienced increasing problems with pressure equalization of his middle ears. During the last ascent he felt a pressure in his right ear increasing to a sharp pain. Three to four minutes after surfacing, he experienced a queer feeling in his face and went to the diving physician’s office. On examination less than 5 min after surfacing, the right side of his face was completely paralyzed. He was unable to close his right eye and the right side of his mouth did not respond when trying to smile. His right ear felt plugged and he experienced a transient sharp pain which disappeared after a couple of minutes. The right tympanic membrane was bulging. Within 10 min he was being slowly recompressed in a pressure chamber and the feeling of pressure in the ear decreased and the paralysis gradually subsided. According to the physician accompanying the diver in the chamber, orbicularis oculi muscle function reappeared first, followed by frontal muscles. Finally, on reaching a depth equivalent to 2 m, orbicularis oris muscle function and the tympanic membrane normalized. During the slow ascent to surface pressure, he used decongestant nose drops. The next day he took a sympathomimetic drug (phenylpropanolamine chloride) orally before diving, which proceeded uneventfully. He had performed approximately 40 uneventful dives before the described dive, and has experienced nothing similar since.

3. A 23-yr-old Navy diving student performed a scuba dive to 5 m depth in the sea (msw). He experienced pain in his right ear during ascent and went to the dive physician’s office where otoscopy revealed a significant bulging of the right tympanic membrane, whereas the left ear was unremarkable. To help relieve the overpressure in the right middle ear the diver received decongestant nose drops and sympathomimetic tablets (phenylpropanolamine chloride). After about 10 min, he developed a right-sided peripheral facial palsy with Bell’s phenomenon. As medication had not helped relieve the pain in the ear, he was recompressed in a pressure chamber about 15–20 mins later. The pain and paresis disappeared immediately upon reaching 1 m depth, and otoscopy performed by an accompanying physician revealed a normal tympanic membrane. As the chamber was decompressed slowly over 5 min, the patient again felt a slight pressure in his right ear and otoscopy revealed a slight bulging of the tympanic membrane, but the paresis did not recur and a neurological examination, including audiogram, was completely normal after reaching surface pressure.
4. A 20-yr-old Navy diver finished an open sea scuba dive to 35 m without ill effects despite a slight common cold. Two to three hours later he made several breath-hold dives in a submarine escape training tank to less than 10 m, due to difficulty in clearing his left ear. After the last dive he felt a persistent pressure in his left ear, followed by a “strange” feeling in the left side of his face and tongue while showering 5 min later. He could not close his left eye and had difficulty controlling the left side of his mouth. The dive physician observed a classical left-sided facial palsy with Bell’s phenomenon, and his left tympanic membrane was seen to bulge outward. The diver continued to move his lower jaw to clear his ear, and approximately 25 min after the onset of facial paralysis the ear suddenly vented, the feeling of pressure in the ear disappeared, and in the course of 30 s the paralysis disappeared while the diver was observed by two physicians. A complete clinical neurological examination was negative, and the tympanic membrane was in the mid position. One week later, while traveling in a commercial aircraft, he again felt pressure in his left ear but no paresis developed and the feeling of pressure disappeared during landing. He has had no recurrence despite continued flying and diving.

5. A 38-yr-old commercial diver with 11 yr of diving experience performed a surface-supplied, open-sea dive to 7.5 m despite a slight common cold and some difficulties with pressure equalization of his middle ears. Immediately upon surfacing, he recognized that something was wrong with his face and went to the dive supervisor and said: “Look! I can’t close my eye or move my face.” The supervisor observed that the diver’s right eye was “big and wet” and the right side of the face was paralyzed, reminding him of people with cerebral stroke. He immediately recompressed the diver in the chamber, where he breathed pure oxygen. At approximately 1.5 m, the paralysis disappeared, but as the supervisor did not know what this was, he gave the patient a complete US Navy Table 6 treatment. The diver emerged symptom-free and has never experienced anything similar since.

All five subjects described were young, healthy males, highly selected with respect to ear physiology, although one had a history of repetitive acute otitus media as a child. That diver experienced a dive-related paresis twice, both times on the left side, and was the only one to stop diving because of the paresis. He was also the only sport diver. One of the three Navy divers was an instructor and the two others were students. The only commercial diver had extensive diving experience.

Four of the incidents occurred in shallow scuba or surface-supplied air diving and 1 developed after repetitive breath-hold diving. Four of the divers admitted that they had dived in spite of having a common cold and hence had difficulties with the pressure equalization of their middle ears. Four of them also reported the feeling of pressure and/or pain in the affected ear during and after ascent. In all cases the paresis developed in less than 10 min after surfacing. In 2 of the subjects the ears cleared spontaneously within 25 min, and simultaneously all symptoms and signs disappeared. The 3 remaining subjects were recompressed within 20 min, and all symptoms and signs cleared before reaching an equivalent depth of 2 m. Three of the cases were left-sided and 2 right-sided. The case reported previously by one of us (8) was also right-sided, so in our experience both sides are equally susceptible. All healed without residual effects.
DISCUSSION

It appears that the described variant of peripheral facial palsy must be pressure-related and thus deserves the designation facial baroparesis, baroparesis facialis (15), or alternobaric facial palsy (8). Parts of the facial nerve in some individuals can be exposed directly to the middle ear pressure through defects in the bony wall of the facial canal (8), and the middle ear pressure can exceed the mean capillary perfusion pressure even in otologically healthy subjects (18). If middle ear pressure in such people exceeds the capillary perfusion pressure of the nerve, then an ischemic compression neurapraxia occurs. The neurapraxia disappears when the pressure is relieved enough to restore the microcirculation of the nerve if the ischemia had not yet inflicted permanent injury. Unfortunately, defects in the facial canal wall have neither been demonstrated nor excluded in cases with facial baroparesis, and middle ear pressure has not been measured during an attack. Although we have no proof that the theory of ischemic compression neurapraxia is correct, available evidence favors that theory, and no alternative theory satisfactorily explains the observed phenomenon.

Although we know, from personal communications, that the described condition is infrequently reported, it is still rare and does not represent a significant problem in diving. However, it can severely affect the professional career of a diver if misdiagnosed as air embolism. If the other obvious differential diagnosis, decompression sickness, is chosen, it can lead to considerable inconvenience.

As soon as a facial baroparesis is suspected the diver should try to perform a Toynbee maneuver (swallow with pinched nose) to release the elevated middle ear pressure. If unsuccessful, one could try decongestant nose drops and/or oral sympathomimetic drugs, even though their ability to help middle ear ventilation is not documented. Under no circumstances must recompression be delayed if the two first therapeutic measures fail.

A diver who has experienced an ischemic compression neurapraxia of the seventh cranial nerve, caused by elevated middle ear pressure during and after ascent, should be advised not to dive whenever there is reason to expect difficulties with the pressure equalization to the middle ears, such as respiratory tract infections or allergy.

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parésie faciale due à la compression
paralysie faciale
neurapraxie
FACIAL BAROPARESIS

REFERENCES
