Facial baroparesis: a review

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Molvær OI, Eidsvik S. Facial baroparesis: a review. Undersea Biomed Res 1987; 14(3):277-295.—If impaired eustachian tube function causes an overpressure to remain in the middle ear after ascent in diving or aviation in a subject with a defect in the wall of the facial canal, an ischemic neurapraxia of the seventh cranial nerve may occur. This type of facial palsy is designated facial baroparesis, baroparesis facialis, or alternobaric facial palsy. If the middle ear pressure is asymmetric the subject may also have alternobaric vertigo. A causative relationship between middle ear overpressure and facial palsy is supported by the palsy’s rapid onset following a reduction in ambient pressure and by its quick disappearance after either an increase in ambient pressure or release of the middle ear overpressure. Transient compression-induced ischemic neurapraxia of the facial nerve is also demonstrated in animal experiments. A similar palsy, ischemic neurapraxia of the fifth cranial nerve due to compression in the maxillary sinus, has been reported in divers. Although it is under-reported, facial baroparesis occurs infrequently, with 23 subjects mentioned in the available literature. Nevertheless, it is important to be aware of its existence, because misdiagnosis as type II DCS or air embolism results in unnecessarily long recompression treatments and pointless delay of resumption of diving. In the worst case, a misdiagnosis might cause a diving license to be revoked.

facial baroparesis
baroparesis facialis
alternobaric facial palsy
facial palsy
ischemic neurapraxia

The changing ambient pressure in aviation and diving has added another variant to the variety of causes of peripheral facial palsy. In our experience this alternobaric variant is not widely known, and can easily be misdiagnosed. This can cause unnecessary inconveniences or may seriously interfere with the patient’s professional career as a diver or aircraft pilot. Although this is a relatively rare type of barotrauma, it is important to draw attention to it and to outline its pathogenesis and treatment. Only then will facial baroparesis be recognized and treated appropriately.

Reduced conductivity and neurapraxia due to extremely elevated hydrostatic pressure have been demonstrated in isolated nerve preparations (1, 2). However, ischemia is the cause of the type of compression neurapraxia termed facial baroparesis. Ischemia
from compression is also thought to be the cause of a similar neurapraxia of the fifth cranial nerve in divers (3–7).

ANATOMIC AND PHYSIOLOGIC CONSIDERATIONS

The seventh cranial nerve

The course of the facial nerve in the fallopian canal through the middle ear is illustrated in Fig. 1. From the geniculate ganglion as far distally as the origin of the stapedius nerve or the chorda tympani, the facial nerve is composed of a single bundle with a well-defined perineurium and a thicker but less dense epineurium that separates the nerve from the periosteum of the canal. This single nerve funiculus occupies from 25 to 50% of the cross-sectional area of the bony canal. At least five interrelated pressure systems in delicate balance with each other regulate the perfusion of the nerve (8). To maintain adequate circulation in the nerve bundle, the pressure gradient across the system must be:

\[ p_A > p_C > p_F > p_V > p_{FC} \]

where \( p_A \) is the pressure in the nutrient arteries in the epineurium, \( p_C \) the capillary pressure inside the funiculus, \( p_F \) the infranuclear pressure, \( p_V \) the pressure of the veins in the epineurium draining the nerve, and \( p_{FC} \) the facial canal pressure (Fig. 2). The margin of safety is narrow, and even a small increase in pressure in the canal can disturb the infranuclear circulation and thus deprive the nerve fibers of adequate nutrition and oxygen.

Fig. 1. The course of the seventh cranial nerve, nervus facialis, in the temporal bone. 1. The facial nerve. 2. Dehiscence in the fallopian canal toward the middle ear cavity.

Even though the facial nerve lies in a bony channel from the geniculate ganglion to the stylomastoid foramen, parts of the nerve may be directly exposed to the pressure in the middle ear through defects or dehiscences in the canal wall along the middle ear cavity or along the air cells in the mastoid process. In the first author's experience with middle ear microsurgery and temporal bone dissection, such dehiscences are not rare. In chronic otitis with cholesteatoma, defects of the fallopian canal wall are common. However, such defects are also regularly detected in normal ears with no history of disease or trauma. Dietzel (9) found dehiscences in 57% of 211 temporal bones, and states that they are developmental in origin. A variant occurring in more than half of the subjects cannot be considered an abnormality. Additionally, gross defects, such as bifurcation and marked prolapse of the nerve, have been observed (10).

Roffman et al. (11) induced neurapraxia in the facial nerve of cats by applying a calibrated pressure to an exposed part of the nerve. The neurapraxia invariably appeared at a pressure between 150–200 mmHg, corresponding to 2–2.75 msw. The animal's blood pressure was then raised by infusing levarterenol i.v., or by coarcting
the abdominal aorta and volume overloading the rostral vascular system. Whenever blood pressure exceeded the calibrated pressure applied to the nerve, neurapraxia disappeared. If the blood pressure was allowed to drop below the pressure on the nerve, neurapraxia returned rapidly. Tzadik et al. (12) used the same technique, but applied pressure to the nerve just short of inducing neurapraxia. The animal’s blood pressure was then reduced by injecting nitroprusside i.v. When blood pressure fell below the pressure applied to the nerve, a neurapraxia promptly developed, and whenever blood pressure could be restored, neurapraxia resolved. These investigations convincingly demonstrate that compression neurapraxia in the physiologic pressure range is caused by ischemia, and that the lesion is readily reversible, provided the ischemia has not lasted long enough to inflict permanent anoxic injury to the nervous tissue. Irreversible injury to the facial nerve of cats occurs if compression ischemia lasts more than about 3.5 h (13), and there is no reason to believe that the human facial nerve should be more resistant.

Within the fallopian canal system nerve fibers are the most protected from increased pressure, while the venules are least protected (8). Thus, a diver with dehiscences in his facial canal ascending with a closed eustachian tube, the increasing middle ear pressure will compress the venules in epineurium and obstruct the venous outflow from the nerve, causing an increased intrafunicular capillary pressure. Due to the tensile strength of the perineurium, this hyperemia will lead to increased intrafunicular pressure and further decrease the intrafunicular circulation (8). A vicious cycle is established in which the hypoxia can reach a level where compression neurapraxia occurs. If middle ear pressure is relieved at this stage by opening the eustachian tube or by myringotomy, or the perfusion pressure is increased sufficiently, circulation of the nerve will quickly be restored and normal nerve function will return rapidly.

However, if middle ear pressure is not relieved, and perfusion pressure is not increased sufficiently, the following chain of events may be envisioned: anoxic injury to the capillary endothelium leads to leakage and edema, first in the epineurium and then inside the funiculus. The perineurium of the funiculus acts as an effective diffusion barrier, causing the proteinaceous fluid to accumulate in the endoneural spaces. The increasing intrafunicular pressure deprives the nerve fibers of nutrition and oxygen and thus interferes with their metabolism. Nerve fibers are deformed, and if this condition continues they become demyelinated and axons are interrupted, causing the nerve fiber to degenerate. Due to obstruction of longitudinal intraneural vessels the lesion may extend both centrally and peripherally. However, even at this stage a delayed, partial motor recovery may occur if adequate perfusion of the nerve is restored.

If the pressure is high enough to compress the intracanicular arteries and thus interfere with arterial supply to the nerve for a sufficiently long time, constrictive endoneural connective tissue will replace the damaged nerve tissue, the internal structure of the bundle will be destroyed, and the nerve converted to a fibrous cord beyond functional recovery.

The eustachian tube

The eustachian tube is named after B. Eustachio, who gave the first detailed description of the tube in 1562 (14). Toynbee discovered that the tube was closed at rest and opened only by contraction of the pharyngeal muscles. Proctor, who has
studied the eustachian tube extensively, maintains that some features of its function are still unknown (15, 16).

Air is absorbed from the middle ear continuously, with O₂ diffusing twice as quickly as N₂. If the eustachian tube is kept closed, middle ear pressure can drop as much as 20 cmH₂O in 4 h. Normally this is avoided by opening the tube with swallows and jaw movements. Adults swallow approximately once a minute when awake and once every 5 min during sleep (17). Therefore, the middle ears are normally ventilated frequently enough to avoid problems during changes in atmospheric pressure due to meteorologic conditions or the faster changes brought about by riding elevators in high buildings, automobiles in mountain areas, light aircraft at low altitude, or cable cars. However, with ascent to or descent from high altitude, problems such as ear pain are more frequent than when lesser altitude changes are involved, and one may be forced to ventilate the ears consciously. Even in commercial passenger aircrafts, where the cabin altitude regularly reaches 8000 ft (2438 m), active pressure equalization maneuvers may be necessary.

During ascent the drop in ambient pressure causes a pressure differential between the middle ear and the epipharynx. When this pressure differential has reached a certain level, the eustachian tube is passively forced open by expansion of the gas in the middle ear, and excess gas is released. The elastic properties of the tissues then close the tube, and it does not open again until further ascent causes the pressure differential to reach the required level. According to Donaldson (14) and McGibbon (18) (quoting Armstrong) the tube opens when there is a drop in ambient pressure of approximately 20 cmH₂O, corresponding to an ascent of 150 m from sea level.

Opening the eustachian tube is facilitated by a surface tension-lowering substance, the eustachian surfactant, as demonstrated in dogs (19) and in rabbits (20). There is no reason to believe that this should not be the case in humans as well.

Tjernström (21) has shown that the tube is passively forced open at middle ear pressures varying from 15–80 (mean 43.9) cmH₂O in otologically healthy subjects. During upper respiratory tract infections, swollen lymphatic tissue and edema of the mucous membrane in the wall of the eustachian tube increase the middle ear pressure required to force the tube open. In such cases the pressure can even become high enough to cause the tympanic membranes to rupture during ascent, as observed by the authors. The pressure differential required to rupture the tympanic membrane is quoted very differently in the literature, ranging from a little more than 100 to 3000 cm of water (22–30). From this we can conclude that during ascent from depth, middle ear pressure in divers can occasionally reach levels far above the capillary blood pressure which averages 43.5 cmH₂O. The capillary blood pressure has a range of 28.6–65.0 cmH₂O (31). It varies directly with arteriolar vasodilatation from emotion, heat, or trauma and with venous pressure, which is affected by hydrostatic pressure or venous obstruction. It varies inversely with arteriolar vasoconstriction caused by emotion or cold.

From investigations into alternobaric vertigo it is known that a relative overpressure in the middle ear also stimulates the vestibular apparatus. If a pressure differential exceeding approximately 66 cmH₂O arises between the two ears, the diver experiences vertigo accompanied by nystagmus. Alternobaric vertigo most frequently occurs during ascent both in diving and aviation and is not rare, especially in experienced divers (21, 32–44). Consequently middle ear pressures in excess of mean capillary perfusion pressure must occur at times during diving.
Pressure effects in the middle ear in diving

The effects of pressure during diving on vessels in tissues adjacent to gas-filled spaces in the body are illustrated in Fig. 3. For simplicity the body is presented as a water filled plastic bag and the middle ear as an air-filled box with rigid walls, connected to the airways through a tube (eustachian tube) with a valve mechanism. The vascular system is shown as a tube-loop with a pump (the heart).

In Fig. 3 A the entire system is in pressure equilibrium at the surface and the capillary blood pressure produced by the “pump” is approximately 45 cmH₂O above ambient pressure.

From Fig. 3 A to B the entire system has been submerged with the valve (eustachian tube) in the open position. The increase in ambient pressure is transmitted to all parts of the system, including the vascular system, thus maintaining the capillary pressure at 45 cmH₂O above the raised ambient pressure. According to Boyle’s law, air was forced into the middle ear through the open eustachian tube to compensate for the relative pressure drop there and balance the increased tissue pressure caused by the

Fig. 3. The mechanism of compression barotrauma (left) and barotrauma of ascent (right). For simplicity, the human body is here illustrated by a water-filled plastic bag (4 in A) containing: 1. Gas-filled bag (lungs/airways). 2. Valve (closing/opening mechanism of eustachian tube). 3. Gas-filled box with rigid walls (middle ear cavity). 4. Fluid-filled tube loop (vascular system). 5. Pump (heart).
ambient hydrostatic pressure. The whole system is again in pressure equilibrium, although at a higher level, and the capillary perfusion is normal.

From Fig. 3 B to C the system descends further, but now the valve is closed. The increasing ambient pressure is again transmitted to all parts of the system, including the vascular system, except to the box with rigid walls (middle ear) since the closed valve does not allow additional air to enter. Consequently, the pressure inside the box will remain at the previous pressure. Because the rise in ambient hydrostatic pressure is added to the intravascular pressure produced by the "pump," the pressure differential across the capillary wall will increase. This intravascular overpressure will cause dilatation and, depending on the distance descended, leakage and rupture. This is the classical mechanism of barotrauma of descent.

As soon as the valve opens to allow complete pressure equilibration in the system, the situation will normalize if no edema or bleeding has occurred. This is shown in Fig. 3 D where the perfusion pressure is again the same as in A and B.

During ascent ambient hydrostatic pressure that has been compressing the entire submerged system will decrease. As a consequence of the drop in ambient pressure, gas in the box will expand according to Boyle's law, the valve will be passively forced open, and excess gas will escape, keeping the pressure equilibrium in the system intact. Thus, in Fig. 3 E the perfusion pressure will be the same as in A, B, and D.

If during further ascent the valve is closed, pressure will be kept constant inside the box while the ambient pressure will drop, as will the pressure everywhere else in the bag, including the vascular system. Thus, when reaching the surface the only intravascular pressure is that produced by the pump, approximately 45 cmH₂O, while the pressure in the box is still equal to the hydrostatic pressure at the depth where the valve closed, i.e., 80 cmH₂O (0.8 msw) in this example. Consequently, the capillaries will be squeezed flat and no circulation will occur, resulting in ischemia. Applied to our diver with a dehiscence in his facial canal, an ischemic compression neurapraxia would occur if the ischemia lasted for a sufficiently long time.

In aviation the whole sequence starts in Fig. 3 D. Thus landing at the original altitude (i.e., returning to the original barometric pressure) should be sufficient treatment. This is consistent with the fact that the symptoms and signs did not persist after landing in any of the 3 reported cases of this kind (45, 46).

DIAGNOSTIC CONSIDERATIONS

When a diver develops a facial palsy shortly after surfacing, one must consider four different etiologic possibilities:
- The paresis or paralysis is not caused by diving and coincidentally occurred in close relation to a dive.
- Cerebral air embolism.
- Decompression sickness (DCS).
- Peripheral ischemic compression neurapraxia.

The first possibility is remote, and the clinical course and possible additional examinations will decide the nature of the palsy.

A cerebral air embolism with a facial palsy as the only symptom is unlikely, especially if the dive history is unremarkable. If the paralysis is peripheral this differential diagnosis can be ruled out. In the case of a central, monolateral facial
palsy, the patient will retain the ability to frown and close the eye on the injured side, because the frontal and orbicularis oculi muscles are innervated from both the right and the left motor nuclei in the pons (4 in Fig. 4). Thus, if the diver is unable to frown and close his eye on the injured side, a cerebral air embolism can be excluded as the cause of the palsy.

Decompression sickness is a great imitator and may appear in monosymptomatic forms, but we have never experienced a case of DCS where peripheral, monolateral facial palsy was the only symptom or sign, nor have we come across any in the extensive literature on DCS. We thus consider this diagnosis unlikely. A thorough examination of the dive profile and dive history is indispensable, though, before this diagnosis is excluded.

The remaining diagnosis, peripheral ischemic compression neurapraxia, is not merely an exclusion diagnosis. In most cases it can be supported by positive criteria. Usually the diver reports trouble with pressure equalization of the middle ears during the dive, especially on the side of the palsy. The cause of the reduced eustachian tube function is most frequently a common cold or other type of upper respiratory tract infection. Although taught not to dive during such infections, divers often disregard this warning. The diver also experiences a feeling of pressure or even pain in the ear(s) during and after ascent. After surfacing, the ear in question feels plugged, and on otoscopy the tympanic membrane is found to bulge outward due to the relative overpressure in the middle ear.

As illustrated in Fig. 4 the facial nerve carries fibers with different qualities. Ischemic dysfunction of the motor fibers (9 in Fig. 4) will cause the most striking symptoms. A paresis or paralysis of one side of the face is easily recognized. The inability to close the eye will be especially alarming, a sign present when the upper motor branch is significantly affected. Less readily recognizable are symptoms from the chorda tympani portion of the nerve (8 in Fig. 4). Reduced salivation from the sublingual and submandibular glands on one side will never be detected by the diver. A changed quality of taste on one side of the distal tongue is usually described as either “a metallic taste” or “the tongue feels strange.” Involvement of the stapedial nerve (7 in Fig. 4) will not be detected by the patient. Reduced lacrimation in one eye, due to involvement of the superficial petrosal nerve fibers, will probably only be recognized by the patient if the lesion persists for some time, as in the Australian case briefly described herein. For these nerve fibers to be affected by the postulated mechanism, the dehiscence must be located in the upper part of the middle ear, close to the geniculate ganglion (see 2 and 3 in Fig. 4).

If the brief case descriptions presented here leave readers with questions regarding details in symptomatology, the original reports should be studied.

In divers with ischemic compression neurapraxia treatment should not be delayed while the level of the lesion is established by means of special tests.

It is possible to demonstrate the fallopian canal in the temporal bone by means of conventional x-ray tomography, but the method is not sufficiently accurate to definitely decide whether there are dehiscences in the wall of the canal. A high-quality CT scanner can give a fairly reliable answer to that question, but this examination is expensive and time consuming, and we have not subjected any of our cases to such an investigation.

TREATMENT MODALITIES

The described compression ischemia in a surfaced diver can be treated in three different ways:

I. The relative overpressure in the middle ear, as demonstrated by a bulging tympanic membrane, may be relieved by:

a. Contracting the muscles that open the eustachian tube (of which the most important are m. levator and m. tensor veli palatini) by yawning or swallowing.

b. Toynbee’s maneuver (swallowing with pinched nose).

c. Decongestants (oral and/or topical) may be tried, especially if the diver has an upper respiratory tract infection. Divers regularly use such medication to overcome pressure equalization difficulties, but we are not aware of any controlled trial proving its efficiency. Phenylpropanolamine chloride should be avoided before sport competitions because according to international regulations it is an illegal stimulant.

d. Myringotomy. This possibility will probably never become necessary and has not been reported in this condition.

II. Instead of relieving the relative overpressure in the middle ear, the ambient pressure can be raised to an equivalent level. As we have demonstrated in Fig. 3 the ambient pressure will be transmitted throughout the body and thus be added
to the pressure in the vascular system. Consequently, the capillary circulation will begin to return when the sum of the pressure produced by the heart (approximately 45 cmH₂O at capillary level) and the ambient hydrostatic pressure exceeds the middle ear pressure. This will occur at depths in excess of 35 cmH₂O. At a depth of 80 cmH₂O (0.8 msw) the perfusion pressure will be back at normal and the ischemia will resolve. This reasoning is simplified, but correct in principle and can be applied to the pressure gradient theory (8) illustrated in Fig. 2. See also Fig. 3.

III. Instead of resolving the ischemia by restoring the circulation, one could improve the oxygenation locally by breathing pure oxygen at surface pressure. The high diffusion gradient toward the ischemic area should help to relieve the effects of ischemia until excess gas in the middle ear is either vented through the eustachian tube or absorbed through the mucosal lining of the middle ear.

Two or more of the above remedies could be combined. Hyperbaric oxygenation (HBO), for instance, combines the principle of breathing pure oxygen, as used in III, with raised ambient pressure, as advocated in II. In the following it will be seen that varieties of all three therapeutic modalities have proved successful.

REPORTED CASES

Only 23 subjects with transient peripheral monolateral ischemic compression neurapraxia of the seventh cranial nerve from diving and aviation are reported in the available literature, but from personal communications we know that the condition is underreported and sometimes misdiagnosed. As shown in Table 1, 20 of the 23 cases occurred from diving and only 3 from aviation. Information about the age is missing in 6 of the cases. The mean age of the remaining 17 subjects is 29.2 yr, the median age 28 yr, and the age span is 17–58 yr. In 4 cases information about sex is missing; of the remaining 19 subjects, 18 are male and 1 is female. This skewed sex distribution probably illustrates the traditional male dominance in diving rather than suggesting any sex difference in the incidence of facial canal defects or resistance to compression-induced ischemic neurapraxia.

In 1967 Bennett and Liske (45) reported two male American pilots aged 30 and 40 yr, respectively, who experienced transient seventh nerve paralysis during ascent in aircrafts. One experienced three episodes on the right side within some weeks, associated with difficulties clearing his right ear. The episodes lasted from 3 min to 3 h and they all cleared in close and direct relation to the spontaneous venting of the relative overpressure in the middle ear. It is interesting to note that in this case the facial nerve seemed to tolerate a degree of ischemia sufficient to produce neurapraxia for approximately 3 h, apparently without permanent injury. Sequelae are reported in the cat after approximately 3.5 h of ischemia (13). It is puzzling that this pilot had always experienced difficulty in equilibrating middle ear pressure during his flying career of approximately 2200 h, but never experienced facial nerve trouble until he had 3 episodes in the course of some weeks.

The other pilot experienced 2 episodes, one on each side, in the course of 2 wk. Both lasted 35 min and cleared when the ear vented spontaneously, releasing the increased middle ear pressure. The middle ear pressure relative to ambient pressure in these cases is not known, but from the ascents performed the theoretical maximum
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Abbreviations = DD: Differential diagnosis  
N: Normalization  
RS: Recompression in the sea  
S: Sequela  
RC: Recompression in a chamber.
can be calculated and varies between approximately 174 and 350 cmH₂O, which is far above the mean capillary perfusion pressure.

In 1972 Bannery (47) reported one 30-yr-old male sport diver who experienced two episodes of facial baroparesis on the left side. X-ray tomography suggested a dehiscence in his left fallopian canal. The first episode, a complete paralysis, occurred on surfacing from a scuba dive to 5–6 msw. After 0.5 h it was treated by a slow, in-water recompression. The diver descended to 4–5 msw in 5 min, and after 5 min at depth all symptoms and signs were gone. There was no relapse after a slow, 5 min ascent to the surface. Two weeks later he experienced another episode after a series of up to 100 breath-hold dives. Even during breath-hold diving, extra gas is added to the sinuses and middle ears at depth to accommodate to the raised ambient pressure. This episode was successfully treated by recompression in the water, and all symptoms and signs disappeared after some minutes. In decompression sickness, in-water recompression is generally not recommended or is at best controversial. In facial baroparesis, however, a short drop to 2–3 msw is all that is required, provided the diver ascends slowly, performing Toynbee’s maneuver during ascent. Thus, in the absence of a pressure chamber on site this therapeutic modality is recommended, even as a diagnostic test.

Bannery (47) also quotes Demard, who had treated a similar case which lasted for several weeks. He was unable to demonstrate any dehiscence in the fallopian canal by x-ray tomography. There is no mention of recompression in this case, so it is likely that the ischemia was of sufficient duration to cause severe edema, which could explain the delayed healing. The examination technique used to search for a dehiscence is not accurate enough to definitely state that a defect was not present. Bannery cites another case of facial paralysis of 48-h duration in a diver, but further details are not offered.

In 1974 Fortes-Régo (48) reported a 28-yr-old male sport diver who experienced one episode on the right side as he came out of the water after a scuba dive to 18 m in fresh water. Due to the lack of medical service in the area he was not examined until 4 mo. later when the paresis was still not completely resolved. As in Demard’s case mentioned above, the reason for the protracted course and possible permanent injury could be prolonged ischemia due to the lack of recompression and the diver’s possible failure to vent his middle ear. Subsequent edema, demyelination, degeneration, and possible intrafunicular fibrosis would be a natural consequence of prolonged ischemia.

In 1978 a 29-yr-old male airline passenger was reported to have experienced three episodes of left facial paralysis on ascent to or at altitude during overseas flights (46). On each occasion the paralysis resolved before landing. The cabin altitude in a commercial aircraft cruising at 35,000 ft (10,668 m), which is normal for overseas flights, is approximately 8000 ft (2438 m). If the flight started at sea level the theoretical maximum pressure differential between the middle ear and the cabin atmosphere at cruising level would be 266 cmH₂O, which greatly exceeds mean capillary blood pressure.

In 1978 McCleve (49) reported a patient who sustained temporal facial paralysis immediately after ascent in scuba diving, as verified by movies, but further details are not given.
In 1979 Molvær (50) reported the case of a 25-yr-old male scuba diver who experienced a transient peripheral facial palsy on the right side a few minutes after surfacing from a depth of 12 msw. He also suffered alternobaric vertigo, indicating reduced eustachian tube function. He had donated 500 ml of blood less than 24 h before the dive and was thus in a phase of blood dilution, which could make him more susceptible to ischemic compression neurapraxia. This diver could easily have experienced a middle ear overpressure far above capillary blood pressure while ascending from 12 msw to the surface. After 20 min the symptoms started to subside, and within 1 h he was completely restored to normal. This slow, spontaneous healing could be caused by a spontaneous, slow venting of the postulated overpressure in the middle ear.

In 1981 an Australian case was reported (51) in which a 58-yr-old male hookah diver experienced right ear pain and right peripheral facial palsy shortly after surfacing from a dive to 43 ft (13 msw) for 75 min, which is well within the limits for no stop recompression. He had encountered considerable difficulty in clearing his ears. After 70 min he was able to close his eye. He was then recompressed to 30 psi (21 msw) in a monoplace chamber breathing oxygen, and all acute symptoms disappeared. After the HBO treatment, barotrauma and slight deafness of the right ear were demonstrated. This barotrauma could have been inflicted during the sea dive or during the recompression, or both, or could have been a delayed barotrauma occurring after the oxygen breathing, a well-known injury in aviation (52). The diver also suffered dryness and soreness of his right eye due to a nonfunctioning lacrimal gland, explainable by compression ischemia of the nervus petrosus superficialis major, a branch leaving the facial nerve close to the geniculate ganglion (see 1 and 2 in Fig. 4).

In 1982–1983 Becker (53, 54) reported a 28-yr-old male American commercial diver who experienced transient right peripheral facial palsy in 3 separate scuba dives to 60, 40, and 15 fsw (18, 12, 4.5 msw) within 3 mo. On all 3 dives he encountered difficulty equalizing the middle ear pressure, especially on the right side, and worst on ascent when he experienced dizziness as well, indicating alternobaric vertigo. A metallic taste on the right side of the tongue preceding the facial paralysis indicates he had early involvement of the chorda tympani branch of the seventh cranial nerve (8 in Fig. 4). After several minutes the ear vented spontaneously and within 45 min all symptoms and signs had cleared. The course was identical during all 3 episodes. Examination revealed poor eustachian tube function and absent acoustic reflex on the right side, suggesting injury to the stapedius nerve, a branch from the facial nerve to the smallest striated muscle in the body (7 in Fig. 4). Becker also mentions three other cases, but details are not given.

In 1983 Vincey and Renon (55) reported 2 cases from France in which divers experienced transient right-sided facial palsy in connection with scuba diving. One, a 29-yr-old female sport diver, recognized a pain in the right ear and facial palsy 5 min after the last of 4 dives to 16 msw. When examined 40 min later the paresis was gone, but slight signs of middle ear barotrauma were found. A transient hearing loss of 60 dB at 6 kHz in the right ear could have been caused by additional barotrauma to the inner ear.

The other, a 24-yr-old male Navy diver, recognized facial palsy on the right side as he left the water after a dive to 50 msw for 25 min. He made a decompression stop of 3-min duration at 3 msw. If the 25 min refers to bottom time and his breathing mixture was air, he omitted approximately 30 min of decompression time according
to the U.S. Navy air decompression tables (56). In addition to the facial palsy, he reported tongue deviation to the left. If that observation is correct the XIIth cranial nerve was also affected, but on the left side. Since the hypoglossal nerve is nowhere related to a gas-filled space, barotrauma cannot have caused the reported malfunction. This fact, combined with the possible omitted decompression time, may suggest decompression sickness as a differential diagnosis in this case. Twenty minutes later recompression and adjuvant therapy led to normalization before reaching a depth of 30 m.

In 1983 Renon et al. (57) reported another French case, a 24-yr-old male diver who experienced a right facial palsy after a 20-min dive to 24 m. Difficulties with pressure equilibration of the ears were encountered during descent and pain in the right ear during ascent. At the surface he also recognized reduced hearing in the right ear. Normobaric oxygen breathing was started at once, and after 15 min the paresis cleared.

A British case was reported in 1983 by Shepard et al. (6). This involved a 35-yr-old male salvage diver who reported pain behind the right ear, which he could not clear, and subjective deafness 10 min after surfacing from a dive to 12 fsw (3.7 msw) for 25 min. He complained of numbness of the right side of his face where a facial paresis was observed. Within 10 min of the onset of symptoms the ear cleared and all symptoms and signs rapidly resolved. He had previously experienced difficulty with ear clearing and symptoms of alternobaric vertigo, indicating poor eustachian tube function. Thus a middle ear overpressure exceeding capillary blood pressure is likely in this case.

In 1984–1985, 5 more cases from Norwegian waters were reported (58–60). One was a 17-yr-old male sport scuba diver who experienced two episodes on the left side, the first after a no stop dive to 20 msw and the second to only 4 msw. He always had some difficulties equalizing the pressure to his left ear, and on these two occasions he had a slight common cold as well. The two dives were 6 mo. apart. During ascent he felt pressure in his left ear and a few minutes after surfacing the left side of his face became paralyzed. After 15 min the pressure in his ear gradually decreased and the paralysis disappeared.

Case two was a 19-yr-old male Navy diving student who encountered difficulty with pressure equalization of his ears due to a common cold while performing a scuba dive to 9 msw. During ascent he felt pressure, increasing to a sharp pain in his right ear. A few minutes after surfacing, the right side of his face became paralyzed. The right ear felt plugged and the tympanic membrane was bulging. Within 10 min he was recompressed in a chamber, and when he reached 2 m depth the tympanic membrane was in mid position and all symptoms and signs had disappeared.

Case three was a 23-yr-old male Navy diving student who experienced pain in his right ear during ascent from a scuba dive to 5 msw. A few minutes after surfacing he developed a facial palsy on the right side, and his right tympanic membrane was bulging. He was recompressed 15 min later in a chamber, and at 1 m depth his tympanic membrane had returned to mid position and all symptoms and signs had disappeared.

Case four was a 20-yr-old male Navy diver who performed repetitive breath-hold dives to 10 m depth in spite of having difficulty clearing his left ear due to a slight common cold. After his last dive he felt a persistent pressure in his left ear, and after a few minutes a peripheral facial palsy developed on the left side. His left tympanic
membrane was bulging. After less than 30 min the ear suddenly vented, the tympanic membrane returned to normal position, and all symptoms and signs disappeared in 30 s.

The last case was a 38-yr-old male commercial diver who dived to 7.5 msw in spite of having a slight common cold and difficulties clearing his ears. On surfacing he developed a facial paralysis on the right side. He was recompressed in a chamber, and on reaching 1.5 m depth the paralysis disappeared.

DISCUSSION

No case of ischemic neurapraxia of the seventh cranial nerve is described in He diving. Since the condition occurs infrequently this could be just chance, or due to helium's properties as a light, fast-diffusing gas used in saturation diving with slow decompressions. Facial baroparesis would therefore be unlikely to occur with He diving even if the diver had dehiscences in his facial canals and suffered a common cold during decompression.

Given that dehiscences are common, one might wonder why facial baroparesis is not observed more frequently. We know that the condition is underreported, misdiagnosed, and even concealed by the diver. But more puzzling is that in most cases it seems to occur only once in a lifetime, like a Bell's palsy, in spite of regular flying or diving. Moreover, chordae tympani are directly exposed to the middle ear pressure whether dehiscences are present or not, leading one to suspect that injury should occur commonly in diving. However, judging from middle ear surgery, this facial nerve branch seems to be rather resistant to trauma. Additionally, the transient symptoms of reduced sense of taste on the ipsilateral, distal part of the tongue, possibly combined with a slight sensation of numbness and a metallic taste in the same area, would hardly be reported or even recognized as being related to the activity of diving or aviation. Since facial baroparesis rarely occurs more than once or twice in the same person in spite of regular exposure to an alternobaric environment, one may speculate that certain conditions must be present simultaneously to precipitate the palsy. Reduced eustachian tube function due to a common cold in addition to changing ambient pressure is a common denominator in 4 of the divers described by Eidsvik and Molvær (60). Bennett and Liske (45) found chronic rhino-pharyngitis and sinusitis in one of their subjects who also was a heavy smoker. In fact, in most of the reported cases the eustachian tube function is stated to be reduced either chronically or in connection with the actual exposure. Another conceivable cofactor in the pathogenesis would be a concomitant drop in blood pressure, analogous to Tzadik's cats (12).

It has been speculated that in the majority of cases not related to diving or aviation, acute peripheral facial palsy is part of a cranial polynuropathy which may be caused by an infection with one of the neurotrope viruses (61). Perhaps a latent, subclinical infection of that kind would render the nerve more susceptible to neurapraxia in compression ischemia in divers with facial canal dehiscences. The need for such "priming" of the nerve for an ischemic neurapraxia to occur in connection with middle ear overpressure in a case of fallopian canal dehiscences could explain why this type of palsy happens only occasionally in persons who regularly expose themselves to great changes in ambient pressure.
The geographic distribution reflected in Table 1 may be caused by the distribution of diving activities in the world, a difference in interest in diving medicine in different areas, a difference in the publishing policies in different areas, or an incomplete literature search due to the authors’ lack of knowledge in many of the world’s major languages.

From Table 1 it seems evident that the right side is more frequently affected than the left, but we are unable to explain why. Only one case, an aircraft pilot (45), was affected bilaterally, but at consecutive times. To our knowledge, no case has been reported where both sides were affected simultaneously. Why the aircraft pilot did not experience bilateral palsies instead of one right and one left, is somewhat puzzling, but could be explained by asymmetric and changing eustachian tube function.

Spontaneous or aided venting of the excess middle ear gas resulted in quick healing of 17 episodes in 9 subjects, whereas recompression in a chamber effectively dealt with 5 episodes in 5 subjects. In-water recompression proved equally efficient in 2 episodes in 1 subject, as did normobaric oxygen breathing in 1 episode in 1 subject. The only reported case with permanent motor injury (48) and the 2 cases with a protracted course of motor dysfunction (47) received no treatment. But the case with prolonged (permanent?) lacrimal gland hypofunction had been treated by HBO (51).

It is possible that the 7 cases (8 episodes) treated with recompression and surface oxygen breathing would eventually have recovered spontaneously. In our opinion, though, it would be unethical to withhold any available effective means of therapy. To the extent that experimental observations of irreversible injury in cats after 3.5 h of facial nerve ischemia (13) can serve as a guide, treatment of human cases should be instituted within 3 h of onset.

Three of the cases recompressed in a chamber breathed air, while 2 received HBO. It is interesting to note that HBO has also been used in the treatment of idiopathic facial palsies with favorable results (62, 63). Inasmuch as edema and resultant compression ischemia are thought to be the main pathogenetic mechanisms in such cases, HBO might work by reducing swelling through vasoconstriction and thereby restoring circulation and lymphatic drainage of the edema. Before that mechanism is effective, oxygen diffusion into the ischemic area should be beneficial.

In cases of baroparesis of the facial nerve Renon et al. (57) recommend the use, for some days, of vasodilators, corticosteroids, and antiplatelet aggregating drugs in addition to recompression. We have not used these medications in any of our cases. If the ischemic neurapraxia is not relieved quickly, venous stasis and edema can be a problem. We do not know whether corticosteroids would prove beneficial in that case. Since the basic problem is probably a vasoconstriction rather than a vasoconstriction, the use of vasodilators may not help in the acute phase. Later there is a theoretical risk that it may harm by increasing the edema. Unlike the problem of intravascular gas phase in decompression, the cases with compression neurapraxia should not suffer hemoconcentration or platelet adhesion. Consequently, any medication to reduce platelet aggregation should not be needed. In our opinion the 3 treatment modalities outlined in this article, or combinations of them, should prove sufficient if instituted without delay. Later, additional therapy, such as the corticosteroids proposed by Renon et al. (57), could be considered.

CONCLUSION

In conclusion we must state that we are unable to prove what the pathogenetic mechanism of facial baroparesis is, although all available information supports the
ischemic compression neurapraxia theory. Barotrauma of descent could cause edema and even bleeding in exposed parts of the nerve, and we cannot exclude this mechanism as a "primer" for the further events. However, available evidence depicts barotrauma of ascent, with compression of the nerve, as the culprit. While spontaneous recovery was not uncommon in this rare type of palsy, both recompression and surface oxygen breathing proved effective in 8 episodes in 7 subjects.

Drawings by Ivar Rønnevold. — Manuscript received for publication April 1986; revision received August 1986.

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