Chapter 7: Pathophysiology of the ears and nasal sinuses in flying and diving

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No study of otolaryngology is complete without a description of the physiological and pathological conditions which may affect the ears and nasal sinuses in aerospace and underwater. This is borne out by the importance of aviation, both civil and military, in the modern world and by the rapid development of the underwater industry. This view is consolidated by the millions of passengers who fly each year, and the thousands who fly and dive for a hobby.

Physical laws relating to aerospace and the underwater environment

The response of gases when subjected to pressure must be understood as this governs the behaviour of gas in the tympanic cavity, the middle ear, and the nasal sinuses. This is exemplified by Boyle’s law, which states that the pressure and the volume of an enclosed fixed mass of gas are inversely proportional.

It is convenient for descriptive purposes to start at sea-level; during an ascent through the atmosphere there is a progressive reduction in pressure. In general terms, at 18,000 feet (5,500 m) above sea-level the pressure is half that at sea-level, and is halved again at 34,000 ft (10,300 m); the change in differential pressure with altitude is greater at relatively low altitudes than at a greater height. In practice, during ascent through the atmosphere, a given mass of gas contained within an elastic structure will expand.

In the middle ear, this gaseous expansion will push the tympanic membrane to the natural limit of its excursion, to be followed by an easy and involuntary escape of air along the eustachian tube (Hartmann, 1879; Armstrong and Heim, 1937). Movement of the membrane may be restricted by scarring or by calcareous deposits, and this may give rise to aural pain during ascent.

During descent from altitude, with an increase in the atmospheric pressure, there is a decrease in volume of the middle-ear gas. The eustachian tube must be opened by swallowing movements to adjust the volume. If this mechanism fails or if it is delayed, an increasing differential pressure will act on the soft nasopharyngeal end of the tube to close it. When this pressure is greater than can be generated by the tubal dilator muscles, the tube will stay closed and is said to be 'locked'. Thereafter, with continued descent the pathophysiological changes of barotrauma are inevitable. Armstrong and Heim (1937) showed that positive extratympanic pressure of 90 mmHg (12 kPa) will lock the tube, but this figure depends on the intrinsic strength of the tubal dilator muscles, and so it is variable.

Boyle's law is still applicable underwater. At water surface, the ambient atmospheric pressure is 14.7 pounds per square inch (psi) or a pressure of 1 atmosphere (1 atm or 101 kPa). In descent from the water surface, the pressure increases rapidly in a linear fashion as a result of the density of the water, so that at 33 ft (10 m), the pressure is double that at the surface (2 atm or 202 kPa). Every 33 ft (10 m) of descent adds 1 atmosphere of pressure (101 kPa). Compare this with the aviator’s situation, when descent from 18,000 ft (5,500 m) to sea-
level will encompass a change in pressure of only 0.5 atmosphere (50.5 kPa). In ascent to the surface from depth, a reverse state of affairs will apply.

The role of pressurization and the pressure cabin

The adverse physiological effects of flight at high altitude are almost entirely a consequence of the accompanying reduction of barometric pressure, so that the most logical way of securing satisfactory conditions for the occupants of high-flying aircraft is to provide within the aircraft an atmosphere that is at a pressure appropriate to bodily needs.

As the cabin pressure has to be greater than that of the surrounding atmosphere, the difference between the two pressures will be represented by a differential pressure tending to force the cabin wall outwards. It follows that the absolute pressure within the pressure cabin will be equal to the barometric pressure existing at an altitude below that at which the aircraft is flying. It is convenient to consider conditions inside the aircraft in terms of the altitude that is being simulated by pressurization. In pressure cabin aircraft, the terms 'aircraft altitude' and 'cabin altitude' are appropriate.

The effect of employing a pressure cabin is to reduce the range over which barometric pressure is acting, but it should be remembered that below 15,000 ft (4,500 m), the rate of pressure change will predispose to barotrauma, and this is an important factor in deciding the rate at which pressurization should diminish when an aircraft is descending.

The practicalities of the situation have resulted in the concept of both a high and a low differential cabin. The high differential cabin is employed in transport aircraft in which the conveyance of passengers makes it necessary to maintain a cabin altitude of 8,000 ft (2,440 m), even when the aircraft, like the supersonic transport, is flying at 60,000 ft (18,300 m). The maximum differential pressure involved is 430 mmHg (56.9 kPa). The low differential cabin is employed in military aircraft, when failure of the pressure cabin in combat must be accepted as an operational hazard. The risks of too great a pressure change occurring during sudden decompression are minimized by employing a relatively low cabin differential pressure. In spite of this, many military aircraft are commonly fitted with cabin pressure controllers, whose characteristics can be altered in flight. Although no variation can be made in the maximum permissible differential pressure, it is possible to select the altitude at which pressurization will start after take-off, and the rate of change of cabin pressure during ascent and descent.

In the example shown (Brown, 1965), the alternative pattern, if adopted, would reduce the risk of barotrauma when carrying out a rapid descent below 8,000 ft (2,400 m). For example, if the aircraft were descending at 1,000 ft/minute (5 m/s) below 8,000 feet, the rate of change of the cabin altitude would be 500 ft/minute (2.5 m/s) - an effective reduction.

Physiology of the eustachian tube

The function of the auditory tube is to maintain the equality of air pressure around the tympanic membrane, necessitated by the absorption of gas through the mucous membrane, and variations of ambient atmospheric pressure. This is achieved by opening the tube to permit the passage of air. In its natural state the tube is collapsed, so that its closure is a
passive process, assisted by relaxation of the associated muscles. Other related factors, probably acting in combination, are the elasticity of the cartilaginous support, the venous pressure, and the presence of a blanket of mucus in the lumen of the tube (Farmer, 1985).

The auditory tube is opened by swallowing, yawning and gaping movements. The muscles involved in the control of patency of the tube can be divided into two groups:

1) those muscles which by having an insertion into the walls of the tube exert direct action

2) those muscles which by anatomical association assist and influence tubal opening.

The muscles in the first group comprise the tensor palati, the levator palati, the salpingopharyngeus and the tensor tympani muscles. The tensor palati muscle is the most important, and McMyn (1940) believed it to be the prime mover in opening the tube, a view supported by Rich (1920) and Macbeth (1960). It is likely that tensor palati and tensor tympani muscles, with a common embryological origin, and with the same nerve supply, act synergistically. The levator palati muscle supports and holds the tubal cartilage to permit the tensor palati to act on the curve of the tubal cartilage, and so open the lumen. The muscles in the second supporting group are the upper parts of the superior constrictor of the pharynx, and the sphincter of the nasopharyngeal isthmus, of which the palatopharyngeus is a major contributor.

Otic barotrauma

Unobstructed ventilation of the middle ear will produce no changes and hence no symptoms. The term 'otic barotrauma' defines any damage to the ear which results from pressure, and it has been known as aerotitis media, aviation pressure deafness, and otitic barotrauma. This condition is likely to occur in any situation where a change of pressure acts on the middle ear and tubal system. However, concern here is with the occurrence of such a mechanism in flight, in diving, and in the simulation of these physical environments in decompression and compression chambers. In addition, patients treated in hyperbaric chambers may also suffer from otic barotrauma (Morrison, 1972).

Factors contributing to barotrauma

Any conditions which narrows the tubal lumen by oedema or by increasing the amount or viscosity of the mucus coating the mucous membrane will, by impeding the flow of gas along the tube, or by impairing the ability of the tube to open, predispose to barotrauma. The commonest predisposing causes of acute barotrauma are acute and chronic infections in the nose, particularly coryza, nasal allergy and vasomotor rhinitis - and to these should be added malformation of the nasal skeleton.

The role of overpressure in the nasopharynx

It has been seen that the eustachian tube will open passively in ascent, from overpressure of gas in the middle ear. In descent, the tube will also open from overpressure of some degree applied to the end of the tube, as in the Valsalva manoeuvre. This technique
is frequently used in flight and, in effecting tubal clearance, it is no less successful when used in diving. The subject attempts forcible expiration with the lips closed, and the nostrils occluded by digital compression of the nose. In doing so, the air pressure in the nasopharynx is raised to force air along the auditory tube to the middle ear. The manoeuvre has the disadvantage that it may cause syncope from the increase in central venous pressure and pooling of venous blood resulting from the raised intrathoracic pressure. In addition, pulmonary stretch reflexes may induce cardiac arrhythmias (Duvoisin, Kruse and Saunders, 1962), so that there is a potential hazard with this method.

A procedure developed by Frenzel (1938, 1950) consists of closing the glottis, the mouth and nose, while at the same time contracting the muscles of the floor of the mouth and the superior pharyngeal constrictors. It is independent of intrathoracic pressure and can be performed in any phase of respiration. Chunn (1960) found that with this manoeuvre, the mean tubal opening pressure was 6 mmHg (0.8 kPa) compared with a mean opening pressure of 33 mmHg (4.4 kPa) with the Valsalva method.

Pathophysiology of barotrauma

The pathophysiology of otic barotrauma was given a definitive description in the work of Dickson, McGibbon and Campbell (1947), in which cats were decompressed to a pressure corresponding to an altitude of 20,000 ft (6100 m) and then recompressed. The histological changes seen were all vascular in nature, and included mucosal congestion, oedema, haemorrhage, effusion and polymorph infiltration. These are related to the subambient pressure in the middle ear, which will become clinically manifest by invagination of the drumhead, congestion, solitary or multiple haemorrhagic bullae, blood or fluid in the middle ear and, in some instances, rupture of the tympanic membrane.

In the situation of a descent without ventilation of the middle ear from, say, an altitude of 10,000 ft (3050 m) to sea-level, where the ambient pressure is 760 mmHg (101 kPa), the middle ear will contain air at a theoretical pressure of 523 mmHg (70 kPa) and the pressure difference across the tympanic membrane will be 237 mmHg (31 kPa). The absolute pressure within the blood vessels in the tympanic membrane is the sum of the ambient pressure (atmospheric pressure) plus the present blood pressure. If the capillary pressure is of the order of 20 mmHg (2.7 kPa), then the absolute blood pressure in the capillaries will be 780 (760 + 20) mmHg (103.7 kPa); and as the pressure of the tissue fluid surrounding the vessels is only 523 mmHg (70 kPa) the vessels will become passively engorged.

Rupture of the tympanic membrane

The anteroinferior portion of the tympanic membrane is the site where tears commonly occur, but previous scars are also a site of predilection. In flight, barotrauma associated with a fast rate of descent may cause avulsion of the drumhead from the tympanic ring. King (1976) reported rupture of the drumhead in 38 of 897 ears which had sustained barotrauma in flight. Underwater, rupture of the drumhead is likely with unrelieved tubal obstruction on descent to depths greater than 16 ft (4.8 m).
Rupture of labyrinthine windows: inner ear barotrauma

Hughson and Crowe (1933) demonstrated that, during a rise in pressure of the cerebrospinal fluid, the round window membrane would bulge outwards into the tympanic cavity. In 1971, Goodhill reported spontaneous rupture of the round window membrane caused by a rise in intracranial pressure resulting from coughing, sneezing or straining, and he believed that a sudden change in the middle-ear pressure in flight might have the same effect. Work by Tingley and MacDougal (1977) suggested window rupture as the cause of symptoms of alternobaric vertigo in some instances.

The operation of stapedectomy may involve a special risk in flying and diving. The possible fate of the stapedectomized ear in flight has been investigated by Rayman (1972); sudden pressure change may involve the disruption of the artificial stapes from the oval window, or its impaction into the inner ear. Either of these situations will produce sudden severe sensorineural loss, and possibly incapacitating vertigo. This places a special responsibility on the surgeon when advising this operation and he should warn of the possible hazards to the ear. In this connection, a fat and wire assembly is considered the safest of the many different combinations of prosthesis and graft that are utilized in this procedure.

The concept of round window fistula is of particular relevance to the hyperbaric environment of the diver (Edmonds and Freeman, 1972; Edmonds, 1973b). Goodhill (1971) suggested that in some individuals an infantile type of cochlear aqueduct persisted, with the consequent loss of the protective effect of the long, narrow adult aqueduct in the reduction of fluctuations in pressure differential between the cerebrospinal fluid and the perilymph. Increased cerebrospinal fluid pressure could cause a sudden rise in perilymph pressure within the scala tympani, leading to rupture of the round window membrane. In diving, the physical exertion plus the hyperbaric environment will increase the cerebrospinal fluid pressure. A susceptible diver, if exposed to a sudden and rapid increase in depth, may thus suffer both from middle-ear barotrauma and round window fistula.

Delayed otic barotrauma

Otic barotrauma is normally associated with physiological changes at the time of the alteration in pressure. However, there are occasions when subjects may experience an incidence of deafness and discomfort in the ear several hours later. This delayed form of barotrauma occurs after long flights, when the breathing of 100% oxygen has resulted in a raised tension of that gas in the middle ear. With passive collapse of the tube, as happens in sleep, and with no active inflation of the middle ear, the absorption of oxygen through the middle-ear mucosa results in the development of a significant pressure differential. Comroe et al (1945) reported the condition originally, and Jones (1958, 1959) has also studied the significance of oxygen absorption in this context. The signs are minimal, consisting of invagination of the drumhead and, in some cases, a suspicion of fluid in the middle ear.

Chronic otic barotrauma

This is a clinical rather than a physiological entity in which one episode of barotrauma predisposes to another. Two factors are considered to be responsible:
(1) the original predisposing factor may itself be chronic

(2) oedema and interstitial bleeding in the tubal mucosa, resulting from the original barotrauma, may reduce the tubal lumen, so predisposing to further attacks; in practice, in both aviation and diving, sufficient time is not always given for the basic lesion to recover before the patient is again exposed to pressure change.

'Reversed ear' (reverse ear squeeze) - barotrauma of the external auditory meatus

This condition will occur on descent, both in water and in the air, if an obstruction at the meatal entrance prevents an increase in pressure in the external canal, in the presence of ambient atmospheric pressure in the middle ear through an open eustachian tube. The tympanic membrane bulges outwards leading to possible rupture, and blood blisters may form in the external canal which may also rupture. In flying, reverse ear squeeze is caused by the fitting of a tight earplug, whereas in diving, the most likely cause is the compression, by increasing water pressure, of the soft hood of a Scuba suit against the pinna (Jarrett, 1961).

The management of otic barotrauma

The immediate aim of treatment is to relieve pain, and simple analgesics will usually suffice. Pain is greatest at the time of change of pressure, and eases once the situation has stabilized. Persistence or worsening of pain suggests that otitis media has supervened, although this is rare.

The second principle of treatment involves the ventilation of the middle-ear cleft. Decongestants in spray form for the nose, or antihistamines, are helpful both as a first-line treatment and as a continuing supportive measure. Early eustachian catheterization and inflation used to be the popular form of treatment, but they are considered unproductive in a severe otic barotrauma. Instant relief can be obtained by myringotomy, a method much in favour in the USA, and this was recommended over 40 years ago by Canfield and Bateman (1944).

Delay in recovery, the persistence of fluid in the middle ear, or recurrent barotrauma all call for myringotomy, suction, and the insertion of a ventilation tube. This method is of value to professional aviators as it permits the continuation of trouble-free flying while the aural condition resolves. This is not the case in divers, who, once a ventilation tube is fitted, are advised to stay out of water until resolution is complete. As a precipitating factor for otic barotrauma is commonly found in the nose or sinuses, a thorough search of this area must be made for any lesion or abnormality, which, if present, should be treated on its merits. It has been shown (Dickson and King, 1956) that, where surgical treatment is indicated and carried out, a high rate of functional recovery can be expected. The passage of time has not changed this view, which has been reaffirmed by McNicholl (1982). He found that 34 of 37 naval divers were able to equilibrate middle-ear pressure during descent only after undergoing nasal septal surgery.

If rupture of the tympanic membrane occurs it is best left undisturbed, apart from careful cleansing of the ear to remove blood or loose clot. Many of these ears will heal of their own accord. Of those that do not heal, some form of tympanoplasty may be required to
produce a safe ear, although it should be stressed that there is no physiological reason why an individual with a clean dry central perforation should not fly.

Where flying personnel and divers are concerned, the surgical result from tympanoplasty must conform to the physiological requirements demanded by the individual's working environment. This means that the repaired tympanic membrane should look reasonably normal, move well during pressure change, and that the hearing should be within the practical limits required by the patient's occupation. These functional and practical criteria are not always easy to meet. In any event, and no matter what treatment is given for barotrauma, sufficient time should be allowed for the condition to resolve itself, and for the eustachian tube to open properly, before a return to flying or diving is permitted. Apart from visual inspection, pure-tone audiometry and tympanometry are useful and often assist in making an assessment.

**Sinus barotrauma**

The paranasal sinuses also contain air, with the consequence that barotrauma of these structures may occur in flying and diving. The syndrome encompassing the development of pain in the frontal area or the cheeks during or shortly after pressure change, and sometimes associated with rhinorrhoea and epistaxis, is called sinus barotrauma. Other descriptive terms are aerosinusitis, barotraumatic sinusitis and dysbarism.

The real frequency of the condition is not known, and estimates vary. Dickson and King (1954) reported a ratio of 5:2 when comparing otic with sinus barotrauma in a series of 328 patients suffering from barotrauma. In describing the distribution, King (1965) reported involvement of the frontal sinus(es) in 80% of cases of sinus barotrauma, with antral implication in 29%, with some 10% of cases having both the antra and the frontal sinuses affected.

The production of pathology as a result of a change of pressure will hinge on the degree of patency of the sinus ostium, and hence, to a large degree, on nasal function. Unlike the middle ear, with ventilation through the eustachian tube, there is no voluntary control over the diameter of the sinus ostium so that equalization of pressure may be difficult. The Valsalva manoeuvre may sometimes be effective, but it cannot be relied upon.

The sinus ostium may be reduced by a plug of mucus, mucosal oedema, or by some mechanical feature such a polyp or neoplasm, although this latter occurrence is rare. The obstruction is often of a valvular nature, so that air passes easily in one direction only. Adequate ventilation of the nasal sinuses is related closely to nasal function, with the consequence that factors creating oedema of the nasal mucosa are commonly found as predisposing causes, for example coryza and its infective sequelae, vasomotor rhinitis, seasonal allergy, and the effects of mechanical obstruction from nasal injury and deflection of the nasal septum.

During descent in air or water (the compression phase), sinus barotrauma may be caused by obstruction of the ostium from the nasal side. With unrelieved obstruction, and continuing descent, the decreasing volume of air in the affected sinus exerts a suction effect on the lining mucosa and enhances the ostial block. On ascent, the ostium may be blocked
from within the sinus cavity, and the symptoms are caused by the unrelieved expansion of the gas contained in the sinus.

During descent, either in air or in water, the absolute blood pressure increases steadily. If there has been ostial obstruction during this phase, the gas in the sinus will remain at a relatively low pressure. The pressure differential between the mucosa and the interior of the sinus will increase, and the mucosa will become engorged. This can lead to rupture of mucosal vessels, the formation of a subepithelial haematoma, and even frank haemorrhage. The degree of pathological change is proportional to the magnitude of the pressure differential and the period of time over which the pressure inequality is unrelieved. Because of the greater pressure differential in diving, the mucosal changes can be expected, on average, to be greater than those experienced in flight.

Pain is the main symptom, which is frequently localized to the affected sinus. It often originates above the eyes, and spreads to the temples and the vertex; facial discomfort and pain in the upper teeth may also occur. The pain is of sudden onset, it is often severe, and may precipitate fainting. Nasal bleeding can be profuse and there is often a discharge of straw-coloured fluid from the nose. There may be a blood clot in the nasal fossae, and the affected sinus may be tender.

Radiological examination can help to determine the site and size of the lesion in as many as 75% of cases, and the use of radiology can be useful in assessing the progress of treatment. However, it should be remembered that this type of examination does not necessarily distinguish between lesions contributing to the production of barotrauma, and those which are the result of it (McGibbon, 1947).

**Management**

Treatment follows accepted principles which are aimed at relieving pain and achieving adequate ventilation of the affected sinus. This latter is accomplished by nasal decongestants and antihistamine preparations. Any predisposing factor should be dealt with in order to reduce the risk of recurrence, and simple surgical treatment, such as submucous resection of the septum, antrostomy, polypectomy, or enlargement of the frontonasal duct, may be required.

**Delayed sinus barotrauma**

The production of delayed sinus barotrauma is uncommon, and generally occurs after flights in which high concentration of oxygen are inhaled, thus raising the tension of the gas in the sinuses. If an ostial block occurs, oxygen is absorbed through the mucosa; a pressure differential develops which may engender local sinus pain and may be followed by generalized headache.

**Pneumocoele**

Pneumocoele of the antrum is rare, and is aggravated by high altitude flying. Although few cases have been reported (Noyek and Zizmor, 1974; Zizmor et al, 1975), the condition is included here for completeness. A structural anomaly at the ostium creates a valve that
permits air easily to enter the antrum, but prevents it from escaping. Flight may induce a feeling of pressure in the cheek, worsened by sneezing. In one reported case, the Valsalva manoeuvre produced a fluctuant swelling through a dehiscence in the lateral wall of the antrum; radiology showed an expanded translucent antrum.

**Dental barotrauma**

Pain of dental origin may occur both in flying and diving. In view of the close proximity of the second premolar and the first two molars to the antral floor, it is important to differentiate dental barotrauma from maxillary sinus barotrauma. In the case of military aircrew, pain of this type is not uncommon. Ashely (1977) reported that one in five fighter pilots in the US Air Force suffered from such pain, while a French Air Force survey reported an incidence of 6% in flying personnel. In non-vital teeth, the pulp may become necrotic, probably in consequence of restoration or the use of instant fillings. Gas formation occurs in the autolysing pulp, and its expansion on ascent forces infected material into the periapical tissue, with the likely development of an abscess in the following day or two. In partially vital teeth, there may be gas bubbles in the pulp cornua under a deep cavity, although the source and composition of the gas is unknown. Severe pain in such teeth may be experienced in flight at altitudes up to 5000 ft (1500 m).

The commonest occurrence of dental pain in flight is in vital teeth with pulpitis and, in these cases, pain typically occurs on ascent at about 7000 ft (2000 m). Such teeth need treatment, and it is sensible to stop flying for 10-14 days after the completion of treatment of a suspect tooth. In diving, implosion of a tooth can occur in descent when a cavity with thin cementation is present (Edmonds, 1976), while in ascent, pain has resulted from lifting of enamel or crown (Leitch, 1985). In view of the poor critical localization of pain from the dental pulp, all suspect teeth should be checked and their response to cold stimulation determined.

**Inner ear injuries in a hyperbaric environment**

Injuries to the cochlea and the vestibular apparatus can occur after exposure to a stable, hyperbaric environment in diving, particularly during deep dives. According to Dalton's law, each gas in a mixture of gases exerts its own pressure independently of the other contained gases, and the total pressure of the gas mixture is the sum of the partial pressures. The solubility of a gas is proportional to its partial pressure. Of the inspired, oxygen constitutes 21%, while nitrogen comprises 78%. Nitrogen is inert and, with increasing depth, more dissolves in body tissues and fluids. During decompression, nitrogen may be released as bubbles from the tissue, which will cause the 'bends'. To prevent or reduce the possibility of bends and nitrogen narcosis, an oxyhelium gas mixture is employed for deep and 'saturation' dives. In saturation diving, divers must stay at a pressure level for a sufficient time to enable their tissues to reach a state of equilibrium with the gases to which they are exposed. The usual method is for the divers to live in a complex of chambers, and to move to and from their working site in a pressurized bell (Leitch, 1985).

Lesions of the cochlea or vestibular apparatus are precipitated at the beginning of decompression when the oxyhelium gas mixture is changed to compressed air. It is unusual for both cochlea and vestibular mechanism to be damaged. The 'isobaric countercurrent
diffusion theory', developed by Farmer (1977), attempts to explain the formation of bubbles in the peri- and endolymphatic fluids by the counterdiffusion of two inert gases across the round window membrane. This is not surprising in view of the fact that when the breathing mixtures are changed, the perilymph and endolymph are saturated with helium, while the middle ear rapidly fills with compressed air.

**Decompression sickness**

Decompression sickness is a condition which has been recognized in divers and caisson workers since the middle of the nineteenth century, yet only in the last 50 years has its effect on those exposed to subatmospheric pressures been clearly described (Fryer, 1969). Paralysis, fits and other neurological manifestations from this cause are rare in aviators, and disturbances of taste, smell and hearing have not been recorded in this group.

In otological terms, consideration must be given to the way this condition affects divers, for whom decompression sickness remains a limiting factor on ascent from deep or prolonged saturation dives. As a dive increases in depth, the respired gases in the diver are absorbed into the circulation. The quantity of gas absorbed will depend on its solubility and partial pressure; it will also depend on the vascularity of the tissue, and the rate of diffusion of the gas. During decompression a reversal of this process occurs; the dissolved gases come out of solution and appear in a gaseous state in the lungs, or as bubbles in the tissues or body fluids. In accordance with Boyle's law, bubbles increase in size as decompression proceeds. Bubbles may be present within the inner ear, as well as in the eighth nerve pathway and its central connections. Intravascular gas emboli can also occur. The pathological changes, including haemorrhage, and the diverse signs and symptoms of decompression sickness have been described by McCormick, Philbrick and Holland (1973). This risk may be reduced by careful control of the depth and duration of the dive and the rate of ascent to the surface. Susceptibility to decompression sickness increases with age and obesity, while the effects of cold and dehydration, and the after-effects of alcohol, have been recognized.

The aural symptoms of decompression sickness, vertigo and hearing loss, either singly or together, are not common but are well recognized; the imbalance which can occur is referred to by divers as 'staggars'. Coles (1976) reported the incidence of these symptoms as 6 or 7% in some 100 cases of decompression sickness affecting experimental deep divers at the Royal Naval Physiological Laboratory (Alverstoke, Hants, UK). As long ago as 1909, Keays suggested that in 5% of all cases of decompression sickness, vertigo was the most characteristic symptom. It should be remembered that signs of sensorineural hearing loss in a diver may not be evidence of decompression sickness, but can be related to the high ambient noise in which the diver works, as well as to noise exposure unrelated to diving.

**Other changes in hyperbaric states**

In addition to bubbling, hyperbaric states are associated with a reduction in capillary flow and with stasis in the microcirculation. Combined with this is an elevation of the level of the serum lipid and serum cholesterol. Workers at the Institute of Naval Medicine (Alverstoke, Hants, UK) (Martin and Nichols, 1972) have found a significant reduction in the circulating platelet level after decompression. Platelet aggregates form around developing gas bubbles, and have been shown to initiate clotting (Philip, Schacham and Gowdey, 1971). This
contributes a potential hazard to the cochlea and vestibule, although no part is more at risk
than another. Microemboli from lipid, platelets or gas bubbles will lead to degeneration of the
affected sensorineural epithelium.

**High pressure nervous syndrome**

Compression, particularly rapid compression, in an oxyhelium atmosphere to depths
greater than 500 ft (152 m) may produce symptoms of the high pressure nervous syndrome
(Bennet and Towse, 1971). These symptoms are dizziness, nausea, intention tremor, and
decrement in standing steadiness. The symptoms abate in a matter of hours. Electronystagmographic (ENG) studies during high pressure nervous syndrome (Farmer, 1977)
have shown no evidence of vestibular end organ dysfunction and the mechanism of these
symptoms remains unknown, although an alteration in cerebellar function has been postulated.

**Pressure (alternobaric) vertigo**

Attention so far has been directed to the traumatic effects of pressure change on the
ear and sinuses, but it has long been recognized that changes in ambient pressure can also
cause a transient disturbance of vestibular function in the absence of overt aural pathology.
The occurrence of vertigo in aviators during ascent and descent was first described by van
Wulfften Palthe (1922). However, the condition was not clearly recognized until Jones (1957)
reported that 10% of the Royal Air Force pilots whom he had interviewed had experienced
such symptoms. A later survey by Lundgren and Malm (1966) found an incidence of 17% in
flying personnel of the Royal Swedish Air Force. The disability is even more common among
divers who are exposed to greater and more rapid changes in pressure than air-crew. In a
group of Swedish sport divers, no fewer than 33% reported that they had experienced vertigo
when diving, which Lundgren (1973) attributed to pressure change in 26% of those
responding to his questionnaire.

**Clinical features**

The characteristic features of the syndrome, termed 'pressure vertigo' by Jones (1957)
and 'alternobaric vertigo' by Lundgren (1965), is the vertigo, of sudden onset, which coincides
with the passive equalization of middle-ear pressure, either during a rapid ascent or on
producing an overpressure in the middle ear by means of a Valsalva manoeuvre during
descent or when on the ground. Typically, the vertigo is short lived, decaying within 5
seconds or less, although it can be of such an intensity that the induced nystagmus impairs
vision. Less commonly, the vertigo is weaker, with only a sensation of turning without
impairment of vision, but the vertigo is more persistent, lasting for up to one minute or
longer. There is considerable intersubject variability in the plane and direction of the vertigo,
although it is usually of a consistent pattern in any one individual. Pressure vertigo is more
likely to affect air-crew and divers when there is difficulty in equilibrating middle-ear
pressure, usually from congestion and inflammation of the nasal mucosa resulting from a
common cold or other infection of the upper respiratory tract; such an association is found
in about 70% of cases. There are, however, a few individuals who suffer from pressure
vertigo even in the absence of infection. Studies by Ingelstadt, Ivarsson and Tjernström (1974)
suggest that these susceptible individuals require a higher pressure differential between the
middle ear and ambient pressure than is the norm in order to open the eustachian tube and vent gas to the immediate surroundings.

**Pathophysiology**

The mechanism by which the sensory receptors of the vestibular apparatus are stimulated by changes in middle-ear pressure is still a matter for conjecture. The dominant symptom, vertigo, strongly suggests that it is the ampullary receptors of the semicircular canals, rather than the maculae, that are stimulated. Furthermore, the transient nature of the disturbance accords with the theory proposed by Jones (1957), namely that the cupula is deflected when the overpressure in the middle ear is suddenly relieved on passive venting, or when middle-ear pressure is raised momentarily above ambient pressure by a Valsalva manoeuvre. Overpressure in the middle ear may not be transmitted equally to the fluid systems of the inner ear by the round and oval windows, for the stapes footplate might move against the pressure gradient caused by the outward displacement of the tympanic membrane. It is conceivable that, with the sudden restoration of middle-ear pressure, there is a movement of endolymph and perilymph which causes a displacement of the cupula of one or more of the semicircular canals of the ear involved. Unfortunately, little is known about the transient response of the hydrodynamic systems of the inner ear to large amplitude pressure changes, even in the normal ear, so it is difficult to explain why some individuals show an altered pattern of end-organ activity with such a stimulus while others do not.

The work of Ingelstedt, Ivarsson and Tjernström (1974) and Tjernström (1974a) has shown that five out of 79 otologically healthy subjects exposed to simulated ascents with a pressure change of 66 mmHg (8.8 kPa) in 25 s (equivalent to an ascent from ground level at approximately 5000 ft (1500 m)/minute) developed vertigo when middle-ear volume was allowed to equilibrate passively. Indirect measurement of the middle-ear volume was used to identify the timing of tubal opening. It was found that vertigo and the concomitant nystagmus were not induced at the moment of tubal opening but, rather, that vestibular stimulation occurred when the relative overpressure in one ear was about 44 mmHg (5.9 kPa) or higher. However, not all subjects who had a high opening pressure developed vertigo. The additional requirement for the induction of symptoms was a definite asymmetry of the middle-ear pressures, caused by one ear equilibrating with low tubal opening pressures and the other needing a high opening pressure.

The demonstration that vertigo and nystagmus could be induced in susceptible subjects by an overpressure in the middle ear when there was free communication of air between the middle ear and the external canal led Tjernström (1977) to propose that pressure vertigo might be caused by a relative ischaemia of the sensory epithelium. He suggested that the overpressure in the middle ear is effectively transmitted to the fluid system of the inner ear because of poor patency of the cochlear aqueduct when there is a rapid pressure change. The estimated pressure in the capillaries of the inner ear is less than 40 mmHg (5 kPa) above ambient pressure, so if fluids of the inner ear were pressurized to more than 40 mmHg above ambient pressure, by an overpressure in the middle ear, then circulatory insufficiency affecting structures within the inner ear would be likely to ensue.

Although such a mechanism cannot be refuted, it must be pointed out that the vestibular reactions induced by the pressure change in the experimental studies of Ingelstedt
and Tjernström were relatively weak. Vascular insufficiency could well be responsible for the low grade and sometimes sustained vertigo that is reported by a minority of flying personnel with 'pressure vertigo', but in the authors' opinion, it is unlikely to account for the severe, although brief, disturbance of vestibular function that can be precipitated by equilibration of middle-ear pressure during ascent or active over-inflation (Valsalva manoeuvre) during descent.

**Management**

The established association between the incidence of pressure vertigo and the impairment of middle-ear ventilation by upper respiratory tract infections implies that the most important prophylactic measure is the restriction of the duties of air-crew and divers when they are suffering from coryza or other conditions in which there is congestion of the mucous membrane of the nasopharynx. Unfortunately, in some cases, it is the occurrence of pressure vertigo that first tells the individual that he is developing a common cold.

On their return to flying or diving following an upper respiratory tract infection, susceptible individuals should be advised to equilibrate middle-ear pressure frequently so as to minimize the development of high pressure differentials. The use of nasal decongestants may also be beneficial.

The repeated occurrence of vertigo associated with changes of middle-ear pressure, in the same way as any other form of persistent vertigo, merits withdrawal from flying or diving duties, and a full investigation. If evidence of tubal dysfunction is not found, the integrity of the round window should be determined by tympanotomy. Tingley and MacDougal (1977) have described two aircrew with symptoms not dissimilar to those of pressure vertigo, who were found to have a small defect in the round window membrane when this structure was visualized.

**Motion sickness**

Motion sickness, or kinetosis, is a syndrome characterized primarily by nausea, vomiting, pallor and cold sweating, which is induced when an individual is exposed to certain types of real or apparent motion stimuli. Motion sickness is a generic term which embraces sea-sickness, air-sickness, car-sickness, space-sickness, swing-sickness, simulator-sickness and so on, the name identifying the provocative environment or vehicle. Yet despite this diversity of causal stimuli, the responses of the afflicted individual are essentially the same and have a common aetiology. Motion sickness is not a pathological condition but is the normal response of an individual, with an intact vestibular system, to motion stimuli with which he is unfamiliar and to which he is consequently unadapted; only those individuals without labyrinthine function are truly immune.

However, it should be pointed out that the signs and symptoms of the motion-sickness syndrome are a common feature of organic disease of the vestibular sensory system, in particular those conditions in which there is a sudden and asymmetrical modification of the activity of the sensory receptors of the vestibular apparatus. Although the primary cause of the malaise engendered by motion stimuli and by vestibular dysfunction may differ, the basic
aetiology is the same, as is the process of adaptation to the altered sensory information and the therapeutic benefit of certain drugs.

**Clinical features**

The development of the motion-sickness syndrome typically follows an orderly sequence, the time-scale being determined by the intensity of the provocative motion and susceptibility of the individual. There are, however, considerable individual differences in susceptibility as there are in the incidence and order of occurrence of particular signs and symptoms. The earliest symptom is commonly a sensation of epigastric discomfort, best described as 'stomach awareness'. With continued exposure, nausea increases in intensity and the cardinal autonomic signs appear, namely pallor and sweating. Vasoconstriction is most noticeable in the face, particularly about the mouth, while sudomotor activity is usually confined to those areas of skin where thermal rather than emotive sweating occurs. There is frequently a feeling of bodily warmth and the afflicted individual seeks cool air to obtain symptomatic relief, although this is short-lived. Associated, but more variable, early signs and symptoms are increased salivation, eructation and flatulence, headache, and an ill-defined dizziness. There may be an alteration in the pattern of respiration, with sighing and yawning, which may lead to hyperventilation, particularly in those who are anxious about their disability or their safety in a hostile motion environment (for example, storm conditions at sea or severe turbulence when flying).

The aforementioned signs and symptoms develop relatively slowly, but with continued exposure to the provocative motion there is commonly a sudden intensification of malaise which culminates in vomiting or retching. This is usually followed by a temporary amelioration of symptoms before the subject's condition again deteriorates and emesis ensues. This cyclical pattern of recurrent vomiting, with waxing and waning symptoms, may continue for several days if there is no escape from the provocative motion environment. Those so afflicted can be severely anorexic, depressed and apathetic, incapable of carrying out allotted duties or even caring for their own safety. Their debility may be further compounded by dehydration and a disturbance of electrolyte balance brought about by repeated vomiting (see Money, 1970; Reason and Brand, 1975; or Benson, 1984, for a more detailed review of the clinical features of motion sickness).

In those situations where there is continued exposure to provocative motion, as aboard ship in storm conditions, most individuals exhibit a progressive reduction in the severity of symptoms as they adapt to the motion. The time course of this adaptation is variable, but typically it takes 2-4 days before a significant level of protective adaptation is achieved and the signs and symptoms of motion sickness are dispelled.

In conventional aircraft, prolonged exposure to provocative motion is, these days, relatively uncommon and flights are rarely longer than 10-12 hours. The situation is different in spaceflight where the astronaut is in an abnormal force environment (that is null gravity or weightlessness) for many days or even months. The natural history of space-sickness is in most respects similar to that of terrestrial motion sickness, as described previously (Benson, 1977; Homick et al, 1983). The important difference is not in the signs and symptoms, but rather in the nature of the provocative stimulus. In conventional (terrestrial) motion sickness, it is the complex motion of the vehicle imposed on the person within that induces malaise,
whereas in space-flight, it is the movement of the astronaut within the vehicle that is the provocative factor. The time-scale of adaptation to space-sickness is similar to that described for sea-sickness. Most astronauts who have suffered from space-sickness have been able to move about and make rapid head movements without discomfort after 3 days in weightlessness, and all but one have been symptom-free by the sixth day of space-flight.

Adaptation to an atypical motion environment involves a modification in the way sensory information is processed by the central nervous system and establishment of new motor patterns that are beneficial to the individual. However, on return to a normal (that is a stable 1 g) environment, these sensory and motor patterns are no longer appropriate and can cause perceptual and equilibratory disturbances until readaptation has taken place. The mal de debarquement that sailors experience on return to land after a voyage of sufficient duration for them to adapt to the motion of the vessel, is but one example of this phenomenon. Likewise, astronauts on their return to earth have reported vertiginous sensations on making head movements, and they have an impairment of postural equilibrium that is present for many days, or even weeks, after a long flight.

**Aetiology**

The vestibular system has a significant role in the genesis of motion sickness because the human being, like other susceptible animals, does not suffer from motion sickness unless possessing a functional labyrinth (James, 1882). The knowledge that the absence of vestibular function afforded protection against the disability led to the hypothesis that motion sickness was caused by vestibular overstimulation, but this concept is untenable. Quite strong and unfamiliar motion stimuli, such as repeated stops of a rotating chair or the cyclical oscillation experienced on horseback, do not readily induce sickness, whereas much weaker stimuli, such as the cross-coupled (Coriolis) stimulation of the semicircular canals (produced by head movement while rotating on a turntable) can be highly provocative. Furthermore, the vestibular overstimulation hypothesis does not account for the visually induced forms of motion sickness (for example stimulator- or Cinerama-sickness), neither does it attempt to explain adaptation and readaptation (mal de debarquement) phenomena.

The alternative and more acceptable explanation is that the essential cause of motion sickness is the presence of sensory information about bodily motion which is at variance with inputs that, from past experience, the central nervous system would 'expect' to receive (Reason, 1970). Central to the sensory conflict or neural mismatch hypothesis is the existence, within the central nervous system, of a model of afferent and efferent activity associated with body movement that is derived through daily experience of the process of volitional control of body movement and maintenance of postural equilibrium. In normal locomotor activity, disturbances of body movement, such as when one accidently trips, are typically brief and the mismatch between actual and expected sensory inputs from the body's motion detectors is employed to initiate corrective motor responses. However, when there is a sustained change in the sensory input - as occurs, for example, in atypical motion environments or when there is vestibular disease - then the presence of the mismatch, between actual and expected sensory inputs, indicates to the central nervous system that the internal model is no longer appropriate. The process of adaptation thus involves the modification or rearrangement of the internal model so that it corresponds more closely with the contemporary sensory afference, and the mismatch signal is reduced to an acceptable level.
An essential feature of the neural mismatch hypothesis is that the presence of a sustained mismatch signal has two effects: one, it causes a rearrangement of the internal model; and two, it evokes the sequence of neural responses that constitute the motion-sickness syndrome. There is clearly benefit to the organism to be derived from modifying sensory and motor responses, for this allows it to function more effectively in a novel environment. The question, however, of whether motion sickness has survival value, or whether it is just a design defect that has only recently (in an evolutionary time-scale) become apparent with the use of mechanical aids to transportation, is a matter for debate (Oman, 1980).

Motion of the body is detected principally by the eyes and the vestibular apparatus, although changes in the body's orientation to gravity and imposed linear accelerations are also transduced by mechanoreceptors in the skin, muscles, capsules of joints and supporting tissues, which may be considered to act synergistically with the otolith organs. It is postulated that within the central nervous system there is a neural centre that acts as a comparator of the signals from the receptors with those from the internal model that stores the signature of 'expected' signals. The output of this comparator is the mismatch signal that, on the one hand, is responsible for modifying the internal model and, on the other, for activating the neural structures mediating the signs and symptoms of motion sickness. How this activation is achieved, that is whether by purely neuronal or whether by neurohumoral mechanisms, has yet to be determined. However, in the heuristic model, the presence of a leaky integrator serves to explain the slow development of symptoms following exposure to provocative motion. In addition, it is necessary to postulate the existence of a threshold function in the system, in order to account for the development of protective adaptation without induction of motion sickness and for the large intersubject differences in susceptibility.

*Features of provocative stimuli*

Two categories of motion cue mismatch can be identified, according to the sensory systems involved: one is a visual-vestibular mismatch, the other a semicircular canal-otolith (or intravestibular) mismatch (Reason, 1970). Within each category, the nature of the mismatch may be further subdivided by identifying those situations in which both sensory systems simultaneously provide discordant cues (type 1 mismatch), and those in which one sensory system signals motion in the absence of the expected signal in the other modality (type 2 mismatch). Examples of the different types of mismatch causing motion sickness in aerospace and maritime environments are presented in Table 7.1.

Intravestibular mismatch of one type or another is the most frequent cause of motion sickness. For example, when a head movement is made in an aircraft which is turning, both the semicircular canals and the otoliths can provide erroneous and incompatible signals which are likely to differ substantially from those generated by the same head movement in a norma, stable, 1 g environment. Likewise, in the weightless environment of space flight, head movements, particularly in pitch and roll (that is the sagittal and coronal planes) are provocative because the canals correctly signal the angular movement, but the otoliths fail to provide information about head orientation (as they do on earth) or are stimulated atypically by the linear accelerations produced by the head movement.

Another potent cause of motion sickness is the changing linear accelerations to which those aboard an aircraft flying through turbulent air or a ship in rough seas are exposed. Such
motion is provocative because it is the otoliths (and other gravireceptors) that are stimulated in the absence of the expected signals from the semicircular canals. However, it is worthy of note that the incidence of sickness bears an inverse relationship to the frequency of linear oscillatory motion; stimuli at frequencies above 0.5 Hz rarely cause motion sickness but, as the frequency decreases, susceptibility rapidly increases to reach a peak at 0.1-0.2 Hz (O’Hanlon and McCauley, 1974).

Table 7.1. Identification of type of motion cue mismatch in aviation and marine environments where motion sickness is provoked

<table>
<thead>
<tr>
<th>Category of motion cue mismatch</th>
<th>Visual (A) - vestibular (B)</th>
<th>Canal (A) - otolith (B)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type 1 A and B simultaneously signal contradictory information</td>
<td>(a) Looking from side or rear window of aircraft (b) Inspection through binoculars of ground or aerial targets from moving aircraft (c) Watching waves from side of ship</td>
<td>(a) Making head movement while rotating (cross-coupled or Coriolis stimulation) (b) Making head movement in abnormal force environment which may be stable (eg, hyper- or hypogravity) or fluctuating</td>
</tr>
<tr>
<td>Type 2(a) A signals without expected B signal</td>
<td>(a) 'Simulator-sickness'. Piloting of fixed base simulator with moving external visual display (b) Cinerama-sickness</td>
<td>(a) Making head movement in weightless environment (space-sickness) (b) Pressure (alternobaric) vertigo (c) Caloric stimulation of semicircular canals</td>
</tr>
<tr>
<td>Type 2(b) B signals without expected A signal</td>
<td>(a) Looking inside aircraft or ship when exposed to motion</td>
<td>(a) Low frequency (&lt;0.5 Hz) linear oscillation (b) Rotation about non-vertical axis.</td>
</tr>
</tbody>
</table>

Space-sickness may be attributed to a type 2a mismatch in which the canals are stimulated without the expected otolithic signals. This category of mismatch can be identified as the cause of the motion-sickness syndrome, but without a motion stimulus, that is commonly associated with the atypical stimulation of the semicircular canal receptors by physical agents or disease processes. Both aviators and divers may experience such symptoms in association with pressure (alternobaric) vertigo (see above). Sickness accompanying caloric stimulation is seen more frequently in the clinic than elsewhere, but asymmetrical thermal stimulation of the vestibular apparatus does occasionally occur in divers and may engender nausea as well as disorientation.

Motion sickness can be induced in the absence of vision, but in many provocative environments the visual stimulus contributes to the sensory conflict. Furthermore, there are
circumstances, such as certain aircraft simulators or cinematic displays, in which the dynamic visual cues, in the absence of motion of the observer, cause sickness (that is a type 2a visual-vestibular mismatch); but more commonly, visual information is in conflict with information about whole-body motion provided by vestibular and other mechanoreceptors. A person who is within the cabin of an aircraft or boat has no visual information about his motion in space even though he may be receiving complex vestibular cues about the motion of the vehicle (type 2b mismatch). Conversely, when he is on deck or looking out of the aircraft, visual motion cues can be in accord with inertial cues if a stable reference, such as the horizon, is available, and this can be of benefit in reducing the incidence of sickness. However, if the object of regard has real or apparent motion, as, for example, when looking at waves close to the ship or when using binoculars to observe objects on the ground from a helicopter, then the visual motion cues contribute so sensory conflict (type 1 mismatch) and they increase the probability of the observer becoming motion sick.

Neural centres and pathways involved in motion sickness

Neural mismatch is a useful concept with which to collate aetiological factors of motion sickness, but it is now necessary to try to give neurophysiological and neuroanatomical substance to the theory. Regrettably, the picture is far from complete, although certain elements are reasonably well understood as a result of experimental work on animals. It is well established that the vestibular apparatus and the vestibular cerebellum (uvula and nodulus) are essential for the development of the motion-sickness syndrome and, by inference, the integrity of the vestibular nuclei is also mandatory (Money, 1970). The activity of the vestibular nuclei is influenced not only by the vestibular input but also by visual, somatosensory and cerebellar afferents, so the convergence necessary for the comparator to function can be identified at this level. However, it is perhaps more likely that the vestibular cerebellum functions both as a comparator and as a neural store, and that it controls and mediates the process of adaptation.

The nature of the 'mismatch signal' and the means by which it initiates the sensory and autonomic responses of the motion-sickness syndrome are even more speculative. The signal acts, whether by neural or by humoral mechanisms, through centres in the area postrema of the medulla, close to the chemoreceptive trigger zone, and also through the vomiting centre, to initiate the integrated motor response of vomiting (Borison, 1985). Modification of the activity of hypothalamic nuclei is reflected by the increased secretion of anterior pituitary (antidiuretic) hormone, and there is also increased secretion of other pituitary hormones, notably prolactin, growth and adrenocorticotropic hormones. The secretion of antidiuretic hormone parallels the development of the motion-sickness syndrome and is responsible for the oliguria that is a consistent finding in those suffering from motion sickness (Eversmann et al, 1977). Yet, neither hypophysectomy nor partial destruction of the hypothalamus prevents the development of motion sickness in dogs. Indeed, decerebrate dogs and, anecdotally, decerebrate humans are not immune.

Incidence of motion sickness and factors influencing susceptibility

There are very considerable intersubject differences in susceptibility to motion sickness, but provided that the motion is of sufficient intensity and duration, only those without a functioning vestibular system will not develop symptoms. The incidence of sickness
in a particular motion environment is determined by, on the one hand, the physical characteristic (for example, intensity, frequency, duration) of motion, and, on the other hand, by the intrinsic susceptibility of the person exposed to the motion stimulus and the nature of the task he or she has to perform.

In large civil transport aircraft, the incidence of air-sickness is a fraction of 1% of passengers, although in smaller aircraft of 'feeder' airlines, which fly at lower altitudes with greater exposure of the passengers to gusts and turbulence, the incidence can be of the order of 5-10%. In military aviation, and in particular in high performance training and combat aircraft, 50-60% of the student pilots and navigators experience sickness at some time during training and in about 5% it is of sufficient severity and frequency to lead to the withdrawal of the student from flying training. The severe turbulence to which the crew of 'hurricane penetration' flights are exposed causes symptoms in 90% of experienced flight personnel and all air-crew who have not previously flown in such conditions are affected (Benson, 1984).

Space-motion sickness of varying severity affects approximately 50% of astronauts. Perhaps somewhat surprisingly, susceptibility to space-sickness does not appear to correlate with the individual's susceptibility to sickness induced by provocative motion on earth (Reschke et al, 1984).

Sea-sickness, apart from its greater antiquity, can have a numerically higher incidence than that of air- or space-sickness, for, in general, the motion is more severe and more prolonged than in other modes of transportation. The severity of the motion stimulus is determined by the sea state and vessel size. Thus in an inflatable life raft in a rough sea, all but the very resistant succumb within a few hours, while aboard a Royal Navy frigate in a similar sea state, the incidence of sickness was of the order of 25%. A survey of Royal Navy sailors (Pethybridge, Davies and Walters, 1978) revealed that 70% admitted to having suffered from sea-sickness during their service afloat and 49% had been sea-sick in the preceding year.

In a particular individual, motion sickness may be more readily evoked by one type of provocative motion than another. Nevertheless, there is a correlation, if not a highly significant one, between susceptibility to space-sickness would appear to be an exception to this relationship. The intensity, character and duration of the motion stimulus is the principal determinant of whether a person develops sickness, but a number of factors concerning the physical and mental constitution of the individual have been identified as influencing susceptibility (Reason and Brand, 1975). It is well established that susceptibility changes with age. Motion sickness is rare below the age of 2 years, but with maturation, tolerance decreases rapidly and susceptibility is at a peak between the ages of 3 and 12 years. Over the following decade, susceptibility decreases quite markedly and this decrease continues, of more slowly, with increasing age. The increase in tolerance has been documented for both air-sickness and sea-sickness, although the elderly are not immune. A recent survey of sickness aboard a Channel Islands ferry revealed that 22% of those suffering from sea-sickness were over the age of 59 years (Lawther and Griffin, 1981).

Females are more susceptible to motion sickness than males of the same age (Reason, 1967). The reason for this sex difference, which has long been recognized and which applies to both children and adults, is not understood. It is most likely to be a consequence of
hormonal factors, as in women the incidence of motion sickness reaches a peak during menstruation and susceptibility increases during pregnancy.

Nausea and vomiting are not uncommon symptoms of fear and anxiety, hence it is commonly assumed that anxiety increases susceptibility to motion sickness. Positive but weak correlations have been established between psychometric measures of neuroticism and susceptibility, which show that extroverts have a higher tolerance and adapt more rapidly than introverts. Significant correlations have also been demonstrated between susceptibility and performance of the rod and frame test, with 'field dependent' subjects having a higher tolerance than those who were 'field independent' (Barrett and Thornton, 1968). However, these dimensions of personality are not sufficient to explain the large differences in susceptibility that exist in a group of men or women of similar age and exposure to motion. Studies carried out by Reason (1970) suggest that these differences are attributable to other constitutional factors relating to the way the individual transduces sensory signals (receptivity), how he or she adapts to motion stimuli (adaptability) and to the ability to retain protective adaptation (retentivity). Thus a person who has low receptivity, high adaptability and good retentivity has a high tolerance to provocative motion, whereas the highly susceptible individual has high receptivity, adapts slowly and has poor retention of the little protective adaptation acquired.

Although fear and anxiety are considered to be of only secondary importance in the aetiology of true motion sickness, it must be acknowledged that the motion-sickness syndrome can be a conditioned response or the manifestation of a phobic neurotic reaction. The student pilot whose symptoms become progressively more severe on successive flights, or the passenger who is sick on stepping aboard a boat, are examples of those conditions in which the mind, rather than the motion, is the cause of the malaise.

**Prevention and treatment**

**Behavioural measures**

Motion sickness can be regarded as a self-inflicted condition, for it is effectively prevented by avoidance of exposure to provocative motion. If so wished, people need not travel in aircraft, ships, space vehicles etc, although few in the modern world could accept such a restriction on their mobility. For those who do find themselves exposed to motion stimuli that are likely to induce sickness, there are a number of simple measures that are of benefit in preventing, or at least delaying, the onset of symptoms. These may be summarized as follows:

1. occupy a position aboard the ship or aircraft close to its centre of gravity in order to minimize the intensity of motion stimuli

2. minimize unnecessary head movements; this is facilitated by the provision of head support and good body restraint, and may be further aided by a reclined or supine position

3. take up a position in which there is a good view forward of a stable external visual reference, such as the horizon when aboard an aircraft or ship
(4) if deprived of an external visual reference, close the eyes to reduce visual-vestibular conflict

(5) be involved in a task which occupies the mind and minimizes introspection; optimally, be in control of the vehicle; pilots rarely suffer from air-sickness except when flying as passengers.

These measures can be of immediate value to those who find themselves exposed to provocative motion, but in the long term, it is adaptation which is the most powerful prophylactic. Adaptation is 'nature's own cure' and is the preferred method of preventing, or at least reducing susceptibility to, motion sickness. It is of special importance to air-crew who, in general, should not fly when under the influence of anti-motion sickness drugs. The basic principle governing the acquisition and maintenance of protective adaptation is that air-crew should be introduced gradually to the provocative motions of the aircraft, and that adaptation, once achieved, should be maintained by regular and repeated exposures. Intersubject differences in receptivity, adaptability and retentivity do, however, imply that adaptation schedules must be tailored to the individual, and must preclude definition of the interval between exposures. Some air-crew are troubled by air-sickness if 2-3 days elapse between flights; other, with better retentivity, can spend several weeks on the ground without any increase in susceptibility.

There is, unfortunately, a small percentage of air-crew who do not develop sufficient adaptation during the course of their normal flying duties and who continue to suffer from air-sickness. In a number of studies (Dobie, 1974; Cramer, Graybiel and Oosterveld, 1976; Stott and Bagshaw, 1984), it has been shown that the majority of those who fall into this category can be helped by ground-based training which, in general, involves graded incremental exposure to cross-coupled (Coriolis) and other provocative stimuli. This desensitization therapy may be coupled with biofeedback training aimed at teaching the subject how to control autonomic responses (Levy, Jones and Carlson, 1981), although this procedure does not appear to have yielded better results than were obtained by a more mechanistic approach to therapy. The procedure currently employed by the Royal Air Force has allowed 80% of the air-crew, referred because of intractable air-sickness, to return to normal flying duties without disability from air-sickness (Bagshaw and Sott, 1985).

**Drugs**

Many studies, both in the laboratory and in the field, have demonstrated that certain drugs can reduce the incidence of motion sickness in a given population at risk (Graybiel et al, 1975). A large number of drugs have been studied, but none can afford complete protection. Of the relatively few drugs with proven prophylactic potency, all have side-effects, commonly that of sedation, which limit the usage of these drugs by those in whom the impairment of skilled performance would jeopardize safety. There is a place, however, for the administration of anti-motion-sickness drugs to student air-crew, particularly in the early stages of flying training, but under no circumstances should drugs be taken by a pilot when he flies solo. Such a restriction does not apply to the use of drugs by passengers to alleviate air-or sea-sickness. Nevertheless, where personnel are required to work at peak efficiency, the possible decrement in performance as a result of sickness must be balanced against that produced by medication.
The choice of drug is, in part, dependent upon the duration of exposure to provocative motion and, in part, upon individual differences in effectiveness, and upon the incidence of side-effects. The pharmacokinetics of drugs of proven efficacy differ considerably. A single oral dose of 0.3-0.6 mg hyoscine hydrobromide acts in 30-60 minutes and provides protection for about 4 hours, whereas promethazine hydrochloride (25 mg) and meclozine hydrochloride (50 mg) take 1-2 hours to act and are effective for 12 hours or longer. The other useful drugs, dimenhydrinate (50 mg), cyclizine hydrochloride (50 mg) and cinnarizine (30 mg), are absorbed at about the same rate as promethazine, but their duration of action is shorter (around 6 hours). Thus for short exposure, as in training flights, hyoscine is the drug of choice unless its side-effects - sedation, blurring of vision and dry mouth - are particularly troublesome. For more protracted exposure, one of the longer-acting drugs should be given, with repeated dosage if indicated. The repeated administration of oral hyoscine is not recommended because the side-effects are cumulative. However, with the advent of the Transdermal Therapeutic System (Ciba-Geigy), an adhesive 'patch', placed behind the ear can provide a loading dose of 200 microg of hyoscine and controlled release at 10 microg/h for up to 48 hours. Although a therapeutic efficacy comparable to oral drugs has been claimed (Graybiel, Cramer and Wood, 1981), more recent studies have indicated greater variability between subjects in the protection afforded (Homick et al, 1983).

The prophylactic potency of hyoscine or one of the antihistamines has been shown to be enhanced when the drug is administered in conjunction with either 5 mg d-amphetamine sulphate or 25 mg ephedrine sulphate. The combination has the further advantage that sedation is less than when the anti-motion-sickness drug is given alone.

Most of drugs, detailed above, may be given by intramuscular injection, promethazine hydrochloride (25 mg) being preferred when the accompanying and relatively long-lasting sedation is acceptable, or even, as in passengers aboard ship, desirable.

Spatial disorientation

Spatial disorientation is a term used to describe a variety of incidents in which the aviator or diver fails to sense correctly his position, motion or attitude with respect to a fixed reference, such as the gravitational vertical or the surface of the earth or sea. The erroneous or illusory perception that characterizes a disorientation incident may, on the one hand, be no more than a trivial and transient distraction, but, on the other, it can jeopardize safety and lead to loss of life. In aviation, spatial disorientation of the pilot has long been recognized as a cause of aircraft accidents, but despite the benefits afforded by modern flight instruments, accidents still occur. In military aviation, spatial disorientation is a primary or contributory cause of some 10% of accidents, whereas in private flying the incidence is even higher (25% in the USA). In diving, as in flying, it is the inexperienced divers who are most at risk, for they are the more likely to be alarmed by unexpected and bizarre sensations and, as a result, may fail to carry out those basic procedures, such as ascent to the surface, that would ensure their safety.

Aetiology

Man's ability to determine his spatial orientation is dependent upon information provided by the eyes, the vestibular apparatus and other mechanoreceptors stimulated by
accelerations acting on his body. Although visual information is of primary importance, on the ground man still maintain his postural equilibrium and spatial orientation during normal locomotor activities, even when deprived of vision. The non-visual receptors, and their associated central sensory processing, are functionally adapted to transduce and perceive the motion stimuli experienced in everyday life on the surface of the earth, where the sustained force of gravity (indistinguishable from linear acceleration) is a stable reference of verticality. However, in the aerial or subaquatic environments, the human being is subject to motion which has angular and linear accelerations which may differ substantially in direction, intensity or frequency from those normally experienced on the ground. Consequently, errors in the perception of spatial orientation occur in these atypical environments, primarily because of the functional limitations of the human's sensory mechanisms.

Many different kinds of erroneous sensations and perceptions, falling within the broad definition of spatial disorientation, have been described and there are many different causes (see Benson, 1978; Gillingham and Wolfe, 1985, for more detailed reviews of the topic), but in the context of this chapter discussion will be confined to perceptual errors attributable to vestibular mechanism. Although spatial disorientation can be caused by misinterpretation of visual information, in general, most disorientation incidents and accidents occur when normal visual cues are either absent or deficient, as, for example, when flying in cloud or at night, or when diving in turbid water or an enclosed space.

**Failure to perceive motion and changes in attitude**

In the absence of vision, detection of angular and translational (linear) movements is governed by the transduction of the motion stimulus by the receptors of the semicircular canals and otolith organs. These sense organs are functionally adapted to respond to the motion stimuli that occur during normal locomotor activity, but both in flight, and underwater, the body can be exposed to linear and angular movements which are below the threshold of detection. Thresholds are dependent upon a number of variables, notably, the axis or plane of motion, its frequency spectrum and its duration. Transient movements (that is those taking less than 10 s) are unlikely to be detected if the change in angular velocity is less than about $2^\circ/s$ or the linear acceleration is less than 0.05 m/s$^2$. With more prolonged stimuli (that is greater than 20 s) typical threshold values are 0.3$^\circ/s^2$ for rotational movements and 0.1 m/s$^2$ for linear movements. It is worthy of note that for such sustained stimuli the critical factor is the acceleration of the movement, which, if below threshold, can engender large changes of attitude of which the aviator or diver may be completely unaware in the absence of visual cues for orientation.

**False sensations of angular motion**

In general, misleading sensations of angular motion are a consequence of the dynamic limitations of the semicircular canals. These end-organs correctly transduce the angular velocity of transient (that is less than 10s) head movements, provided that the threshold is exceeded. However, once a steady rate of turn is achieved, there is no longer an adequate stimulus and the deflected cupulae in the plane of the motion slowly return to their neutral position and the associated sensation of turn dies away. Provided there is no appreciable
change in angular velocity, the turn can continue without the aviator having any sensation that the aircraft is in fact turning.

Recovery from the turn is associated with an angular acceleration in the opposite direction to that on entering the turn. The cupulae are deflected from their rest position and will erroneously signal rotation in the opposite direction, at a rate commensurate with the change in velocity that has occurred. This false sensation decays somewhat more quickly than the decay of the correct sensation during the initial phase of the turn, but the presence of inappropriate eye movements induced by the vestibular stimulus can degrade vision and impair the pilot's only reliable source of information. The intensity of these post-rotational effects is a function of the duration of the rotational manoeuvre and of the angular velocity achieved; accordingly, disorientation is most likely to be a problem on recovery from prolonged, high-rate, rolling or spinning manoeuvres.

Cross-coupled or Coriolis stimulation of the semicircular canals occurs whenever an angular movement of the head is made while rotating about another axis. However, disorientating sensations are evoked only when rotation is prolonged and the semicircular canals do not correctly signal the sustained turn. For example, if the pilot were to move his head in pitch at the beginning of a prolonged spin, his sensation of both head and aircraft motion would be correct, but if the same head movement were made some 15-20 s later, the head movement would elicit an entirely illusory sensation of rotation in roll. Head movements made during the recovery phase cause even stronger and more bizarre sensations. As a general rule: a head movement made in one axis, after rotating for some time about an orthogonal axis produces an illusory sensation in the third orthogonal axis.

The semicircular canals may also be stimulated other than by angular accelerations. The effect of pressure change is discussed earlier in this chapter and it will suffice to point out that, on occasion, the onset of vertigo can be quite intense and can be accompanied by nystagmus of sufficient severity to impair vision and degrade the pilot's or the diver's only reliable source of information about his spatial orientation.

Pressure vertigo is a potential cause of spatial disorientation in both aviators and divers, but it is only the latter who are likely to experience vertigo caused by thermal stimuli. As is well known from the caloric test, irrigation of the external canal with water at a temperature more than a few degrees above or below body temperature is an effective stimulus to the semicircular canals, although if both ears are at the same temperature, little nystagmus or vertigo will be evoked. However, problems arise when diving without a hood if there is inequality in the thermal stimulus to each ear. This most commonly occurs when there is wax in the external canal which impedes the heat transfer between the labyrinth and the water (Edmonds, 1973a).

**False sensations of attitude**

In the presence of the constant acceleration of earth's gravity, the otolith organs and the other gravireceptors provide information which allow the orientation of the head and body to be sensed with accuracy. Normally, one is able to distinguish changes of attitude from transient linear accelerations, but perceptual errors occur when the imposed linear acceleration or deceleration is sustained, as in an aircraft when power is applied or dive-brakes are
operated. In such circumstances, the resultant of the imposed acceleration and gravity is accepted as the vertical reference and consequently there is an illusory sensation of a nose-up attitude during acceleration in the line of flight and of a nose-down change of attitude during deceleration. This somatogravic illusion may also be accompanied by a perceived movement of visual objects, particularly isolated lights, which appear to move upwards during acceleration and downwards during deceleration. This visual or oculogravic illusion, in the same way as the somatogravic illusion, takes time to develop and does not reach maximum intensity until the imposed acceleration has been sustained for 40-60 seconds.

The failure to sense accurately the angle of bank during a turn is also attributable to the resultant of the radial and gravitational accelerations being accepted as vertical, for in a coordinated turn the resultant vector remains normal to the aircraft's longitudinal axis and aligned with the long (z) axis of the pilot's head and body.

A false sensation of roll attitude, 'the leans', is one of the commonest illusions experienced by air-crew. It usually occurs on recovery from a prolonged turn or from a previously undetected banked attitude to straight and level flight. In both of these conditions, the aviator feels that he is straight and level before he rolls out. The change in roll attitude is made within a few seconds and is a suprathreshold stimulus to the semicircular canals. This vestibular information is interpreted as roll from the wing-level attitude to one of bank in a direction opposite to that which existed before recovery was initiated. The curious feature of 'the leans' is that it may persist for many minutes even though instruments indicate level flight; yet, characteristically, the illusion disappears as soon as an unambiguous external visual reference is present.

The disorientating sensations produced when head movements are made in a turning aircraft are not solely due to a cross-coupled stimulation of the semicircular canals. The presence of a linear acceleration greater than 10 m/s² (1 g) means that the otoliths will also be stimulated in an atypical manner when the head is moved. The principal effect on moving the head in hypergravity is that of generating an otolithic signal which corresponds to a greater change in attitude, relative to the acceleration vector, than has actually occurred. The semicircular canals and receptors in the neck signal the angular movement of the head with little error, so there is a mismatch which is interpreted as a change of attitude of the aircraft in the plane and direction of the head movement. At higher accelerations (for example 50-60 m/s²), vertigo and sensations of tumbling, as well as an apparent change in attitude, can be evoked by a head movement.

**Prevention**

In general, spatial disorientation, whether in the air or underwater, is not caused by pathology, but is a normal psychophysiological response to abnormal motion stimuli in an environment deficient in reliable visual orientation cues. Thus the aviator's or diver's knowledge about the different causes and manifestations of spatial disorientation, and the conditions in which it is most likely to occur, is of principal importance in the prevention of orientation error accidents. On the one hand, this knowledge will allow him to avoid potentially provocative aerial or underwater environments or, when this is not practicable, to exercise special care in such situations. On the other hand, knowledge that when visual cues are inadequate (for example when flying in cloud or at night), control of the aircraft can be
maintained only by reference to flight instruments, implies that proficiency at instrument flying is mandatory if the aviator is correctly to resolve conflicting sensory cues and maintain proper control of the aircraft.

It is generally agreed that aviators and divers should be advised not to fly or dive when they are suffering from an upper respiratory tract infection, in order to minimize the occurrence of pressure vertigo. In addition, they should be made aware that any impairment of higher mental function, consequent on intoxication by alcohol or other drugs, may not only increase their susceptibility to spatial disorientation but also degrade their ability to resolve perceptual conflict and to make the correct decision in the event of their experiencing any disorientating sensations.