VERTIGO IN DIVING

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With the advent of free diving i.e. diving without the requirement for an attachment to the surface, the importance of vertigo has increased. It is necessary for the diver to have an accurate spatial orientation in respect to both distance and depth, to enable him to return to safety. If the vertigo is of such degree as to prevent the diver compensating, or if it is associated with vomiting, visual disturbances, or unconsciousness, then it is likely to seriously impair his safety. Under normal conditions, spatial orientation depends mainly on information from three systems viz. visual, proprioceptive and vestibular. It is likely that there will be a severe interference in the visual cues when diving under certain adverse circumstances, e.g. in murky water or at night; and proprioceptive information is seriously distorted by zero gravity when the body is immersed in water and becomes neutrally buoyant. In these circumstances, extraordinary dependence may be placed upon the vestibular system, which then has an increased importance. Certain practical cues to orientation are readily available to the experienced diver: exhaust bubbles will float to the surface; the supply and exhalation of air is expedited when parts of the equipment are in certain relationships to the surface; negative buoyancy objects e.g. weight belt, pressure gauge etc., still obey gravity and fall downwards; gas spaces within the body expand during ascent and contract during descent; and when the diver is immobile, his legs tend to sink and his chest to rise. All these cues are of value in the alert experienced and composed diver, but are virtually ignored by the trainee diver during a panic situation.

Because of the paucity of information regarding vertigo in diving, it has been necessary to draw some parallels from the literature on caisson workers and aircrew. Although occasional articles have been presented on this topic, none seems to include specific details of the cases, or a comprehensive discussion of the differential diagnosis to be considered.
SUMMARY OF THE LITERATURE

Sir Leonard Hill (12) described two large series of decompression sickness in caisson workers, in which specific reference to vertigo was made, viz. Heller et al and Keays. Heller et al gave an incidence of vertigo in 1.3% of their cases. They stated there were two types of vertigo in caisson workers: one presenting with temporary deafness and vertigo, lasting for some days and due to unequalisation of pressure on either side of the tympanic membrane during compression (middle ear barotrauma), and one with Ménière's complex of vertigo, vomiting and deafness, which could persist indefinitely and was caused by air bubbles in the cochlear or vestibular areas or their cerebral connections (decompression sickness). The clinical differentiation was clear as barotrauma was experienced during compression and decompression sickness occurred during or subsequent to decompression. Keays, in his series of 3,692 cases of decompression sickness, gave an incidence of vertigo of 5.33%, and attributed the cause to air bubbles in the labyrinth. Almour (1); when discussing the occupational diseases of caisson workers, stated that the Ménière symptom complex may be due either to a haemorrhage within the labyrinthine system, or the liberation of excess nitrogen, during decompression sickness. He also stated that vertigo could occasionally be experienced during compression. In a more recent review of the neurological damage following decompression of caisson workers, Rózsahegyi (19) also referred to a Ménière type syndrome with vertigo, but stated that on examination this was found to be a central lesion, differentiating it from the true Ménière's syndrome of labyrinthine origin. Perusal of the panel discussion which followed this report leaves some doubt as to whether the author was referring also to cases which presented during compression, and whether these cases were related to deafness. In the larger series of decompression sickness in divers, vertigo was reported in 4.3% by Slark (21) and 8.5% by Rivera (17).

Harris (11) reported 9 cases of sudden deafness after diving to depths in excess of 300 feet on helium/oxygen mixtures, and stated that two regained their hearing on being recompressed (strongly supporting the diagnosis of decompression sickness in these two). Three noted improvement in hearing over the subsequent four weeks, and the others were permanently deaf.
deaf. In the seven cases which did not show a therapeutic effect on recompression, there was insufficient clinical information available to assist in diagnosis. Of interest is that none of the cases had vertigo reported during or subsequent to the illness—thus this series cannot be used to support the widespread belief of decompression illness of divers affecting vestibular function. Recent reports from Royal Naval Physiological Laboratories (20) do give isolated case histories suggestive of vestibular dysfunction while decompressing from deep dives, using helium. One diver had permanent cochlear and vestibular damage, one had permanent vestibular damage and two received effective treatment with recompression therapy, although information regarding the symptomatology of these two is not available.

Isolated case histories of divers with vertigo have been reported. Fields (9) reported four cases of vertigo, of a transitory nature, which disappeared shortly after surfacing. Two had a plug of cerumen noted on otoscopic examination, although no aetiological relationship was suggested. It was postulated that the vertigo was nothing more than an unusual displacement of the stapes in the oval window, giving temporary vertiginous symptoms. Jones' (14) observations on fliers have demonstrated the production of transient vertigo when they performed the Valsalva manoeuvre. He coined the term "pressure vertigo" to describe these cases, and attributed the cause to eddy currents passing over the utricular and saccular maculae and the ampullary cupolae, caused by a flow of endolymph as the stapes moves against the oval window. Rawlins, quoted by Cole and Knight (5), described 4 cases of deafness in divers. In one of these cases there was evidence of a bilateral reversed ear syndrome from wearing a C type hood, resulting in a perceptive hearing loss, tinnitus and vertigo. Caloric tests suggested a left canal paresis, with a right directional preponderance, and audiograms showed bilateral hearing loss. Rawlins' other case with vertigo was attributed to decompression sickness presenting with a total loss of hearing on one side and caloric responses much diminished on that side. Rowe (18) stated that vertigo occurred in divers, and that the commonest cause was caloric stimulation. He gave no reasons for this statement, but did report one case of vertigo. McFie (16) described: one case of transient vertigo with a high frequency hearing loss, attributed to a forceful Valsalva manoeuvre; one case with high frequency hearing loss and abnormal caloric tests; and another with vertigo, a hypoactive caloric test, and normal hearing. All three of McFie's cases appear to be related to middle ear barotrauma and a difficulty in performing the Valsalva manoeuvre. In the last case the vertigo
lasted for approximately 1 week, and the caloric tests showed no improvement. Cook (6) recorded one case of vertigo with hearing loss, persisting for 2 or 3 days, and thought to be related to middle ear barotrauma. He attributed inner ear changes to an increase in pressure with subluxation of the foot plate of the stapes. Edmonds (1970)2 reported 5 cases of cochlear damage due to barotrauma. The divers were unable to equalise the pressure in their middle ear with that of the ambient pressure. This was countered by forceful Valsalva manoeuvres, and resulted in permanent changes involving the inner ear. Although this study was specifically designed to demonstrate the permanent neurosensory hearing loss due to middle ear barotrauma of descent with a forceful Valsalva manoeuvre, two of these cases also had vertigo, and the terminology used to describe these cases was "Inner ear barotrauma". The clinical histories of such cases were consistent, with the diver describing difficulty in performing the Valsalva during descent, middle ear pain relieved by ascent and aggravated by descent, and the excessive use of inflammatory pressures to achieve effective Valsalvas. This has also been described by Freeman and Edmonds (10).

Three separate surveys have been carried out to ascertain the incidence of vertigo in diving. Coles and Knight stated that 10% of the 30 divers questioned had a history of vertigo associated with performance of the Valsalva manoeuvre. Lundgren (15) demonstrated a history of vertigo in 26% of 354 divers who answered his questionnaire. He noted that most of the subjects had vertigo during or immediately on completing ascent, and that it varied from a few seconds to 15 minutes duration. Many of the divers believed it to be related to a unilateral difficulty in equalising pressures within their middle ears. He coined the term "alternobaric vertigo", analogous to that disorder described in aircrew. In both the above surveys brief mention was made of the possibility of other causes, but without case reports or discussion on the differential diagnosis. Terry and Dennison (23) also carried out a survey, demonstrating that 40.5% of their 37 divers had experienced vertigo. Over half the subjects attributed their vertigo to the water temperature, and these workers believed that the caloric stimulus played an important role in the development of vertigo amongst divers. They did not believe that inequality of the caloric stimulation was of importance. They stated that vertigo was experienced in less than 5% of all dives, and was thus "an infrequent symptom". In this survey there were as many cases of vertigo experienced during descent as during ascent. It was also noted that two subjects experienced vertigo concurrent with the perforation of a tympanic membrane during the dive.
The literature is thus comprised of surveys conducted retrospectively and each highlighting only one aetiological aspect of vertigo with diving, viz. decompression sickness, forceful Valsalva manoeuvres, alternobaric vertigo or caloric stimulation. Other aetiologies are described in isolated case reports in the general medical literature, or from little known and hard to acquire armed services' journals. None have attempted to produce a differential diagnosis of vertigo due to diving or to discuss the clinical features of each cause.
PROPOSED DIFFERENTIAL DIAGNOSIS OF VERTIGO IN DIVING

The following etiological classification of vertigo in diving is proposed, discussed and illustrated with case histories.

Vertigo in Diving - Classification

1. Caloric Stimulation
   a. Tympanic membrane perforation
      (i) middle ear barotrauma of descent ("ear squeeze")
      (ii) underwater shock wave ("underwater blast").
   b. Unilateral external auditory canal obstruction
      (i) cerumen
      (ii) otitis externa.

2. Inner Ear Barotrauma
   a. External ear barotrauma of descent ("reversed ear")
   b. Middle ear barotrauma of descent ("ear squeeze") with forceful Valsalva manoeuvres ("pressure vertigo")
   c. Middle ear barotrauma of ascent ("alternobaric vertigo").

3. Decompression Sickness ("labyrinthine bends", "staggers")

4. Abnormal Gas Pressures
   a. Inert gas narcosis
   b. Oxygen toxicity
   c. Carbon dioxide toxicity
   d. Others.

5. Unequal Vestibular Responses

6. Miscellaneous
   a. Migraine
   b. Sensory deprivation
   c. Idiopathic vertigo of divers, and other causes.
1. CALORIC STIMULATION

When the diver immerses himself in water there is normally a free flow of water into both external auditory canals, at an ambient temperature which may vary from 0° to 20°. The stimulation in each ear is similar, and thus no vertigo would be expected - and nor does it occur in the vast majority of dives. If the stimulus is disproportionately great on one side, vertigo would be expected - with a latency period varying inversely with the degree of inequality. Alternately, even if the stimulus in each ear is equal, an inequality of the vestibular responses could have a similar result.

a. Tympanic Membrane Perforation

This is a common cause of transient, but often severe, vertigo. It is well recognised by most medical officers dealing with diving accidents, and by divers. It has a characteristic symptomatology. The dominant feature is a loud noise associated with a sensation of cold water rushing into the middle ear. Vertigo follows almost immediately and lasts usually for less than a minute. It is believed that the inrush of cold water into the middle ear through the tympanic membrane is the cause of the transient vertigo, and that this small amount of cold water rapidly warms to body temperature, removing the unequal caloric stimulus. On surfacing, the diver often has blood-stained fluid running from the external auditory canal, probably expelled when the gases in the middle ear expand during ascent, and force the blood out through the perforation.

There may be no vertiginous symptoms in those less common cases in which the tympanic membrane ruptures without entry of water into the middle ear cavity - due either to this space being filled with blood, or to the perforation occurring during ascent. These patients usually present when they notice the hissing of gas through the perforation during Valsalvas or ascents. Occasionally these patients present with vertigo when driving their motor vehicles after the dive. Whether this is due to head movements aggravating positional vertigo, or air currents replacing water in producing caloric stimulation, is unknown.

It is customary to advise against diving, flying or performing Valsalva manoeuvres for a month in patients with tympanic membrane perforation. The ear should be kept dry and no local ear drops are required. Some advise the use of

.../systemic
systemic decongestants, but it is our regime to give these only when otherwise indicated. We routinely administer prophylactic antibiotic cover for 4 days. The tympanic membrane often appears healed within a week, but it remains susceptible to a recurrence if diving is then resumed.

There are two major predisposing causes of tympanic membrane perforation, leading to vertigo while diving:

1. **Middle ear barotrauma of descent ("ear squeeze")**

   This is due to the inability to increase pressure in the middle ear cavity, to equal the ambient pressure. The diver is almost always aware of this inequality, and its origin. Under these conditions, most divers experience considerable pain, preventing further descent in the water and causing haemorrhage within the middle ear cavity, around the periphery of the tympanic membrane, Shrapnell's membrane and along the handle of the malleus. It is commonly inferred that perforation is the ultimate damage from not equalising the pressure in the middle ear cavity, and follows the extreme degrees of haemorrhage described by Teed (22) or McFie (16). Most of the perforations seen by this author were not related to these gross haemorrhages in the tympanic membrane. It is possible that perforation competes with middle ear haemorrhages as a pressure equalising process - the former demonstrating tympanic membrane fragility and the latter demonstrating vascular capillary fragility. On otoscopic examination the perforation is often circular and seen either at the site of previous pathology or posterior of the tip of the handle of the malleus. With audiometry, it is often possible to detect a 5-15 decibel loss in the affected ear. This is of the conductive type, and usually returns to normal within a week or two. Caloric tests are contraindicated. As neither the vertigo nor the hearing loss is permanent, it is believed that the inner ear is not seriously damaged in these cases.

Case 1.

This diver had difficulty in clearing his ears during a descent to 30 feet, using a compressed air breathing apparatus. He finally descended to the 30 feet level, and for approximately 1 minute his ears continued to feel very uncomfortable, until he noticed a sudden bang followed by relief of pain and a loud rushing noise, localised in the left ear. He then developed a rotational vertigo, requiring him to clutch the shot rope, as he lost his sense of balance. The vertigo lasted for

.../approximately
approximately 10 seconds and then diminished. He requested permission to surface, and this was achieved without difficulty until he reached the surface, when there was a recurrence of the vertigo for a few seconds. On otoscopic examination there was slight erythema around the handle of the malleus, with a large perforation in the tympanic membrane just posterior to the lower part of the handle of the malleus. The audiogram revealed a temporary 10-15 decibel loss in the left ear throughout the range of hearing tested (500-8000 cps).

(ii) Underwater shock wave ("underwater blast")

This is an easily diagnosed disorder, and is said to have been common when navy divers were subjected to underwater explosions, but not when they faced the direction of the explosion. The blast wave may involve any area which is capable of distortion, and especially if it is associated with a compressible air cavity. The tympanic membrane is so situated, although whether the shock wave moves easily into the external auditory canal, or receives significant interference with deflection waves, is a matter of individual fortune. In these days of sport diving the underwater shock wave causing perforation of the tympanic membrane and vertigo is commonly a result of being "finned". When a diver swims past another diver, considerable pressure waves are felt from the fin ("flipper") movements. If fins are used near a diver's ear, perforation of the tympanic membrane is possible. When these cases were initially encountered, it was decided to perform some manometric testing within a short distance of the fin action commonly employed by divers. This was readily performed and it was ascertained that pressures varying rapidly from +30 to -10 centimetres H₂O were common. The shock wave, which is a water wave, is probably also responsible for the entry of water into the middle ear following the perforation. Although the case reported here had no permanent vestibular or hearing sequelae, it is believed that these could occur in more severe cases.

Case 2.

This diver was just under the surface and preparing to descend when he was passed by another diver. The fin movement of the second diver was very close to the patient's left ear, and as the fin was moved through the water, the patient experienced a sharp pain, followed by a feeling of water rushing...
into the ear and then severe, but transitory, rotatory vertigo. The latter lasted for approximately 20 seconds and then decreased. He was assisted from the water, and was observed to have a small amount of blood trickling from the external canal. On otoscopic examination a tympanic membrane perforation was observed. There were no other signs of the more common appearance associated with aural barotrauma. There was no hearing loss.

Cases 1 and 2 are typical of others seen with perforation of the tympanic membrane.

b. **Unilateral External Auditory Canal Obstruction**

Although far less dramatic than perforation of the tympanic membrane in its effect, it is possible that the free flow of cold water into only one external auditory canal will induce vertigo. This is certainly not always so, and the evidence incriminating it is not incontrovertible. None of the cases attributed to this cause have shown any permanent vestibular effects, and the only effect on audiometry is that due to the canal obstruction. The two causes observed have been cerumen plugs and otitis externa, although others such as exostosis, foreign body, ear plug, etc., may theoretically have this effect.

(1) **Cerumen Plug**

Two of Harris' cases of vertigo with diving showed no abnormality "except the presence of a plug of cerumen". He drew no causative inference from the finding, probably due to its common occurrence in the general population. The same observation has been noted by this author, with the qualification that plugs of cerumen are known to be rare amongst divers. Those who spend much time underwater quickly lose any cerumen accumulation they may have - and all those who commence diving have an otoscopic examination, with syringing of the ears if there is any suggestion of cerumen occupying much of the canal. The reason for ensuring patency of the external ear canal was, originally, only to test the efficiency of the Valsalva and to prevent external ear barotrauma. The finding of cerumen plugs, uncommon in this selected population, was considered of possible import in those with vertigo.
Case 3.

This diver had no difficulty in equalising his ears during a descent to 30 feet. While swimming along an underwater line he felt as if he were rotating to one side around the line. As the line was on the sea bed, he knew that this sensation was incorrect, and he decided to surface. The vertigo, which lasted for some 10 to 20 seconds, did not trouble him during the ascent and he had no further difficulty. On clinical examination there was no abnormality other than the presence of a large plug of hard cerumen noted in the left ear. Prior to removal of the plug it was decided to carry out electroneystagmograms, together with caloric testing, to ascertain whether sufficient water was likely to pass the possible obstruction. The positional electroneystagmogram was normal, but the caloric demonstrated a false picture of a total left canal paresis. The cerumen was removed, but no evidence of external or middle ear barotrauma was noted. It was postulated that the cerumen plug was large enough to obstruct the free flow of water, but not complete enough to prevent some water from equalising the changing pressures within the external ear.

(ii) Otitis externa

Patients with otitis externa should not dive, but they sometimes do. This cause of vertigo has not been reported in the literature on diving medicine, and there is only one such case known to this author. The swelling and congestion of one external auditory canal in this case greatly restricted water entry.

Case 4.

This man developed vertigo at a depth of 25 feet, 15 minutes after he reached the bottom and had been working in the one position. He noted that his environment, including the engineering task he was performing, appeared to be rotating to one side. He then ascended to the surface, without difficulty. A well meaning but injudicious aural investigation was performed prior to a competent medical examination, and the patient was examined with these results at hand. There was a slight degree of hearing loss throughout the whole range of hearing on the left hand side, and electroneystagmography with calories revealed a total left canal paresis. On otological examination the reason for the apparent left canal paresis became obvious, when it was noted that there was a gross otitis externa with congestion and oedema almost totally occluding the external canal. The patient was aware of the condition prior to the
dive, but this did not deter him. Treatment of the otitis externa resulted in a return to normal of the cochlear and vestibular function assessments.
2. INNER EAR BAROTRAUMA

This refers to the tissue damage resulting from distortion of air spaces, when their volumes change in accordance with Boyle's Law. In relationship to the ears, there are 3 clinical types of barotrauma, depending on the site of the damage and whether it is due to contraction (descent) or distension (ascent) of the air spaces.

. External ear barotrauma of descent ("Reversed ear")
. Middle ear barotrauma of descent ("Ear Squeeze")
. Middle ear barotrauma of ascent.

The involvement of the inner ear secondary to barotrauma of the external or middle ear cavities i.e. "inner ear barotrauma", may produce either transitory or permanent damage, and result in hearing loss, vertigo, or both. A common factor in the production of vertigo due to barotrauma is a distension of the middle ear cavity with the associated movement of the ossicle chain. Although this is axiomatic with external ear barotrauma of descent and middle ear barotrauma of ascent, it is also probable in those cases of middle ear barotrauma of descent - when a forceful Valsalva manoeuvre finally succeeds in greatly distending the previously contracted middle ear cavity.

a. External ear barotrauma of descent ("Reversed ear")

This is due to an obstruction preventing water from replacing the contracting air space in the external auditory canal. It is commonly due to the incorrect use of "O type hoods" or ear plugs, although cerumen plugs have also been suggested. Rawlins' case, reported and assessed further by Coles and Knight, clearly demonstrated a significant degree of vertigo with permanent vestibular damage. Other symptoms of this disorder are mild, but examination of the external canal reveals haemorrhages and vesicular formation. The tympanic membrane is normally free of haemorrhages or perforations (Jarrett, ).

.../b. Middle
b. Middle Ear Barotrauma of Descent ("Ear squeeze") with Forceful Valsalva Manoeuvres ("Pressure vertigo")

Both temporary threshold shifts and permanent neurosensory hearing loss have been demonstrated due to this sequence of events. It is especially common in breathhold diving and when diving with an upper respiratory tract infection. Divers who have considerable difficulty in equalising their middle ear pressures may foolishly try to overcome this by applying extreme pressure when performing the Valsalva manoeuvre. This is transmitted to the middle ear when the Eustachian tube does open, and may then cause temporary or permanent damage to the hearing and/or vestibular apparatus i.e. inner ear barotrauma. Cases of hearing loss alone and in combination with vertigo, have been described previously (Edmonds) and those now presented illustrate the extremes of this type of disorder. Case 5 had permanent involvement of cochlear and vestibular involvement; Case 6 had only temporary involvement of vestibular function.

Case 5.

In October 1969, this man was exposed to many ascents and descents, diving for 30 minutes with a 60% O₂ mixture on a rebreathing set, to a depth of 45 feet. He had difficulty in clearing his ears, and needed to perform forceful Valsalva manoeuvres. There was no other difficulty noted during the dive. When he returned to the boat and climbed inboard, he noted that he had become "dizzy". He described this as a spinning sensation, and he was unable to stand. He obtained some relief by sitting and lying down. This disturbance persisted for only 10 minutes or so. He also noted left ear tinnitus, which persisted for some weeks. He stated that the left ear appeared to be the one affected by this tinnitus. Audiograms demonstrated a marked sensorineural hearing loss on the left side, electronystagmograms showed spontaneous nystagmus, and caloric testing verified a peripheral type impairment of vestibular function. Two weeks later the patient still had evidence of vertigo, noted especially when tilting of the head and lasting for a few minutes. He also noted vertigo when lying down, and although he could lie on his right side without any disturbance, it made it impossible for him to lie on his left side. Even at that date there was a rotatory nystagmus noted, and aggravated when he moved his head from the horizontal to the left position. Repeat audiograms and electronystagmograms performed two years later revealed a permanent neurosensory hearing loss and a left canal paresis on electronystagmography with caloric.

.../Case 6
Case 6.

This soldier had been diving without any problem to depths of 60 feet, until the day of his vertigo incident. On that day he had trouble equalising the pressure in both middle ears, but much more so on the right. This occurred at the 12 feet and 20 feet depths. This middle ear squeeze prevented him descending further, and he was sent to the medical department. He noted that he was very unsteady while walking, and that the floor appeared to be moving upwards. When immobile, he felt as if he were falling and this was aggravated by any sudden change in posture. On examination he had Grade II middle ear barotrauma on the left and Grade III on the right (according to McFee's grading). With his eyes closed he fell consistently to the left side. The vertigo diminished in intensity over the next week, and audiograms and vestibular function tests performed at that stage showed some positional spontaneous nystagmus, slight in degree, and not of diagnostic significance. The caloric tests were normal and the audiogram demonstrated a high tone loss in both ears around 4000 cps, but within normal limits and consistent with exposure to gunfire.

c. Middle Ear Barotrauma of Ascent (Alternobaric Vertigo)

One aspect of this disorder has been aptly described by Lundgren. The unequal release of gas from the middle ear cavities during the initial stages of ascent results in a pressure difference between the two middle ears, and probably an unequal stimulus to the vestibular system. During this, the patient may develop a mild vertigo, usually rectified by further ascent, which opens the less patent eustachian tube. When this does occur, the pressures are then both equalised with the ambient pressure, and the stimulus to vertigo ceases. Also, subsequent opening of the tube seems easier. All of Lundgren's cases of middle ear barotrauma of ascent were mild in their symptomatology i.e. the eustachian tube finally opened and thus prevented further aural damage. The equalisation is usually accompanied by an escape of gas bubbles down the eustachian tube, and is felt by the diver. Such is not always the case and there have been instances of progressive pain during ascent with ultimate perforation of the tympanic membrane. The appearance of the tympanic membrane in most cases is quite unlike that of middle ear barotrauma of descent, as there is haemorrhage only around the margin of the tympanic membrane, with virtually no haemorrhagic area around the handle of the malleus.
malleus. Case 8 is a typical case of Lundgren's alternobaric vertigo but Case 7 demonstrates a residual effect - a positional vertigo lasting much longer than Lundgren's maximum of 15 minutes.

Case 7.

This diver had spent 90 minutes at a depth of 20 feet, chiselling a metal chain. He was requested to surface, which he did. Just after he left the bottom, only a few feet and ascending slowly, he felt as if he were spinning around the vertical shot rope, which was moving in an anticlockwise direction. He could not understand what was happening, and assumed that he must have been imagining things. He then looked towards the surface and noticed that it was also spinning with the same circular motion. He stopped his ascent at about the 10 feet mark, and at this stage the vertigo had ceased. He then continued his ascent after a few seconds and had no further trouble. There had been no previous difficulty in equalising the middle ear pressures, although he did note that when he attempted to resume diving after the incident, he had a difficulty in equalising his right ear. That evening he felt he was developing an upper respiratory tract infection, and had one episode of vomiting. He also noticed a pain over his right ear and mastoid region. The following morning, when arising from bed, he had a recurrence of severe vertigo, with all objects again moving in an anticlockwise direction. He obtained some relief with the resumption of a recumbent posture. On examination there was no evidence of aural barotrauma in either ear, his audiogram was within normal limits, but compared to his pre-incident audiogram there was a slight loss of hearing in the right ear, 20 decibels at 4000 cps, and 30 decibels at 8000 cps. The electronystagmogram demonstrated no abnormality.

Case 8.

While testing the feasibility of using an ECG lead for an electronystagmogram, a medical officer was compressed to 180 feet for 5 minutes and then decompressed. He was lying motionless on the floor of the RCC with his eyes shut. Approximately 10 seconds after commencing the ascent, at a depth of 170 feet, he noticed a subjective feeling of an increased pressure build up in one ear, with the other ear clearing spontaneously. Concurrently he noted a severe rotational vertigo lasting between 10 and 15 seconds. This cleared soon after the gas escaped from the blocked eustachian tube. There were no other episodes during the remainder of the ascent, nor during the stay at 180 feet. The detection of the eye movements on the ECG record was fortuitous, and noted quite independently of the subjective report.

.../3. DECOMPRESSION
3. **DECOMPRESSION SICKNESS**

Vertigo has often been reported in association with this condition, but there is considerable doubt about the validity of the diagnosis when, as in some of Rózsahegyi's cases, there are no other manifestations of the disease. Recompression therapy should usually only be considered in those cases who develop this symptomatology after decompression has commenced - or within the 12 hours following it. When auditory involvement does occur in decompression sickness, it tends to be severe, often doing permanent damage if recompression therapy is not given. It is often peripheral in site, although it commonly affects the cochlea and vestibule separately. This dichotomy of symptoms may be due to selection in reporting of cases, or it may reflect involvement of the separate vascular supply. The increased incidence of auditory damage with decompression sickness does seem to have accompanied the advent of deep diving with helium. These manifestations also occur earlier than would be expected on the basis of the traditional decompression theories. It has occurred at depths of: At 490 feet ascending from 800; 340 feet from 400; 230 feet from 600; 1260 feet from 1500.

When vertigo does occur with decompression sickness the immediate complications of drowning, vomiting with dehydration, electrolyte disturbances, and distress in a patient who is otherwise seriously ill, are complemented by other long term sequelae. With such permanent vestibular damage there is a likelihood that the diver may not be able to continue with his occupation, and may be restricted from other occupations such as flying or driving vehicles. If incorrectly diagnosed during decompression (e.g. attributed to seasickness) then further decompression will result in total damage of the vestibular apparatus. Recompression therapy, if promptly instituted, should result in cure. Simple tests of vestibular function can and should be performed under hyperbaric conditions, when doubt exists regarding efficiency of treatment, and to obtain serial recordings. These investigations include electronystagmography and iced water caloric tests, and will be described in a separate report (Blackwood and Edmonds). Vestibular dysfunction and its clinical manifestation of vertigo may occur as an isolated lesion, as in Case 9, or as part of a much wider clinical picture, as in Case 10.

.../Case 9.
Case 9.

This man was a very experienced diver who had no previous history of any difficulty with his ears. He developed severe vertigo at a depth of 140 feet, after he had been incorrectly decompressed from 515 feet. His vestibular function was permanently destroyed on one side and he was unable to drive, fly or dive following this illness. During the decompression, which was continued despite the patient's symptoms of severe vertigo, vomiting and prostration, there were no other manifestations of decompression sickness. Two years later he had positional vertigo of the peripheral type, with total hypofunction of one vestibular apparatus, and a perfectly normal audiogram up to 8000 cps.

Case 10.

This man was a caisson worker who developed joint bends following compressions to 23 psig. He did not inform his supervisors of this, and exposed himself to the same pressure for another 6 hours. Two hours following decompression he developed a severe vertigo and was unable to stand or walk. He then developed retrosternal chest pain, vomiting, severe malaise, headache, skin mottling, paraesthesia over the left side of the body, and pains in both knees and one hip. He was given a therapeutic recompression, with removal of most symptoms but persistence of the vertigo, to a reduced degree. Then followed a recurrence of symptoms, which included pain in the left retromandibular region, hearing loss in the left ear, paraesthesia and sensory disturbances over the left side of the face and body, and he was unable to stand with his eyes closed. He also had loss of motor power on the left side, nystagmus on looking to the left, and positive rhombergism. With the second therapeutic recompression he lost the abnormal sensations on the left side and the pain in the left retromandibular region. He regained motor power and co-ordination. Otological investigations revealed only a right canal paresis, with spontaneous nystagmus demonstrated on the electronystagmogram. There was no evidence of hearing abnormality after the second therapeutic recompression. It was thought that the sensory and motor abnormalities, referred to the left hand side of the body, were indicative of a right sided neurological lesion. The persistent otological abnormalities suggested involvement of the right vestibule or its arterial supply. A subjective left sided deafness had been relieved by the recompression therapy, and was unable to be demonstrated after this therapy. The other symptoms and signs suggested that multiple sites were involved in this case, and that the persistent vestibular abnormality was only one part of the much wider syndrome of decompression sickness.
4. **ABNORMAL GAS PRESSURES**

This field has been virtually untouched by medical practitioners knowledgeable in the field of otology. The difficulty of differentiating vertigo from such terms as dizziness, lightheadedness and disorientation makes any review of the diving literature almost valueless. Each of the conditions described below will seriously interfere with cerebral function and thus a subjective assessment of vertigo is particularly difficult, and objective measurement becomes imperative.

a. **Inert Gas Narcosis**

As divers descend beyond 100 feet while breathing air, they become progressively sedated and narcotic from the influence of nitrogen. The breathing of air at depth produces nitrogen narcosis and "dizziness", described by Bennett. If this dizziness is a genuine vertigo then, like the other symptoms of nitrogen narcosis, it should be quickly corrected by reducing the inert gas pressure i.e. with ascent. Divers often complain of disorientation and unsteadiness, but I can find no clearly described case of rotational vertigo attributable to inert gas narcosis, other than that in the "high pressure nervous syndrome" as described in the RNFL reports of Buhlmann et al (4). In this syndrome vertigo was noted while the subjects were breathing helium and oxygen, at an environmental pressure of 31 ATA. It was brief and had no sequelae.

Electronystagmography performed on subjects exposed to depths of 180 feet in the recompression chamber are described by Blackwood and Edmonds.

Case 11.

This medical officer performed his first dive to a depth of 180 feet, connected to a companion diver by a buddy line. The sea bed was not in sight, but visibility was good. After approximately three minutes, and without any significant change in depth, it became apparent that the buddy line, and the companion diver, seemed to be rotating in a clockwise direction. As both divers were stationary, this was recognised as an illusion. It continued for a minute or two, and with the safety precautions being used, apprehension did not interfere with the clinical observations. Ascent was noted to relieve the vertigo, and descent produced a recurrence. The only other symptom of inert gas narcosis was a mild euphoria, and this was also

.../relieved
relieved with ascent. No vertigo was noted during the descent or at shallower depths. Nor was there any difficulty in performing the Valsalva manoeuvre during descent, or any sensation of eustachian tube blockage during ascent. Subsequent dives did not result in any similar episodes, however this subject has noted a tendency to "lightheadedness" - quite unlike the vertigo described - during recompression chamber dives to 240 feet. There was no abnormality on otological examination following the dive, or subsequently. Tests of hearing and vestibular function remained normal.

b. Oxygen Toxicity

Vertigo is a documented symptom of this disorder when it affects the neurological system i.e. at pressures of 2 ATA or greater. Donald (7) described vertigo as an early symptom in a number of his experiments, in which he exposed volunteers to toxic concentrations of oxygen. He also described vertigo being precipitated during the reduction from high oxygen pressures i.e. an oxygen "off effect", as well as following oxygen convulsions. These situations are only likely when divers use oxygen or rebreathing equipment, and when the safe limits for oxygen pressures or durations are exceeded. Nausea and vomiting are also associated with somewhat lower oxygen pressures, but whether these are related to the vertigo is unknown at this stage. In assessing this possible aetiology, Blackwood and Edmonds exposed subjects to oxygen levels for a brief period (10 minutes at 60 feet depth) and then decompressed them with electronystagmography monitoring throughout. The results did lend support to the concept of oxygen induced vertigo.

c. Carbon Dioxide Toxicity

Disorientation is a characteristic feature of CO₂ toxicity, but vertigo is far less definite. It has been reported, in association with vomiting, by submariners who have become acclimatised to breathing high CO₂ mixtures and then revert to breathing air or oxygen. This became known as the "carbon dioxide off effect". Blackwood and Edmonds failed to verify CO₂ toxicity as a cause of nystagmus or vertigo, but have demonstrated this tendency in some subjects after they cease breathing 35 mm Hg CO₂ and recommence air breathing. A similar state could occur clinically in divers using rebreathing equipment with partially ineffective CO₂ absorbent systems, when they stop...
their high CO₂ production, e.g., if they rest after an energetic swim. Despite the experimental production of CO₂ narcosis in many divers, rotational vertigo was not reported as a symptom in any of these.

d. Other Gases

The effects of hypoxia, hypocapnia and carbon monoxide poisoning may well include vertigo, but they have not been demonstrated in any diving accident case report known to this author.
5. **UNEQUAL VESTIBULAR RESPONSES**

MoFie has suggested that if there is any abnormality of function of one vestibular apparatus, diving should be forbidden, as this would considerably impair the diver's orientation under certain diving conditions.

Vertigo induced by extraordinary stimulation of the vestibular apparatus has already been described under the previous sections. It is possible that given equal stimuli to both vestibules, vertigo could result if there is a relative inequality of the vestibular responses. The stimuli of diving which would usually affect both vestibules equally, include caloric effects, barotrauma, abnormal gas pressures and miscellaneous conditions.

There is only one case known to this author of a patient who had internal ear damage prior to commencing diving, and then developed vertigo. The presence of inner ear damage would normally disqualify a candidate from attempting a diving course. There are other divers who have had a previous diving accident causing inner ear damage and then developed vertigo while diving. Many divers who develop vertigo seem then to be predisposed to its redevelopment, almost irrespective of the aetiology, if they resume diving within a short time. This could result from a temporary vestibular abnormality.

**Case 12.**

This man fired 50 rounds of a .222 rifle without using ear protection. Following this he developed severe tinnitus and noted a partial deafness in the left ear, persisting from that time. Three months later he took up diving, and although he never experienced any difficulty in equalising his middle ear pressures during descent, he consistently noticed vertigo during ascent - on each dive. The vertigo appeared directly proportional to the depth of the dive and the speed of ascent. He finally achieved a depth of 45 feet, but when attempting to ascend 5 feet to swim over a rock, he developed severe vertigo; had to clutch on to one of his companions to steady himself, and was unable to orientate himself or ascend to safety. When he looked at the surface it appeared to be spinning round, and he realised that he was in danger. Using a technique which he had previously found relieved him, he assumed the prone position with his head down and waited a few minutes. He was
then able to ascend very slowly. He developed vertigo during the ascent, and again had to stop and wait. He had blurred vision, a headache and nausea during this episode, but was able to swim to shore before vomiting. After a few moments rest on the beach, he was asymptomatic and was able to walk without assistance.

Audiograms showed the unilateral high frequency dip. On compression and decompression in a chamber, with continuous ENG monitoring, he would develop severe vertigo and nystagmus if he was in the erect position (i.e. the same position as he would normally assume during ascent in water); If he assumed the supine position, this was less noticeable.

In Case 9 (mentioned previously) the diver who had total unilateral vestibular hypofunction, developed a brisk nystagmus on a trial compression with ENG monitor to 30 feet - a year after the incident. He had never had any trouble with equalising his middle ear pressures, yet the pressure changes were now capable of producing this effect.
6. MISCELLANEOUS

a. Migraine

People with a history of this disorder should not dive. It is a well known cause of vertigo, quite unrelated to diving. It is also commonly precipitated by diving—although whether this is due to high oxygen pressures or cold causing vasospasm as a prelude to reactive vasodilation, is not known. In these cases there is no problem of diagnosis as the associated headache has its characteristic clinical features. It often becomes much worse after the diver leaves the water.

b. Sensory Deprivation

This restriction of incoming stimuli is particularly disturbing to novice and amateur divers, especially when night diving or diving in murky water, so that all visual cues are lost. It has been observed that in many of the cases of vertigo in which no definite aetiology has been demonstrated, that complaints are made regarding the poor conditions for visibility. Sensory deprivation has also been mentioned by others as a cause of vertigo underwater, but to the author's knowledge there is no single case report which has been able to demonstrate this cause. It would be difficult to envisage conditions which would be able to be duplicated and prove vertigo from such a cause. It is possible that sensory deprivation will serve to decrease the threshold of vertigo and its demonstrable nystagmus. The effect of sensory deprivation in the production of disorientation, as opposed to vertigo, is unquestioned.

c. Others - Idiopathic Vertigo of Diving

There is no suggestion that the above causes of vertigo in diving are all inclusive. In some cases there are so many possible aetiologies that to select one would infer an extraordinary clinical wisdom (Cases 13 and 14). In other cases, there seem to be no adequate explanations (Case 15).

Case 13.

This man was diving and had reached a depth of approximately 20 feet, but with considerable difficulty in equalising his middle ear pressures. There were extremely poor visual conditions, due to mud sediment following rains.
The incident occurred initially near the sea bed and within a few minutes of descent, and was described as a spinning sensation. He was unable to detect whether he was going up or down in the water. He tried to follow his bubbles and his anchor rope to the surface, and in doing so noticed that while swimming upwards he appeared to be going round and round the rope. When he arrived on the surface he managed to reach his boat, which also appeared to be rotating away from him. This all cleared within 10-15 minutes.

Diagnosis:
Vertigo secondary to (Middle ear barotrauma of descent and/or Valsalva pressure and/or Sensory deprivation and/or (Idiopathic vertigo of divers.

Case 14.

This diver had his first attack of vertigo, in which he noted himself spinning in a clockwise direction around objects, while diving on 100% O₂ at a depth of 15 feet for 12 minutes. On surfacing the vertigo disappeared within seconds. A similar sensation occurred that night when lying in bed, but was abolished by turning on the light. A third episode occurred while performing a night dive, with very poor visibility. It was abolished when he fixed his eyes on to some sea weeds. A similar episode occurred during a daytime dive in very poor visibility, and was abolished when he fixed his eyes onto the shot. A month later he had an attack again while going to sleep. This was abolished by lying on one side, but not the other. There has been no abnormality demonstrated on otoscopic examination during these episodes, but one year subsequent to this he did have aural barotrauma of ascent. Audiograms and electronystagmograms with caloric testing were normal.

Diagnosis:
Vertigo due to (Oxygen toxicity and/or Alternobaric vertigo and/or (Sensory deprivation.
Case 15.

After spending 20 minutes at a depth of 25 feet, swimming horizontally along a jackstay with good visibility, this diver noticed that his vision had become a little blurred, then he developed a retro-orbital headache and nausea. He then became dizzy and this developed into a sensation of turning to the left around the jackstay embedded in the seabed. He was thus aware that his imbalance was an incorrect observation. He ascended without difficulty, other than a slight nausea for approximately an hour, and with no other symptoms. Otoscopic examination was negative. Audiology and vestibular function tests were normal.

Diagnosis: Idiopathic vertigo of divers.

A common syndrome of vertigo underwater, which has not clearly fitted into any of the above classifications, is that experienced by divers who have descended without difficulty, and have reached a level in which they then perform a horizontal swim. We use the term "Idiopathic vertigo of divers". The vertigo usually comes on approximately 5 minutes after the commencement of the dive, and tends to recur when the diver attempts similar dives under similar conditions within the next few weeks. In these cases there is usually no abnormality evident on the formal testing of hearing or vestibular function, and usually no abnormality on otoscopic examination. It is too easy to postulate an inequality of caloric stimulation, a mild degree of barotrauma, variable sensory deprivations, unequal vestibular responses etc. These must remain conjectures without more comprehensive vestibular function assessments than are currently available, or some other evidence. Although it would sometimes be possible to incriminate inert gas narcosis, many of these cases develop vertigo in depths considerably less than 100 feet. It is possible that this particular vertigo results from two factors viz. unequal vestibular responses and caloric stimulation. In support of the latter is the time delay before vertigo is produced (unlike vertigo from barotrauma) and the position of the diver during a jackstay swim. When performing a swim along a line on the sea bed any diver who is slightly buoyant usually assumes a prone position with his head down (inclined at approximately 20-30° from the horizontal) and he swims in this position so as to remain close to the seabed. This ensures a caloric stimulation of the horizontal semicircular canals, 180° from the position assumed during the traditional caloric tests.

.../DISCUSSION
DISCUSSION

The importance of orientation underwater has axiomatic relevance to the safety of the diver. Probably the most distressing factor causing disorientation in diving is vertigo. It is a common disorder amongst divers, as has been demonstrated in all available surveys, and supported by the clinical observations of medical officers experienced in diving. The general surveys tend to stress the incidence of vertigo, as opposed to its aetiologies. The problem of vertigo in diving has received an added impetus following the introduction of free diving and then of helium and deep diving over the last few years. The medical officer confronted with a vertiginous diver has a series of aetiologies to consider, other than those occurring in the general population. The latter are not dealt with in this paper.

1. Caloric stimulation causing the vertigo in three possible ways.

   a. Perforated Tympanic Membrane. The diagnosis of this is obvious on otoscopic examination. The common presenting causes are related to the movement of the tympanic membrane inwards, because of a pressure gradient from the external to the middle ear. It can be ascertained from a history of the conditions leading up to the incident. These include middle ear barotrauma of descent, and being "finned". The results of this injury may be either cochlear and/or vestibular damage, either temporary or permanent.

   b. Unilateral external auditory canal obstruction. The diagnosis of this aetiology for vertigo is obtained on otoscopic examination. Cerumen or an otitis externa are obvious causes. In these cases the interference with hearing is of the conductive type, and readily reversible, whereas the vestibular dysfunction is also of a transitory nature.

   c. Unequal vestibular response to a bilateral and equal caloric stimulus.

2. Inner ear barotrauma. This, by definition, results from a change in pressure. It occurs during ascent or descent, with related changes of the gas volume spaces within the ear. Internal ear barotrauma, from any of the three causes, may result in vestibular and/or cochlear damage, temporary or permanent.

   .../a. External
a. External Ear Barotrauma of Descent is able to be ascertained from the diving information e.g. with the diver having some obstruction to his external ear space. It also is identifiable on otoscopic examination, with haemorrhages and vesicles noted in the external canal. The damage occurs during descent, because of contraction of the air space within the external ear and its transmitted effects on the middle and inner ears.

b. Middle Ear Barotrauma of Descent, with forceful Valsalva manoeuvres. This is a particularly common cause of both hearing loss and vertigo in divers. The diving history is characteristic, with the diver having difficulty in equalising pressures within the middle ear, and over-zealous attempts to correct this by the Valsalva. Both the hearing damage and vertigo may be delayed in onset. Tinnitus is usually noted immediately. Relief of the pain occurs when the diver ascends, but this need not necessarily relieve the internal ear damage.

c. Middle Ear Barotrauma of Ascent. This is usually a mild disorder, and is especially noted during the initial phase of the ascent. Once the Eustachian tube has opened, it then appears to cause no further difficulty during that dive.

3. Decompression Sickness.

Although there is reference to central connections of the vestibular apparatus, most of the cases that have been investigated have demonstrated a peripheral impairment of vestibular function. This can be complete, as can the deafness which may be associated with it in some cases. It is not certain whether the involvement is due to an intravascular bubble obstructing the blood supply to these organs, or whether there are gas bubbles within the organs themselves. The diagnosis is only considered when the diver has performed a dive capable of producing decompression sickness, with the qualification that when deep or helium diving is performed, this disorder may occur earlier than would otherwise be anticipated according to the traditional concepts of decompression. It is important that accurate diagnosis be made, as therapeutic recompression may be completely curative in its effect.

Definitive statements cannot be made regarding the production of vertigo due to altered pressures of gases in solution. Some evidence has accrued in favour of the production of vertigo with inert gas narcosis, from both nitrogen and helium, and from oxygen and carbon dioxide pressure alterations. In these cases there is no known involvement of cochlear function, and the vestibular disorder appears to be correctable once normal gas pressures have been resumed.

5. Unequal Vestibular Responses.

This diagnosis can only be made with assurance following formal vestibular function testing e.g. electronystagmography, with positional and caloric tests. There are many stimuli in the undersea and hyperbaric environments that are capable of producing vertigo, especially if an underlying inequality of vestibular response is present. The stimuli which may well act equally on both vestibular systems, but produce vertigo if the vestibular sensitivity is unequal, include:

- caloric effects
- barotrauma effects (both descent and ascent)
- abnormal gas pressures.

6. Miscellaneous.

There are many factors which may be aggravated by diving and which may produce vertigo. Migraine and sensory deprivation are two that have been proposed on the basis of case histories, however there are many other disorders which may similarly be incriminated. Otitis media has common associations with both diving and vertigo. Many other causes of vertigo may need to be added to the above classification, as more information becomes available. One common presentation is the occurrence of vertigo some 5-10 minutes after the commencement of a dive, but without any other aetiologica factors appearing to be of specific import. This, for want of a better term and to demonstrate our ignorance of this field, we have termed "Idiopathic vertigo of diving". It appears to be totally reversible and is often reproduced if the diver submits himself to similar conditions. No hearing loss is demonstrated in these cases.
With the identification of serious hearing and vestibular dysfunctions amongst the diving population, it is reasonable to obtain baseline data from candidates entering this field. There are also obvious requirements for laboratory investigation of otological problems with diving. In discussing the equipment requirements and procedures, a separate report has been presented (Blackwood and Edmonds).
CONCLUSION

The aetiology of vertigo with diving has been assessed with reference to the relevant medical literature and case reports. It has been demonstrated that vertigo may occur: due to caloric stimulation, in which case there is either a perforation of the tympanic membrane, unilateral obstruction of the external auditory canal or unequal responses to vestibular stimulus; inner ear barotrauma, due to external ear barotrauma of descent, middle ear barotrauma of descent with forceful Valsalva manoeuvres, or middle ear barotrauma of ascent; decompression sickness; abnormal gas pressures, especially inert gas narcosis, oxygen toxicity and the carbon dioxide "off effect"; unequal vestibular responses; and miscellaneous conditions which include migraine, sensory deprivation and idiopathic vertigo of diving. Methods of differentiating the various aetiological conditions, both on clinical grounds and on laboratory investigation, are discussed.
REFERENCES


.../13. JARRETT


