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EDITORIAL

When Samuel Butler said that Life is the art of drawing sufficient conclusions from insufficient premises he was more accurately summarising the development of Science (and Medicine) than most of his contemporaries would have recognised. The truth of this aphorism will be apparent when one contemplates the cycle of development, refinement, and final reassessment with modifications, of the dogmas of any period. It will be noticed that this process is more rapid nowadays than formerly and that Diving Medicine is subject to the same need to change.

It must be difficult for newly trained divers to appreciate that conditions which are so clearly described and differentiated, in detail, in every reputable instruction book written for them, may present a diagnostic puzzle to a doctor asked to give an opinion. Similarly it is an increasing problem to get acceptance of the fact that, despite the enormous amount of work done to calculate safer diving tables, there will never be any tables which guarantee 100 per cent security to the user. This is due to the variability of response of each person on different occasions to a similar load of dissolved gas. Some day divers may wear computers which calculate an ascent profile for each dive after ascertaining the Doppler count of the bubbles in some chosen blood vessel. Then it will become obvious that the safe dose of bubbles is not known. As ever, advances will produce new problems in their wake. The wise diver treats the tables as indicating the outer limits of reasonable security, and dives well within these limits. Computers may calculate dive profiles but such machines are entirely dependant on the information fed them, and our understanding of the processes involved in causing decompression sickness is too imperfect for the necessary information to be correct.

It is indeed unfortunate that Nature does not obey the logic of our understanding of diving. How much simpler it would be if cerebral air embolism or decompression sickness always presented as the medical texts describe rather than as the muddled and misleading mess of information so often offered by those with the less severe examples of these conditions. Cases in this issue relate some of the clinical problems which bedevil those torn between the "pot everything" approach and a more conservative "make a diagnisis first" one. It is now becoming easier, with the increasing availability in Australia and New Zealand of recompression chambers, to choose the Test of Pressure option, which is a diagnostic aid with a therapeutic function. It is wise to keep in mind the near certainty that some residual damage remains after the clinical symptoms and signs are long forgotten lest a generation of divers arises which regards decompression sickness as a matter of little importance.

The clinical papers in this issue illustrate some of the diagnostic and management problems which may be encountered, beginning naturally with the initial assessment of fitness to dive of the applicant with an adverse neurological medical history. While most decisions concerning fitness to dive are beyond dispute there are inevitably a number where factors peculiar to the applicant, or the examiner's special expertise concerning risks of some condition, lead to a divergence from the accepted guidelines. Such instances include those where diabetes or a history of asthma is discovered and the applicant denies the condition has had any effect on his everyday activities. It is hoped to discuss these conditions in greater detail in a later issue and comments are invited from readers. Such conditions are illustrative of what Samuel Butler noted, that our premises rather than adequate data govern our beliefs.

It is such cases which justify the suggested listing of doctors with enough understanding of diving problems to be able to occasionally depart from whatever "guidelines" are thought necessary to increase diving safety. Readers are reminded that in only a tiny minority of fatalities has a medical condition been the sole fatal factor, and in some it was the victim's personality which was the critical factor. It would be administratively convenient to decide on a simple shibboleth, the words "I went to the SUM", but, as Dr Gibbs says in his letter, there are other founts of knowledge. This is obviously a matter for discussion and action.

After the rich supply of clinical reports all dealing with the unpleasant aspects of venturing underwater, ranging from decompression sickness and cerebral air embolism to marine venoms, from deafness to death, it is good to read Martin Marks on how a dive marshal, or any dive leader, can assess the likely safety of a proposed dive. Remember at such times Ken Kizer's valuable concept of "the sand beside the road". Without adequate equipment and appropriate training you stand a good chance of experiencing this factor. So dive by the rules and you will be alive to read the next issue!

SPUMS ANNUAL SCIENTIFIC MEETING 1984

AIR EMBOLISM - A DISCUSSION BASED ON TWO DIVING ACCIDENTS

John Knight

Arterial gas embolism (AGE) occurs in divers, submarine escape trainees and in patients having open-heart surgery or even arteriography. It can occur in people who have suffered venous air embolism if there is a patent foramen ovale or a vascular anomaly in the lungs which by-passes the lung capillaries. Margery Allingham had one of the characters in "The Mind Readers" die from arterial gas embolism when a "sparklet" operated cork remover pierced his chest and heart.

CASE HISTORIES

Case 1

An experienced sports diver in his late 20's finished a decompression dive and ascended normally. He is sure that he was breathing in and out all the way up and came up not faster than 60 feet a minute. On the surface he found swimming difficult as his legs were suddenly weak and difficult to control. He reached the boat and had to be helped in as his arms had become weak. Once on board he collapsed. He could hear people talking but could not see. To the onlookers he was unconscious. He was hyperventilating and having occasional extensor-spasms. He was like this for more than half an hour. Then he woke up and appeared normal except for his left leg. The others in the party, occupied with packing up, let him wander off and drive himself home. A couple of days later he contacted me because he kept on missing his mouth with his teacup and missing the saucer when he put the cup down. Recompression cured him. This case has been reported in more detail in the SPUMS Journal of April-June 1983.[1]

Perhaps the diagnosis of the doctor (another diver) on board the dive boat, that his troubles were due to hyperventilation was right but as hyperventilation follows iatrogenic arterial gas embolism I prefer to explain his problems with an air embolism.

Case 2

A young woman was finishing her diving training course by doing open water dives. The first dive was uneventful. Back in the boat after the second dive she said that she was not well, then collapsed, stopped breathing and went blue. Mouth to mouth resuscitation was started and after about five minutes she started breathing and regained consciousness. About 10 minutes later she went unconscious and stopped breathing. Once again expired air resuscitation was successful. Soon after the boat arrived in port and she was transferred to hospital. There she had no demonstrable neurological abnormalities.

SETT experience has shown that the symptoms of air embolism may come on minutes, sometimes many minutes, after leaving the water. Those on board thought that she had had an air embolism and I agree with them. I believe that her second collapse was a second embolism brought on by sitting up.

People have died ascending from as little as two metres without breathing out and the standard explanation is that pulmonary barotrauma occurs when the lung is over stretched. As a consequence of the lung bursting air can find its way into the interstitial tissues of the lung and of the mediastinum, down below the diaphragm retroperitoneally or intra peritoneally, up into the neck, into the pleural cavity and also into the pulmonary venous system. These latter bubbles then pass through the heart and are pumped out into the aorta and so reach the brain causing arterial gas embolism. It is a reasonable explanation.

We know that human corpses burst their lungs when intrapulmonary pressure, measured in the trachea is about 90 mmHg, which is roughly 1.2 msw.[2] Binding the chest and abdomen allowed the pressures to be approximately doubled before the lung burst. Admittedly Malhotra and Wright only reported 5 experiments, one unbound and four bound cadavers. The unbound body developed a pneumothorax while the others developed pulmonary interstitial emphysema. As they had no circulation none developed air emboli.

The majority of studies of air embolism come from Submarine Escape Training Tanks (SETT). Large numbers of men perform these buoyant ascents every year and a few suffer pulmonary barotrauma and air embolism. Very few have evidence of interstitial emphysema or of pneumothorax. The ascent rate is very rapid. At HMS DOLPHIN the trainee is clipped onto a wire rope running up the centre of the 100' tank to make sure that he does not hit the wall and damage the paintwork, or himself. In spite of the friction of the clip on the rope the survival suited figure is travelling fast enough for those at the top of the tank to see clear between his legs before he drops back into the water. Sports divers do not achieve this speed of ascent. They also wear wetsuits which, in many cases, splint their chests and abdomens. So it might be that SETT trainees and sports divers acquire their air emboli differently.

There are a number of inconsistencies about pulmonary barotrauma and arterial gas embolism. The pages in the 1973 Bennett and Elliot that deal with air embolism suggest that haemoptysis, signifying lung damage, is common.

Macklin reported pneumothorax with massive collapse from experimental over inflation of the lung in cats in 1937.[3] He jammed a catheter into a bronchus and was rewarded with massive air leaks into the lung perivascular spaces, the mediastinum, the retroperitoneal tissues, the neck and the pleural space. What he did NOT report was air in the pulmonary vessels.

Macklin and Macklin published a long paper (77 pages) in 1944 on "malignant interstitial emphysema of the lungs and mediastinum".[4] It was malignant because it led to an anoxic death after steadily increasing dyspnoea and great inspiratory efforts. Their conclusion was the overdistention of alveoli adjacent to blood vessels allowed air to enter the potential space around the blood vessels with each inspiration and each expiration pushed this air towards the hilum, so stiffening the lungs and making the dyspnoea worse. The process was self perpetuating. Certainly increasing dyspnoea, cyanosis and death was a common ending in the influenza pandemic of 1919 and with pneumonias before antibiotics.

When an anaesthetist forgets to release the expiratory valve during controlled ventilation the usual result is surgical emphysema of the neck or a pneumothorax. Yet both these manifestations of pulmonary barotrauma are much less common in divers and SETT trainees than is AGE, which in some parts of the world is the major pressure-related problem of diving.

In the SPUMS Journal of July to September 1983, Takashi Hattori discussed ten years of diving accidents on the Monterey Peninsula in California.5 Only 20% of his cases of AGE had haemoptysis. Hattori treated more cases of AGE than of decompression sickness between 1971 and 1981. In 1982 Harry Oxer presented 6 pressure related accidents from Western Australia to a SPUMS meeting in Melbourne. Five were AGE and one was a case of high altitude decompression sickness.[6] In Victoria there are approximately equal numbers of cases of AGE and decompression sickness.

It is easy to understand how part of the lung, either partly or totally blocked off from the airways, can be over expanded and rupture, tearing capillaries and so allowing air to enter them if they remain open. It is less easy to imagine the whole lung behaving this way. In fact a friend of mine was making an out of air ascent many years ago. Suddenly his regulator was blown out of his mouth. He does not remember anything more of the ascent till he "woke up" on the surface. He had a bit of a cough for a while but felt well enough to have another dive that afternoon. Certainly he had an over pressure accident. Was his unconsciousness due to a faint caused by intrapulmonary pressure interfering with cardiac filling or was it due to an air embolus? No one knows.

The lack of X-ray changes and of pathological changes in the lungs of clinical air embolism cases treated at the Royal Australian Navy School of Underwater Medicine led Carl Edmonds to refer these people to Professor Colebatch for exotic respiratory function tests. Professor Colebatch found that there was evidence of differences in compliance in different parts of the lung in these people.[7] This is unusual. The testing is time consuming and complicated and quite unsuited as a screening test for divers. His explanation for AGE in some of these people is that at the boundary between the normal and abnormally stiff parts of the lung expansion of the normal lung, being more than that of the stiffer lung, tears some of the alveoli between the two areas. This of itself is of no great significance as the intrapulmonary pressures, though raised, are in the normal ratio.

However when the diver, who was out of air, reaches the surface and gasps in his first breath, the normal lung expands maximally and pulls away from the stiffer lung, which holds open the holes in damaged venules and capillaries. Air enters the damaged area, as it does the rest of the lung, on inspiration. On expiration it cannot leave and either goes up the perivascular spaces towards the hilum or enters the damaged blood vessels. His contention is that it requires a deep inspiration to either damage or overfill the damaged area between the normal and abnormally stiff parts of the lung. This is an interesting and appealing hypothesis and explains the following case.

One of my patients developed a pain in his chest doing a series of forced vital capacity tests on a Vitalograph. So I had his chest X-rayed and his mediastinum was very neatly outlined with air. I assumed that he had burst his lung while blowing, but could not understand how or why. When I discussed the occurrence with Professor Colebatch he thought that the maximal inspirations had done the damage and that the forced expiration had pushed the air towards the hilum, which is at least believable.

It seems more likely to me that a recurrence of symptoms following an air embolus is a new embolus rather than anything else. After all the lesion in the lung cannot have healed in minutes or even hours. A clot has probably formed within minutes but the bleeding would presumably distort the lung architecture and compliance so transferring the original problem to some other part of the lung. It is quite likely that there is interstitial air in or adjacent to the damaged lung.

To me the interesting things about AGE are:

(1) that it is often diagnosed as hyperventilation by doctors inspite of very obvious neurological signs;
(2) that it occurs much more often than the other manifestations of pulmonary barotrauma;
(3) if Professor Colebatch is right about the mechanism of air embolus in people with "normal" lungs why do only some have relapses?

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STONEFISH ENVENOMATION

Chris Acott

CASE REPORT

JR trod on something while getting out of a boat in an estuary. He experienced sudden pain which became excruciating over the next ten minutes. He arrived at the Rockhampton Base Hospital about an hour later, in considerable pain. No first aid measures had been tried.

I made the diagnosis of a stonefish sting over the telephone when I asked the Registrar to take the ear piece away from his ear and I could hear the unfortunate patient screaming in the background. His foot was immersed in some hot water, which gave him considerable relief, and he was transferred to the ICU.

The wounds were on the medial aspect of his right foot in the distribution of the medial plantar and sural nerves. These were blocked using Bupivicaine 0.5%. There were 6 superficial puncture wounds, bluish in colour with a reddened periphery. His foot was also oedematous. His right inguinal lymph nodes were palpable but not painful, but he was complaining of considerable discomfort in his calf and thigh. There was no joint pain. Every time his foot was removed from the hot water, he complained of pain.

An intravenous line was inserted. Blood was taken for analysis. 6 ml of antivenom (3 ampoules) were given intravenously as I considered that a 6 ml intramuscular injection would be cruel.

The pain subsided within half an hour. When he arrived the pain seemed so severe that I considered a unilateral sub-arachnoid (spinal) block, but decided to block the sural and medial plantar nerves instead.

After the antivenom he only complained of some moderate discomfort in his foot and a cramping sensation in his calf and thigh muscles which were controlled by incremental doses of intravenous Omnopon. This discomfort continued for a few days. The foot remained swollen for a couple of days. However there was no sign of local infection.

Investigations revealed a CPK three times normal. The blood was taken about 1 hour after the sting. Discussion with Dr S Sutherland revealed this to be the first recorded case of an elevated CPK following a stonefish sting. He assumed that it was due to the local effects of the venom on the muscles of the foot.

JR was discharged three days after admission.

DISCUSSION

This case illustrates several points about stonefish envenomation:

1. The sting is excruciatingly painful. Although the patient was in considerable distress he did not become irrational, which has been reported in other cases. However the severity of signs and symptoms are directly proportional to the depth of penetration of the spines. His wounds were superficial.

2. First aid measures: Considerable relief is obtained by immersing the affected area in hot water (about 50°C), so the venom is probably heat labile.

3. The wound appearance was classical from the description given in “Dangerous Marine Animals of the Indo-Pacific Region”, by Carl Edmonds. “The area has a bluish tinge, and becomes swollen and oedematous. The pain will spread to the regional lymph nodes and involve adjacent muscles.”

4. The best methods of pain relief are by local anaesthesia blocks, antivenom and hot water immersion.

The Venom

It is a heat labile protein with a molecular weight 150,000. It also contains some potent hyaluronidase and capillary permeability factors. There is no protease or phospholipase A2 activity. It has no effect on the blood clotting mechanisms nor does it cause haemolysis.

The venom will remain toxic in the venom glands for several days after the death of the animal. There are 13 dorsal spines to each stonefish and each spine has 2 venom glands which can discharge along the ducts of the spine. The spines are capable of penetrating a sandshoe. The average yield of venom per spine is 6 mg, and the total yield is 49-88 mg.

Tests on various animals showed transient hypo- and hyper-tension. Electrocardiographic changes showed either AV block or VF. Other workers showed it to have strong myotoxic properties which effects all muscle types, causing paralysis probably by blocking depolarization.

Deaths have been recorded in the Indo-Pacific region, but no deaths have been recorded in Australia.

Antivenom

The antivenom is prepared by immunizing horses and is available as a pure equine preparation. Each ampoule contains 2 ml which will neutralize, in vitro, 20 mg of venom. Antivenom is indicated in all cases of stonefish stings. The recommended doses are, for 1 or 2 puncture sites 1 ampoule, for 3 or 4 puncture sites 2 ampoules, and for 5 or 6 puncture sites 3 ampoules. The antivenom is usually given as an intramuscular injection except in severe cases when it is given as an intravenous injection.

First Aid and Hospital Management

1. No attempt should be made to retard movement of venom. To delay the escape of the venom will only enhance local pain and tissue necrosis.

2. Immerse the limb in hot water at about 50°C.

3. Emetine hydrochloride has been tried in the past. Injection around the wound will give some pain relief, probably due to the acidity of the solution.

4. Potassium permanganate should not be used, as it will only cause local tissue damage and aggravate the wound.

Hospital management is directed towards pain relief, antivenom, bed rest, and treatment of any cardiovascular or respiratory problem. Attention should be paid to tetanus prophylaxis and local wound treatment.

THE UNDERWATER TRAINING CENTRE IN MORWELL

John Knight

Why have an underwater training centre 60 km from the coast? Largely because basic training and training in the use of equipment and tools does not need the sea, only water. In fact the sea can be a nuisance because it can get too rough to be safe. The Commercial Diving Center, which is now the College of Oceaneering, in Wilmington, California, has access to very protected water and yet has large on-shore tanks for training. The other reason is that Morwell is the base for the major activities of the National Safety Council of Australia (NSCA) Victorian Division which purchased the Underwater Training Centre (UTC) from its Sydney owners about 2 years ago.

The UTC trains divers to the standards set by Part 1, Scuba Diving (*'persons not normally working underwater but who are required to dive in connection with archaeology, non-commercial research, scientific work and observation tasks"), P*art 2, Restricted Commercial Air Diving (*'personnel who will be engaged in professional and/or commercial underwater operation, at limited depths .... using surface supplied compressed air or self contained breathing apparatus and not having access to a surface compression chamber. Such qualification is the minimum required by regulatory authorities who are responsible for the control of on shore diving, eg construction of jetties, dams.") and Part 3, Prof*essional Air Diving with surface compression facilities *("Such qualification is the minimum required by regulatory authorities who are responsible for the control of off-shore diving, eg oil and gas exploration") of* the draft Australian Standard for the Training and Certification of Divers. Part 4, Bell Diving (*'related to the further training of experienced air divers and underwater workers to permit them to operate safely and competently as bellmen and lock-out divers")* has not as yet been started. There are a number of reasons for this including the present depressed state of the diving industry and its high unemployment and the lack of a safe 100m deep site at sea off the Victorian coast. This depth is necessary if the UTC training is going to be accepted by the UK authorities as equivalent to their standards for bell divers in the North Sea oilfields.

In addition to training divers the UTC is engaged in helicopter-crash survival training. They have the cabin of a helicopter sitting on a bar. It is held vertical by uprights at each end of the bar. The trainees get in, and the assembly is hoisted up and positioned over the pool and then rapidly lowered into the pool simulating a crash at sea. The RAN and ESSO-BHP are using this service.

Another group being taught by the UTC is the NSCA air-sea rescue paramedics who not only have to be able to winch down to a ship but are trained to parachute and scuba dive.

The UTC has a pool that is 6 m in diameter and 9 m deep which is used for most of the training. It is in this that trainees learn the joys of wearing a Kirby Morgan Band mask, so called because the hood is held to the mask by a steel band. These have an oro-nasal mask instead of a mouthpiece on the regulator, which is all part of the full face mask. The regulator can have its working pressure adjusted and can also be converted to free flow. There is a delightful gadget that pushes up and blocks the nostrils so that the diver can clear his ears more easily. The great advantage is that the diver can talk to topside and hear replies. There is a microphone in the oro-nasal mask and earphones lie against the ears. Like any other full face mask it must be held firmly onto the face by straps or it will fill with water.

The KM Superlite is hardly light. It is the much more comfortable successor of the brass "hard hat". The Superlite has the mask part of the KM band mask set in a complete helmet. Worn inside the helmet is a padded hood which provides comfort and insulation. It is worn with a neck dam, a tight fitting neoprene sleeve round the neck which broadens out over the shoulders to be attached to a metal collar that locks onto the helmet. Because the head is in air the diver can hear better than with a band mask. Because the neck dam seals the helmet it can be worn with both wet and dry suits.

The band masks and helmets are surface supplied. The umbilical has a hose for the breathing gas and another for the pneumofathomometer as well as wires for the communications. The pneumofathomometer tube is open at the bottom. Air is blown down the tube at a pressure above that at the diver's depth, then the supply is turned off and the pressure in the system will drop to that of the diver's depth. This pressure can be read off a pressure gauge calibrated for depth. This is a much more accurate system than relying on a possibly faulty depth gauge on the diver's wrist, which of course the supervisor cannot see!

In the pool the trainees work with various power (hydraulic) tools such as are used for construction work. Welding underwater was a skill taught at the Commercial Diving Center when I visited it in 1977. At present the UTC is not teaching this skill.

A commercial diver must not only be safe at work in the water but he must be able to provide a safe environment for others to work in the water. This means learning how to control gas supplies to the diver and how to work the pneumofathomometer. The control panel varies in complexity, being relatively simple for a surface supplied bounce dive. The control panel for a chamber has a full set of controls for each compartment and for the trunking joining any bell to the chamber. There are deep and shallow pressure gauges, ideally one pair for each compartment. But often gauges can be connected to more

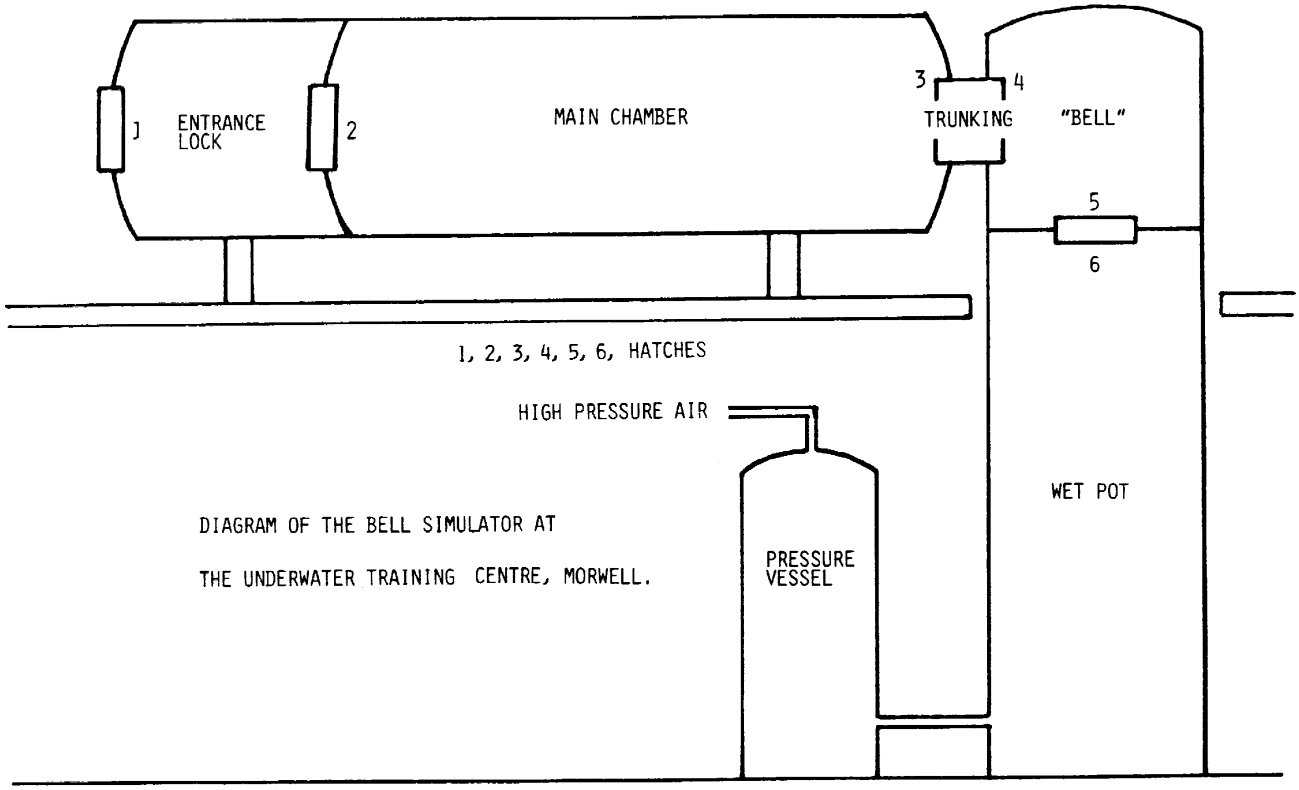


FIGURE 1

The hatches (doors) are on the chamber side (inside) their openings. They will stay shut as long as the pressure in the chamber is higher than outside. The exception is hatch no. 6 which will remain shut as long as the the pressure outside the "bell" is higher than inside.

than one compartment. If the supervisor thinks it is connected to compartment A when it is in fact connected to another, inappropriate action may be taken. This happened a few years ago in the North Sea when the supervisor, thinking the master gauge was connected to the living chamber when in fact it was recording bell pressure which was being dropped, pressurised the men and induced fatal hyperthermia. Besides controls for the chamber there have to be controls for the supply of gas to the bell and to the divers. The diver's life is literally in the hands of the man at the control panel.

The UTC acquired a two compartment multi-man chamber (4.8 m x 1.8 m) from Comex when that company finished work in Bass Strait. It is a deep chamber rated to 200 m. The entrance lock has two entry ports at right angles so that another chamber can be attached and there is still access from outside. At the other end of the main chamber there is an overhead entry hatch and another in the far wall, which leads into the bell simulator (Figure 1).

The interior of the chamber is dimly lit. There are only two lights and they are not actually in the chamber. The lamps are outside the chamber and the light reaches the chamber through a fibre optic system. The main living space is cramped for four men (there are four bunks) and is fitted with CO_2 scrubbers and can easily be used for saturation, but there are no toilet facilities.

The bell simulator, which is attached to the end of the large chamber, is an ingenious device to train people to operate a bell (properly a personnel transfer capsule) to different depths without having to go to sea. It consists of a "bell", fitted with all the normal controls, permanently mounted to a wet-pot. At the UTC the wet-pot, although rated to 200 m is pressurized only to 50 m at present. Pressurisation is achieved by having another pressure vessel full of water connected to the bottom of the wet-pot. High pressure air is blown into the top of this second tank and the pressure rise, but no air, is transferred to the wet-pot. In this way a dive to 50 m can be simulated.

The diver and his attendant enter the "bell" and close the hatches between it and the chamber. They hoist the outside door (No. 6 in Figure 1) at the bottom of the bell almost closed with the chain-hoist. They leave a gap of about 1 cm between the door and its sill. The supervision starts to pressurize the wet-pot. As the water starts to rise into the lower trunking the men in the "bell" pull the chain-hoist chain sideways, so lifting the door to touch its sill. The outside water pressure now pushes the door tighter and tighter shut. The chain-hoist is unhooked and put out of the way. This is the same procedure that is used as a bell is lowered into the sea. Once the "bell" has reached working depth it is pressurized. When the pressure in the bell is the same as outside the bottom door falls open as it is no longer being pushed up by the water pressure.

Now the diver can enter the water and perform his tasks. Training includes diver rescue, which is difficult through the narrow Comex hatches. At the end of the exercise the diver returns to the "bell". The inside door (No. 5 in Figure 1) of the trunking is shut and the "bell" is slightly over pressurised to seal the hatch. Now the higher pressure in the bell keeps it shut. At sea the bell would now be hoisted aboard and the divers transferred to the chamber either to be decompressed or to be held in saturation. The same routine is undertaken in the simulator, the only difference being that there is no mating of the bell to the chamber as that link has never been undone. Nevertheless those in the simulator have to go through the same routine of having the chamber pressurised and then the trunking, joining chamber and bell, pressurised before they can open the bell and crawl through into the chamber.

The UTC hopes to obtain another small chamber to mate to the entrance lock of the large chamber, with a hatch capable of accepting the bayonet fitting of a Dräger Duocom portable 2 man chamber. The NSCA runs a hyperbaric emergency service based on Morwell using Dräger Duocoms as the portable collecting chamber with the patient being decanted into a larger chamber at Morwell. However this chamber is limited to 50 m which might not be deep enough to give relief of symptoms. At present the patient cannot be transferred under pressure to the UTC chamber. An extra chamber, with a flange accepting a Duocom, on the UTC system would allow for such transfer.

Since this paper was presented the UTC has obtained the extra chamber mentioned in the last paragraph.

NEUROLOGICAL CONSIDERATIONS IN DIVING

David Brownbill

At the outset I wish to acknowledge the work that has been done in the field of fitness to dive with neurological conditions by John Hallenbeck and Hugh Greer who are both neurologists and divers and have been very closely associated with the United States Navy.

Any discussion on neurological conditions in diving must consider the neurological conditions that can result from diving and, probably more important to us here, advice to divers for their future activities when they have a pre-existing neurological condition or one that develops as a result of diving. Pre-existing neurological conditions fall broadly into three groups, head injuries, people who have undergone intra-cranial surgery for whatever cause, and those who have unrelated medical neurological conditions. The important problems are epilepsy, the changes that may occur to personality, which I will discuss later, and damage or impairment to the special senses of vision, hearing and balance.

There are two relevant statistical factors about epilepsy. The first being that 5% of the population will at some stage have an epileptic fit of some sort, for example as a youngster with a fever and secondly that 0.5% of the population will actually be termed epileptic, having recurrent fits requiring medication. The whole point about epilepsy is that it involves a low threshold to cerebral discharges. As a result of that low threshold precipitating factors may cause discharges that will result in that which we know as clinical epilepsy. The factors which are significant in diving include cold, decreased oxygen levels, or more importantly raised oxygen levels, decreased carbon dioxide which occurs with hyperventilation, hunger, alcohol and scarring of the brain. Here we might talk about the person who has undergone a craniotomy, surgery for let us say a meningioma. If it is in the posterior fossa and if there is no balance disturbance it is of no significance. However, if the surgery has involved the supratentorial region it carries, in the normal population without diving or the attendant precipitating factors, a chance of post surgical epilepsy approaching 10%. That raises the question of the incidence of epilepsy that one may expect in a person, following such surgery, who is diving and is therefore exposed to these precipitating factors. Epilepsy itself may occur with or without loss of consciousness. It may be just a momentary loss of awareness, but it does always interfere with the control of behaviour and performance and it does always occur without warning. There may be an aura that it may occur but the actual moment of occurrence is without warning. As I said before, in diving, vital considerations involve carbon dioxide or oxygen blood level changes which often happen and which may act as a precipitating factor in a person who already has a lowered threshold.

What advice should be given to somebody regarding diving who has suffered an epileptic fit? Now the first, and I think non-discussable, situation is if they have uncontrolled epilepsy, that is, they have epileptic seizures and are on medication. Without discussion I would say, not only should they not dive, they MUST NOT dive, full stop. The next question is regarding the person who has well controlled epilepsy, that is the person who has been on medication and has been seizure free for two years. Here we have to consider that such medication may make people drowsy and under increased ambient pressure these effects may be increased. Such persons have, of necessity, a remarkably reduced threshold to epileptic discharges which makes them more susceptible to precipitating factors. They should also be advised not to dive. The more controversial area is the one of, so called, arrested epilepsy, the person who has been five years free of epilepsy and has not been on medication. The current wisdom is that they be advised to avoid incidents where the oxygen pressure may increase, to avoid hyperventilation, to take care of currents, cold and stress. In other words they are to dive perfectly and never encounter any problems. I differ from the current wisdom and believe that anybody who has suffered an epileptic seizure should not dive. This advice is mandatory if they have uncontrolled epilepsy. In the other groups they make the decision but my advice is that they should not dive.

Personality was mentioned before and although this is not strictly a neurological consideration it does come into play, for example, following head injuries, cranial surgery or any form of cerebral insult. All of these may alter the personality but experience has shown that they accentuate a pre-morbid personality. Very rarely do these insults make a gentle person aggressive or the other way round. What does tend to happen is that the person's personality traits are highlighted. When this is being looked at one must consider tendencies to aggression or tendencies to schizoid behaviour which may well be accentuated by a past cerebral insult. The changes may affect logical actions under stress, or they may affect the ability to make decisions, for example in dive planning. This is a small but significant consideration.

The next question relates to the diver who has suffered a past decompression insult to the central nervous tissue, either within the head or within the spinal cord. This may include recovered paraplegia or damage to the special senses or involve motor sensory impairment, or loss of consciousness. What advice should be given to the person who clinically has made a complete recovery?

To take the first example, the one who has a recovered paraplegia, a so-called "spinal hit". They lose bladder and bowel control, may have total sensory and motor disturbance and recover with appropriate treatment. If they make a recovery they tend to make a very good one and it may, to all intents and purposes, be complete. I am going to use this as an example of central nervous tissue damage because there are several well recorded cases of such people who have died for unassociated reasons and have come to post-mortem. Sections of the spinal cord have shown quite clearly marked pathological changes of sclerosis and fibrosis of tissue indicating areas of neuronal death that have not had the expected clinical manifestations. Experimentally it has been known for a long time that central nervous tissue that is damaged from whatever cause has scar tissue that is more susceptible to decompression illness than areas that are not involved. Once can be certain that the person who has a completely recovered paraplegia from decompression sickness has marked damage throughout the spinal cord and the advice I would give under these circumstances is that they should not dive, and by extrapolation, I believe people who have had a decompressive insult to the central nervous tissue, that is they have suffered sensory motor disturbance or loss of consciousness, or special sense impairment which would be reasonably believed to have resulted from a bubble, should not dive. There should be no further diving by those exhibiting an incomplete recovery. But there appears to be no reason why the person who has complete paraplegia following trauma should not dive. In fact many have been taught to dive and as long as it is done by experienced teachers, as there are problems with buoyancy, balance, propulsion and problems that occur with distribution of fluids within the body because of the loss of vaso-motor control, I place no bar at all on them diving.

Migraine is, in itself, not disqualifying unless it is severe and frequent. What the person and their diving companions must realise is that if they do suffer a severe migraine attack on returning to the vessel this can cause confusion with symptoms that would arise from an unassociated decompression sickness and therefore one may well be placed in a situation of having a migraine attack treated by recompression. If in doubt that course is by far and away the safer as it does not do any harm to the migraine.

When discussing head injuries one must consider dural penetration by a spicule of bone or early post-traumatic fitting, as in each of these situations there is, unrelated to diving, a greater than 30% chance that at some stage a late post-traumatic fit will occur. That means they have a markedly reduced threshold to electrical discharges and that means they are much more susceptible to the precipitating factors that may result from diving. Of all the people who develop late post-traumatic epilepsy from a head injury, more than a quarter of them have their first fit more than four years following the accident. Looking at people following a head injury, who have suffered an early fit, or a dural penetration, nearly 10% of them will commence fitting after four years and that, of course, has great bearing on the advice one must give them as to the risk they run if they dive.

A valuable yard stick to use in advising people following head injuries is the United States Air Force (USAF) guidelines for evaluation of fitness to fly. For convenience head injuries are divided into four groups from severe to very mild.

The permanently disqualifying conditions are contusional laceration of the brain or an immediate post head injury fit. These two are absolutely disqualifying conditions. The USAF guidelines also include as permanently disqualifying conditions those who have had a head injury with a loss of consciousness of more than 24 hours.

Conditions that will disqualify for two years after injury include unconsciousness for greater than two hours or a post-traumatic amnesia greater than 48 hours or a post-concussive syndrome of sleep disturbance, personality disturbance, headaches or memory disturbance for more than a month. It should be noted that there is no worthwhile correlation between electroencephalographic findings and any prediction of late post-traumatic fitting. The results of such investigations really bear little relation to the decision as to whether someone should dive or not.

The person who is unconscious for 15 minutes or more is not allowed to fly in the USAF for three months. I see no practical or theoretical consideration to regard that any differently from the advice we give to someone who is going to dive.

The last group is of those who should be disqualified from diving for four weeks. This has implications to those of us who look after the younger age group, for example footballers who suffer head injury with unconsciousness that lasts for less than fifteen minutes. This is a mild head injury and yet involves a low threshold to epilepsy, low threshold to personality changes and perhaps decreased reflex reactions under stress. I support, in diving, very much the view of the USAF and would recommend advising anyone with a mild head injury against diving for four weeks.

For commercial diving any central nervous tissue abnormality or doubt of the integrity of the nervous tissue functioning should preclude diving. For the rest of us diving is fun, and there are other sports that can be chased without putting oneself or ones buddies at risk. I do not think central nervous tissue damage, with the few exceptions mentioned, is really compatible with safe diving.

Surgeon Captain RR Pearson

One of the things we have been trying to get a grip on in the Royal Navy and things about which we have had conflicting advice is the relative significance of pre-traumatic amnesia and post traumatic amnesia.

Dr D Brownbill

The two forms of amnesia are those for events before the accident (pre-traumatic) and those for events after the accident (post-traumatic). The one that has a very close correlation with the severity of the head injury is the post-traumatic amnesia. In nearly all instances pre-traumatic amnesia or retrograde amnesia is short even for very severe head injuries. It is occasionally long but when it is long it does not correlate at all well with the severity of the head injury but the post-traumatic amnesia does. Indeed in medico-legal circles experience has lead me to believe that when somebody with a moderate head injury has a retrograde amnesia extending six hours, it is often with the intent of trying not to remember certain aspects.

Question:
What raised levels of oxygen can precipitate epileptic fits?

Dr D Brownbill

The generally accepted figure is that of two atmospheres of inspired oxygen. It would be most unusual if it occurred below a level of 150 mm of mercury of oxygen in the blood. In a person with a low threshold to epileptic discharges if the partial pressure of oxygen in the blood drops below about 80 mm of mercury this may precipitate a fit.

Question:
What about leakage of cerebro-spinal fluid following a head injury? Should these people dive?

Dr D Brownbill

If CSF rhinorrhoea or otorrhoea is proven (and the only accurate way to do this is by specialised dynamic CSF radiological tests) the person should not dive. As with a lot of assessment of fitness for diving, if there is a reasonable doubt that someone has a cerebro-spinal fluid leak they should be advised not to dive. It may be said that this would apply only until they had a craniotomy and repair of a dural defect. But that operation will entail a risk, of about 10%, of post-surgical epilepsy occurring. That in itself, would suggest that diving should not be continued.

Question:
What about a person who has suffered an isolated fever convulsion as a child?

Dr D Brownbill

Such a convulsion fits into the group previously discussed of 5% of the population who suffer an isolated seizure. Clearly one cannot be too dogmatic in this situation. I would be prepared to allow such a person to dive but I would think it right and proper that diving companions should be informed that this person has an increased risk of having a fit.

The reasons people may die when they have a fit underwater involve some things I did not bring up before. What to do if your buddy is having a fit? He has a risk of dying for two reasons. One is that he loses his mouthpiece and drowns. If he does have a fit and is lucky and his mouthpiece is held in position then he will not drown unless he bites through it. If one is close enough and can hold the mouthpiece in position very good, but here is where a certain amount of self-control is needed. The first thought will be “Get him up quickly” but one must not do this, as the second reason such people may die is because in the early stages of a fit they are breath holding. With glottic spasm such a person runs a very good chance of rupturing the lung and receiving an air embolus if they are brought to the surface. Therefore what one should try to do is hold the mouthpiece in until respiratory activity returns and then take the diver, who may still be unconscious to the surface with his head held extended to allow passive exhalation. The period one may have to wait may seem an eternity but it is, in fact, only between 20 or 40 seconds. There are, of course, apparent risks in waiting with such a person before coming to the surface but on the balance of risks I would regard waiting as the safer procedure.

Question:
Should people who are involved in sports such as boxing, where they receive regular mild head injuries, be allowed to dive?

Dr D Brownbill

If these people have not suffered a fit or a significant head injury, I do not think we can be dogmatic about the advice given. We know they are receiving regular cerebral damage but we do not know if they have scars sufficient to increase an epilepsy tendency. One would try to ascertain the number of occasions of loss of consciousness, for example, and the periods of such loss and try to explain to the individual the increased risks that would be run, for example if they encountered a change in oxygen or carbon dioxide levels, and the decision to dive would remain with them.

Question:
People with disabilities such as epilepsy are mostly determined not to be seen as different from other members of the community and may not wish to have their right to dive taken away because of that affliction. Do you not think that because of their wish to participate fully in the community many epileptics should be allowed to dive?

Dr D Brownbill

Actually unless they have suffered repeated cerebral damage from anoxia resulting in aggressiveness and loss of normal thought processes most people who suffer from epilepsy are responsible people who are amenable to logical discussion. If they were told they should not dive because they may drown or they should not drive a car because they may kill themselves they might reasonably say that they are prepared to accept that risk. However, if in discussion one points out such fitting may endanger the lives of their buddies or other people on the roads, my experience suggests that they will readily accept such advice. I agree that it is hard to find a case where a person suffering an epileptic seizure underwater has resulted in a death but I would regard it as impractical to suggest that someone who is on medication for epilepsy be allowed to dive.

Question:
Diabetics may sometimes have changes in conscious level. Should they be allowed to dive?

Dr D Brownbill

A discussion on the management of diabetes is perhaps not appropriate here but it is worth commenting on the prospects of a diabetic suffering a hypoglycaemic attack whilst diving. Such a person who has decided to dive should take precautions by omitting their normal dose of insulin beforehand and they should take some sugar beforehand and even carry a little plastic bag with some sweets whilst diving because they will have warning of an impending attack and they will have learnt to recognise such warnings very quickly. The careful and experienced diabetic, with appropriate advice, should therefore be able to dive but again his buddy should be aware of the problems.

Dr David Brownbill is the Senior Neurosurgeon at the Royal Melbourne Hospital.

A CASE REPORT - SEA SNAKE BITE

Chris Acott

I do not wish to repeat myself and give another talk on sea snake envenomation, however we had another sea snake bite late last year.

A 19 year old youth was swimming in some murky water when he thought he saw a stick on the bottom. He dived down and picked it up. The stick turned around and bit him on his forearm in two places. One bite was at the wrist and the other on the upper aspect of his forearm.

First aid measures were applied immediately, and he was brought into hospital. When the crepe bandage was removed there were no signs of envenomation. He had first degree heart block on his ECG, and I began to rub my hands with glee that it might have been caused by the venom, but disappointingly it turned out to be congenital.

This was the third sea snake bite that has come to the Rockhampton Base Hospital in the past three years. Only a little girl required treatment for envenomation. This agrees with Reid's original work which said that only a one-third of cases show signs of envenomation.

The girl was bitten on the foot, again the snake meant business. Again Reid's work was verified as she showed signs of a massive envenomation. The symptoms occurred within 2 hours (Reid's 2 hour rule) and required 8,000 units of the antivenom.

Reid divided his cases of sea snake envenomation into 'serious' and 'non-serious' by his 2 hour rule. Serious envenomation was indicated by myalgic pains especially of the neck muscles, trismus, ptosis, ophthalmoplegia, myoglobinuria and a leucocytosis of greater than 20,000, all occurring within 2 hours, if no first aid measures had been applied. Serious cases required up to 10,000 units of the antivenom, while the non-serious required only about 3,000 units.

Sea snake venoms are either neurotoxic or myotoxic or a combination of both. The neurotoxic venom acts either pre- or post-synaptically or with a combination of both. The myotoxic venom causes muscle destruction with myoglobinuria and myoglobinanaemia which may lead to renal failure.

One should use either sea snake antivenom or Tiger snake antivenom. As a last resort poly-valent antivenom can be used.

SPUMS NOTICES

OBJECTS OF THE SOCIETY

To promote and facilitate the study of all aspects of underwater and hyperbaric medicine.
To provide information on underwater and hyperbaric medicine
To publish a journal.
To convene members of the Society annually at a scientific conference.

MEMBERSHIP OF SPUMS

Membership is open to medical practitioners and those engaged in research in underwater medicine and related subjects. Associate membership is open to all those, who are not medical practitioners, who are interested in the aims of the society.

The subscription for Full Members is $30.00 and for Associate Members is $20.00.

Membership entitles attendance at the Annual Scientific Conferences and receipt of the Journal.

Anyone interested in joining SPUMS should write to:

Dr Chris Acott,
Secretary of SPUMS,
Rockhampton Base Hospital,
Rockhampton QLD 4700.

NOTES TO CORRESPONDENTS AND AUTHORS

Please type all correspondence, in double spacing and only on one side of the paper, and be certain to give your name and address even though they may not be for publication.

Authors are requested to be considerate of the limited facilities for the redrawing of tables, graphs or illustrations and should provide them in a presentation suitable for photo-reduction direct. Books, journals, notices for symposia etc., will be given consideration for notice in this journal.

LETTERS TO THE EDITOR

MEDICAL EXAMINATION OF SPORTS DIVERS

Lloyd Medical Centre
PO Box 131
31 Brisbane Street
Mackay QLD 4740

Dear Sir

While applauding the general policy of more stringent medical examination of Sports Divers by suitably qualified doctors, I object strongly to the issuing of a list comprising only those doctors who have passed the course at the RAN School of Underwater Medicine (SPUMS J 1984; 14(4): 11).

I have not attended the above course, having previously received training and experience far in excess of that available while serving as a Medical Officer in the Royal Navy. I am accepted as a doctor qualified to perform medicals on commercial divers by the Health and Safety Executive, UK, the Professional Divers Association of Australia, and a number of commercial firms. I know of several other SPUMS members with similar levels of experience who are in the same situation.

By all means issue a list of approved diving doctors, but let it be a comprehensive list which does not discriminate in favour of a select group.

Ian R Gibbs

The above letter was shown to Dr. John Knight whose suggestion the list was. His reply is printed below.

80 Wellington Parade
East Melbourne VIC 3002

Dear Sir

I would like to explain why I recommended a list of doctors who have completed the RAN School of Underwater Medicine (SUM) Courses to the Australian Underwater Federation (AUF). (SPUMS J 1984; 14(4): 11).

Firstly, the SUM has records of who have passed the exams at the end of their courses, so a list of their graduates can easily be prepared.

Secondly, in Victoria two government departments dealing with diving have been reluctant to recognise the UK Health and Safety Executive (H&SE) approved (to examine professional divers) doctors, largely on the grounds that they have no way of checking the standard of the training of the H&SE approved doctors.

Thirdly, no organisation has so far been willing to compile a list of all those doctors in Australia who have been adequately trained in diving medicine. This requires assessment of the training claimed and checking that it was done.

Fourthly, as no one individual or organisation knows the names and addresses of all the doctors in Australia who have been adequately trained in diving medicine I chose to keep the recommended training easily identifiable and verifiable, hence the RAN SUM suggestion, which is administratively simple.

Fifthly, I had no intention of excluding doctors with adequate training from the list. I assumed that, when they heard about the list, they would volunteer, in writing, their training and experience to whoever was publishing the list and request to be included.

It would help to create a complete list of diving doctors in Australia if Dr Gibbs and others in a similar position, write to the Secretary of SPUMS detailing their training and experience so that SPUMS can construct the list.

John Knight

MEDICAL EXAMINATION OF SPORTS DIVERS AND FIRST AID FLOWCHARTS

North Canterbury Hospital Board
Private Bag
Christchurch
New Zealand

18th January 1985

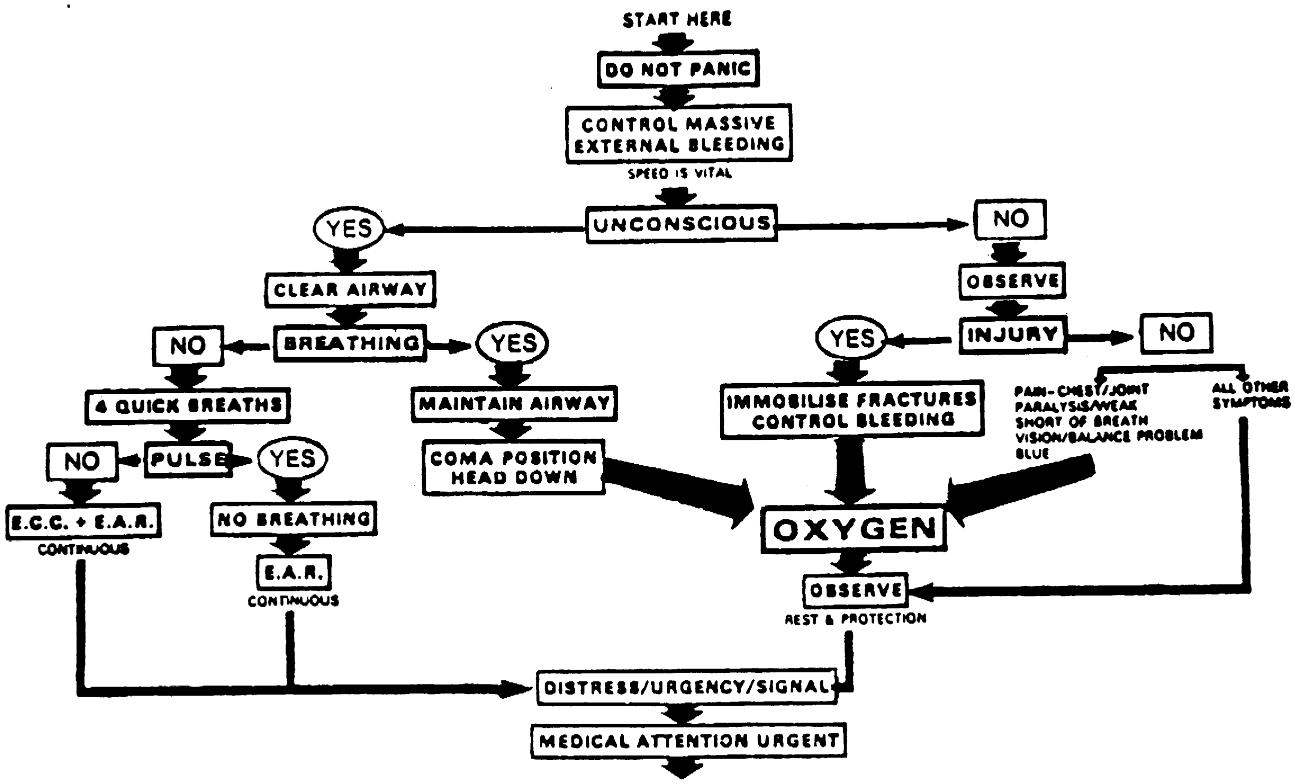
Dear Sir

I was interested to read the correspondence with AUF on the question of medical examination for sport divers. I thought John Knight's reply on behalf of SPUMS was well balanced and sensible. I have felt for some years that the increasing pressure for annual medicals for sport divers should be firmly resisted as it is of little benefit for the reasons given in the letter. I would certainly endorse the current SPUMS approach.

Turning to another matter. Some while ago my article on First Aid for Diving Emergencies was published in the *SPUMS Journal* (1981 Supplement: 63-67) and more recently John Knight's modification of the flow chart for Australian use was also published (SPUMS J 1983; 13(2): 47-51). I thought SPUMS members might be interested in further developments along this line in New Zealand. After wide spread discussions with a number of New Zealand SPUMS members, the Water Safety Council and the NZUA, a format has been agreed to for a plastic card which is to be distributed through the NZUA system. On the card the arrows are in red (Figure 1). The reverse of the card is reproduced as Figure 2.

Mike Davis

The two sides of the chart are reproduced on the opposite page.



HOSPITAL or RECOMPRESSION CHAMBER
(AUCKLAND (09)454 000 Duty Medical Officer or CHRISTCHURCH (03)792 900 Duty A&E Doctor)
RETAIN EQUIPMENT FOR INSPECTION ★ INCIDENT REPORT (NZUA, P.O. Box 875, Ak.)

FIRST AID FOR DIVING ACCIDENTS

This chart provides simple-to-follow procedures for diving accidents. It can only be of use if you have undergone CPR (Cardio-Pulmonary Resuscitation) Training (1).

NOTES ON CHART
E.C.C. = External Cardiac Compression
E.A.R. = Expired Air Respiration

MASSIVE EXTERNAL BLEEDING: control with dressings/towels etc plus firm hand pressure (2).

COMA POSITION: on side, lower arm straight down and behind back, upper hand by face, upper leg flexed; head down 15° (30° if Air Embolism suspected).

IMMOBILISE FRACTURES: use snorkels, fins, paddles etc strapped (e.g. weight belts) to limb; injured leg strapped to uninjured (2).

OBSERVE: Conscious state)
Airway)
Breathing) Principles of C.P.R.
Circulation)

Assess Injuries
Symptoms + Signs + Circumstances ⟶ ? Diagnosis

DISTRESS/URGENCY SIGNAL: if in imminent danger and immediate aid required use MAYDAY. If MAYDAY not warranted but URGENCY required use PAN PAN. Switch to 2182kHz or 156.8MHz (Chl. 16), International Distress frequencies. Use call procedures advised by Marine Division (3).

N.B. 1. CONVULSIONS may occur in an unconscious diver. The principles of CPR apply plus restraint of convulsive movements to protect victim from injury.
2. In contacting assistance, the Police co-ordinate such operations: on shore, dial 111.

ACUTE MEDICAL CONDITIONS WHILST SPORT DIVING (4)

Pulmonary Barotrauma - Air Embolism, Pneumothorax, Emphysema.
Decompression Sickness.
Near Drowning.
Traumatic Injury and or Marine Envenomation (N.B. Spinal Injury).
Shallow Water Blackout.
Ear Barotrauma.
Hypothermia - may contribute to or complicate any diving accident.
Heart Attack.
Gas Impurities: CO_2 Retention.

REFERENCES

1. CPR manual. The National Heart Foundation of New Zealand, P.O. Box 17-128, Green Lane, Auckland.5.

2. First Aid Manual. St. John Ambulance/N.Z. Red Cross 1982.

3. Marine Notice Series B Number 1A - distress and urgency information (issued by Marine Division, M.O.T., revised June 1975).

4. The Divers Medical Companion. Thomas R. and McKenzie B. Diving Medical Centre 1980.

ACKNOWLEDGEMENT: Dr F.M. Davis, Princess Margaret Hospital, Christchurch
PRODUCED BY: New Zealand Underwater Association
P.O. BOX 875, AUCKLAND (09) 895-456/7

NITROGEN NARCOSIS

Ken Kizer

Nitrogen narcosis, that prominent effect of the undersea environment that is always talked about in diving classes and is experienced by almost all divers at one time or another, was the subject of a recent two day workshop sponsored by the Undersea Medical Society (UMS) and the National Oceanographic and Atmospheric Administration (NOAA).

The obvious question is: “What was the interest in holding a workshop on nitrogen narcosis? After all, isn’t everything important about nitrogen narcosis already known?” Well, soon after the workshop started it became evident that such is not the case. In fact, it became quite clear that very few things about nitrogen narcosis are known with certainty, except, of course, that it occurs.

The primary reason to hold the workshop was to address the potential problems that nitrogen narcosis presents for the scientists and other personnel who will be utilizing the NOAA-sponsored undersea habitat that is to be built at the Catalina Marine Science Centre off southern California, the Western Regional Undersea Laboratory (WRUL), as it is now called.

In the WRUL scientists will live in a specially designed undersea habitat for seven to fourteen days at a time, at depths between 60 and 120 feet, breathing a 50-50 mixture of oxygen and nitrogen (nitrox). They will make air breathing excursions to do work at depths well below 200 feet. Besides the questions about whether subsea scientists can work safely and effectively under these conditions, there is a very fundamental question as to how reliable the data will be which they gather. This is not a trivial question when you consider that the experiments that they will be doing may involve hundreds of thousands of dollars and months or even years of preparation.

Although these questions could not be answered, the participants, under the direction of the workshop chairman Dr Kizer, did review what is currently known about nitrogen narcosis and provided some direction for future research in this area. A full report of the workshop will eventually be published by the UMS, but some of the findings might be of interest to readers now.

The role of nitrogen narcosis in modern diving was characterized as “sand beside the road”, meaning that it is not normally a problem because most divers stay out of it. However, it is always a potential problem for compressed air breathing divers, and it is an enormous operational consideration for commercial and scientific diving because of the limitations it imposes on air diving. Indeed, narcosis is the primary (but not the only) reason for limiting the diving depth when using air.

The actual biophysical and molecular mechanism of nitrogen narcosis is yet to be defined, although it is known that the elevated partial pressures of nitrogen found at depth acts in much the same way as the anaesthetic gases that are used in surgery, that is, the nitrogen blocks conduction of impulses along nerves.

Of course, the major questions considered at the workshop related to how much nitrogen narcosis influences performance and what can be done to moderate its deleterious effects. Although workshop participants reviewed the scientific literature on this subject, it rapidly became clear that much has yet to be studied. Most of the studies that have been done to date have looked at the effects of narcosis on the performance of single, relatively simple jobs that do not require the diver to deal with distractions or to perform complex tasks involving multiple simultaneous or sequential functions. Similarly, very few studies have investigated the effect of narcosis on overall perception, memory, or integration of multiple bits of information. Obviously, much needs to be done in this regard, some of which would be directly relevant to recreational divers, especially with regard to the role of narcosis in sport diving accidents.

One investigational methodology for future nitrogen narcosis studies that was discussed in some detail was the possible use of video games for hyperbaric chamber experiments or for making daily assessments of diver performances and acclimation in the undersea habitat. These have a number of potential advantages over traditional psychological and performance tests. For example, video games can easily record, store, and analyze test scores; they are self-motivating, and they require only a modest amount of training. Who knows, underwater Pac Man may be just around the corner.

Another topic that received a lot of discussion was the phenomena of “adaptation” to nitrogen narcosis; that is, the process of a diver seemingly becoming less and less susceptible to the effects of nitrogen narcosis after repeated exposures during a short time period. (Unfortunately, this is not a permanent effect.) This process has been variously called adaptation, acclimation, acclimatization, tolerance, accommodation and other things, but it was agreed upon at the workshop that the correct term that should be used in the future was “acclimation”.

Although the effect of acclimation has been repeatedly observed, many questions remain. Is this just a behavioural adaptation or is there really a physiological change? Is it merely a matter of learning to cope with the environment, ie a matter of learning to cope with “underwater stress”? Does this occur more easily in some divers than others? Can the process be speeded up by performing certain pre-dive activities? This list goes on. Unfortunately, no definite answers are available at this time to these or other important questions.

On a related subject, though, one of the things that was evident from reviewing the role of nitrogen narcosis in diving accidents was that it is a frequent contributing factor, although rarely are accidents caused solely by narcosis. It seems to play a major role in causing accidents in which the diver has to deal with some emergency or unplanned situation. The solution to this problem seems to be in overlearning essential skills under controlled conditions. There is a clear message here for training programs, which is just as relevant to sport divers as commercial divers.

A number of other topics are also discussed at the workshop and several recommendations were made for future studies on the subject, but space does not allow for discussion of these things here. They will be detailed in the forthcoming workshop report. In the meantime, though, a detailed bibliography of essentially everything that has ever been written in the English language on the subject of nitrogen narcosis is available from the Undersea Medical Society.*

* Nitrogen Narcosis - A Bibliography with Informative Abstracts. Undersea Medical Society, 9650 Rockville Pike, Bethesda, Maryland 20814, USA. US$8.00

AIR EMBOLISM OR DECOMPRESSION SICKNESS?

A CASE FOR DIAGNOSIS

John Knight

A 27 year old male diver with the proper training was diving at a sinkhole. He states that he did a dive to 34 m for 14 minutes which he said was within the USN No-decompression limits. He went to between 110 and 120 feet, 33-36 m, and should have used the greater depth which means that he overstayed the USN no-decompression limit by 2 minutes. However he did a 5 minute decompression stop at 3 m as a precaution, which took him inside the USN requirements for his dive. He said that he felt stressed by the dive and the water was cold (12°C). As soon as he was out of the water he stripped off his wet suit and went to stand in the sun to warm up.

10 to 15 minutes after surfacing he noticed that his "peripheral vision was dark" (his own words). Within 15 minutes his vision had returned to normal.

Interestingly he commented that an instructor acquaintance of his had noticed this phenomenon on more than one occasion after diving in sinkholes.

When seen and examined a month later he had no neurological deficit. He had worked out this his symptoms were due to cerebral anoxia and wanted to know whether they were due to air embolism or decompression sickness.

Retrospective diagnosis of a transient symptom is always difficult. Here we have to decide between two potentially lethal diagnoses, one of which would bar further diving.

I think that he did not suffer an air embolus due to pulmonary barotrauma. He made a normal ascent, with a decompression stop, and was breathing in and out all the time. His symptoms came on when he was rewarmed, at least 10 minutes after getting out of the water, if his story is correct. So I think that pulmonary barotrauma is very unlikely.

However, there is no doubt that he did have cerebral circulatory insufficiency for a while. Again his normal ascent and decompression stop makes arterial bubbles due to a rapid ascent unlikely, and if he had generated arterial bubbles by rapid ascent one would expect the immediate onset of cerebral symptoms, not a delay of 10 to 15 minutes. Perhaps he could have developed bubbles in his brain which compressed blood vessels leading to ischaemia of the visual pathways, but that does not seem very likely.

My bet is that as he warmed up he restored the circulation to his limb muscles and fat, which had closed down, in response to the cold stress of his dive, after he had loaded these tissues with extra nitrogen early in the dive because he was nervous and overbreathing. As a result bubbles formed in his limb capillaries and were carried to his lungs. Some bubbles bypassed the lung capillary filter, entering arterio-venous shunts and reached the left ventricle and so the aorta and the cerebral circulation.

This would then be a case of decompression sickness presenting as cerebral ischaemia.

Have any of our readers got alternative explanations? If you have please put pen to paper or finger to typewriter.

UNSCRAMBLING HELIUM SPEECH

A group of scientists at Edinburgh University, under their director Dr David Milne, have developed a range of "helium speech unscramblers" capable of taking the diver's "quacking" noises and converting them into recognisable speech on the ship, an invaluable service to the Dive Supervisor and his team. Dr Milne reports that the unscrambler has been tried out in the North Sea work situation for over a year. There are at present two systems in use, a big system for use by a diving company and a smaller system. The former has seven unscramblers, loudspeakers, and tape decks for entertainment circuits and costs about $35,000. The alternative system provides divers' radios which contain a simple unscrambler and a three-person communication, cost about $4,000.

Dr Milne is also working on the development of a through-water, diver-to-diver communication system. This project will employ a more advanced technology than the unscrambler both being based on special circuits which use commercial "charge coupled" devices. Essentially both do the same thing, taking the distorted "helium speech" and storing it for a few milli-seconds before playing back the corrected speech sounds. He is certain that the through-water systems will become increasingly important as they become more sophisticated and flexible. The potential market is large for use in both civil and military diving. Manufacture is now in the hands of Findlay Irvine and sales through Underwater Instrumentation of Aberdeen and London.

A SYSTEM FOR SURVIVAL?

Martin Marks

It is often apparent from incident reports that divers are getting into trouble because they undertake dives that are outside their capabilities. Frequently this is not obvious to the inexperienced Dive Marshall, because the hazard is not a single factor such as a gale or diving in a whirlpool, but is a combination of several lesser things each of which, on its own, would be acceptable but when combined they produce a risky, even dangerous situation.

To help avoid such situations, I have tried to devise a system for rating dives by splitting up the elements and giving them scores. I must emphasise that the intention of this article is to put forward a system for comment, your comment. Do you like the system? Is it too complex? Is it relevant to your sort of diving? Before seizing your pen, read through the outline below and try out the system on some dives that you have done which you know were a bit risky.

SIMPLE APPROACH

The system is intended as a guide for the inexperienced Dive Marshall when planning a dive. It acts as an *aide memoire* in terms of the factors he should consider, and produces general guidance on the acceptability of the proposed dive. It cannot possibly cover every eventuality without becoming unwieldy, and so a simple approach has been adopted.

It is assumed that the diving is taking place in accordance with normal BS-AC practice; that the equipment used is serviceable and appropriate; and that BS-AC RNPL or RN Table II decompression tables are being used and adhered to. Where elements vary during the dive (eg. current) the worst case should be used. A general background of average Branch diving is assumed.

For each of the sections in the questionnaire the Dive Marshall should select the relevant or nearest category and put the score in the column on the right hand side. If the section is not relevant or appropriate, score zero. Some scores can be negative.

ASSESSING SCORES

The scores for all the sections are then totalled to produce a score for the proposed dive which can be compared with the categories below. The system is designed for general guidance only. If two different dives score 34 and 44 respectively, it means that both are dangerous rather than indicating any relative degree of danger. Claims for higher scoring dives should be submitted to your life insurance company.

Assess scores on the following basis:

- Less than 10: This should be a safe dive. However, if all the penalty comes from a single element, the Dive Marshall should ensure he/she understands the hazard involved.
- 10 to 20: An element of risk is building up. Examine the areas of risk to see how it could be modified to reduce the hazard.
- 21 to 30: This dive is too dangerous as it stands. Either considerable modification is needed or the dive should be abandoned.
- Over 30: Positively dangerous.

The National Diving Committee is interested in your reaction to the system, so give it a try and if you have a constructive comment, please write with your views.

1. Planned depth:	less than 30m	0
	30m to 40m	8
	40m to 50m	18
	over 50m	33

2. Water temperature:	warm	-2
	UK Summer	0
	UK Winter	8
	Under ice	18

3. Expected underwater visibility

over 10m	-2
2 to 10m	0
less than 2m or night dive	8
zero viz	18

4. Current: enter the predicted current on the table and use column A for a drift dive, B if divers are to remain stationary on the bottom or C if they must swim against the current:

	A	B	C
Slack	0	0	0
Less than 0.5 knot	5	10	15
0.5 to 1 knot	8	16	24
Over 1 knot	18	24	32

5. Dive duration:	No stops	0
	Stops required	15

6. Dive support:	Boat cover	0
	Shore cover	8
	No cover	24

7. Surface marker buoy:	Used	0
	Not used	15

8. Weather:	Force 0 to 3 score	0
	Force 4	4
	Force 5	8
	Force 6 or more	22

9. Diving in or with unfamiliar equipment: 8 per diver

10. Diving site unfamiliar to Dive Marshall and Dive Leader: 8

11. Low surface visibility or offshore dive out of sight of land: 8

Reprinted, by kind permission of the Editor, from DIVER, July 1984. Martin Marks is the Chairman of the BS-AC Diving Incident Panel.

PROVISIONAL REPORT ON AUSTRALIAN DIVING RELATED DEATHS. 1983

Douglas Walker

SUMMARY

There were thirteen (13) diving-related deaths identified as having occurred in the period under review. Complete details are still unavailable (November 1984) on four of these. There were three breath-hold, seven scuba, two hookah and one rebreather diver deaths. Critical factors for the breath-hold divers were respectively trauma (hit by a motor boat), blackout following hyperventilation, and the combination of fatigue and cold. In the cases of the scuba divers, two were both untrained and inexperienced, one trained but inexperienced, the others were reported as both trained and experienced. In one case there may have been a cardiac problem and aspiration of water. Of the hookah users, the critical factors in one were depth (185 ft), cold, narcosis, nil visibility, entanglement, and insufficient air supply. The other was diving alone to free his fishing nets, the details are still unavailable. The victim using the rebreather was trained but relatively unfamiliar with the set and had done no recent deep dives, probably suffered nitrogen narcosis as the depth was 180 fsw, was alone, and had too low an airflow to support exertion. Nearly all the deaths were potentially preventable.

CASE NOTES

The dive histories reveal deviations from those councils of perfection, the generally accepted principles of safe diving. The victims probably prejudiced their chances of survival little more than did many others, but to them was presented the dive end-point of death. There being no appeal against such terminations it is prudent to gain merit, and preserve life, by following the rules.

Case BH 83/1

Motor boats and swimmers cannot safely co-exist in the same area of water, the risks to the latter being too serious. This diver and his friend were in an area frequently used by swimmers and divers, shallow water over a rocky ledge which ended at deeper water used as a motor boat channel. The buddy was closer to land than his friend when he heard a thud and a cry and then the sound of a motor boat stopping. He swam over to the spot and found the severely injured victim at the surface. He died shortly after he was placed in the boat. It would have been very difficult for anyone in the boat to see a black wet-suited person in the water even had a special watch been kept.

MOTOR BOAT. TRAUMA. BOAT CHANNEL. NO FLAG. BLACK WETSUIT HARD TO SEE.

Case BH 83/2

The victim was an expert spearfisherman training to improve his endurance. He descended breath-hold down a weighted line to hookah-using pearl divers who saw him start his ascent. His body was not recovered. His death was almost certainly due to drowning following a post-hyperventilation blackout.

POST HYPERVENTILATION BLACKOUT. ALONE. EXPERT BREATH-HOLD DIVER.

Case BH 83/3

After spearfishing separately both divers surfaced and found that their boat was adrift. The buddy left the victim at the surface and dived to reposition the anchor. When he surfaced he was unable to find his friend despite a search. The victim was not wearing fins and the sea was cold and choppy. The body was not recovered for eight days with the weight belt still on. No buoyancy vest was worn.

COLD. FATIGUE (INCREASED WORK DUE TO NO FINS). CHOPPY SEA. NO BUOYANCY VEST. FAILED TO DROP WEIGHTS. POOR BOATMANSHIP (ANCHOR INSECURE, UNATTENDED BOAT). SEPARATION SURFACE PROBLEM (UNEXPLAINED).

Case SC 83/1

Four friends, in two boats, anchored off a rocky shore in order to scuba dive. In one boat was a diver with five years experience who “didn’t consider himself proficient” but was nevertheless taking a friend on her first ever scuba dive. Their dive was soon aborted as the girl felt cold. It is not stated whether they wore wet suits. The other pair consisted of the victim, said to have been trained and to have some experience and the buddy, who had loaned him the scuba he was using, whose experience was not recorded. Both wore wet suits. After about forty-five minutes the buddy was low on air and returned to the boat. It is apparent that they dived separately, not as buddies. The other pair brought their boat closer and then one of the group saw the victim’s scuba unit bobbing about at the surface in shallow water and the victim’s body was then found floating face up. As he appeared to be dead no attempts at resuscitation were made. Autopsy showed signs of pulmonary barotrauma and drowning.

SEPARATION/SOLO. LOW AIR SITUATION. PULMONARY BAROTRAUMA. NO GAUGE OR J VALVE. NO BUOYANCY VEST. DITCHED SCUBA. WEIGHT BELT ON (?). INEXPERIENCED.

Case SC 83/2

This death occurred during a post-certification course on wreck diving. During the third dive of the weekend they were on a wreck in 25 m deep water in a calm sea. After the victim and his buddy completed their assignment on the wreck they were joined by the course instructor, who till then had been maintaining general overview of his class while undertaking some underwater photography. It was about 20 minutes from the commencement of the dive that the victim indicated that he was low on air and the three divers swam to the anchor line. Somewhat to the buddy’s surprise the victim was to ascend alone while he continued with the instructor, though he later realised that they had not seen one of the other pairs and it was necessary to ascertain whether they were still down. It was later established that one of the pair had a severe migraine and they had aborted their dive. After a quick search the instructor and the buddy returned to the anchor and started their ascent. The instructor, as was his usual routine, looked down at the dive site as he started his ascent and was surprised to see the victim's camera on the sea bed. He then saw the victim nearby, his mask half full of vomit, regulator out of his mouth. He placed the regulator of his "octopus rig" in the victim's mouth and inflated his own buoyancy vest to assist their ascent. After surfacing he inflated the victim's buoyancy vest and ditched the weight belts of himself and the victim. The resuscitation attempts were unsuccessful. The victim had made mention of some nausea before the dive but had stated firmly that he was fit to dive. It was stated later that he had experienced and successfully managed underwater vomiting on a previous occasion. The onset of the vomiting was probably too rapid for him to drop his weights, inflate his vest, or avoid fatal aspiration. The time between separation and being found was about 7 minutes.

TRAINED. EXPERIENCED. SEPARATION FOR SOLO ASCENT. LOW AIR. FAILURE TO DROP WEIGHTS OR INFLATE VEST. UNDERWATER VOMITING.

<u>Case SC 83/3</u>

As a kindly meant deed the buddy, who was trained and experienced, hired scuba equipment for the victim (but not a wetsuit because he was too large for those available). He had apparently made only a few previous scuba dives, all several years previously. He declined to wear the cylinder in his buoyancy vest "because it rubbed and hurt his chest." The weights provided were found to be excessive (21 lb) but he chose not to reduce them. After a successful dive, when the gauge was showing imminent low air, the sound of a passing motorboat caused them to slightly delay their ascent. As they ascended the victim seemed to be red faced and to have some unknown problem so the buddy attempted buddy breathing. He seemed unresponsive at the surface but started struggling when offered assistance. The buddy attempted unsuccessfully to drop his weight belt and keep him at the surface but lost hold and the victim sank. A search was made by nearby divers, initially without success. When found by the summoned rescue services his mask was off and his tank still contained some air. The water was warm and conditions safe for diving.

UNTRAINED. INEXPERIENCED, SCUBA HIRED BY CERTIFICATED DIVER. EXCESS WEIGHTS. NO WET SUIT. NO CYLINDER IN BUOYANCY VEST. FAILED TO DROP WEIGHTS. LOW AIR MOTOR BOATS IN AREA. UNKNOWN PROBLEM THEN PANIC. VALIANT BUDDY.

<u>Case SC 83/4</u>

The pipe from a dam supplying an irrigation scheme on a farm was leaking significantly between the dam and the pump house and this could not be repaired without stopping the flow of water. Unfortunately no valve had been placed on the pipe's inlet and the exact position of the open end of the pipe was unrecorded. It was suggested that a diver could be employed to find and cover the open end of the pipe, divers having been employed successfully when other owners of dams had problems. The farmer therefore went to the nearest dive shop to enquire about obtaining someone able to perform this job. One of the customers in the shop at the time offered his services, stating that he had performed similar jobs previously. He arrived at the dam with several helpers and entered the water alone, a stout rope round his waist and a metal probe in his hand, intending to identify the pipe's open end before placing a piece of metal sheet over it. The water was turned off at the pump valve, though naturally it continued to escape from the leak. Suddenly the rope almost tore from the tender's hands, then went slack. It had parted as the victim was sucked into the pipe, dying instantly from a broken neck. Frantic efforts were made to open up the pipe and rescue the diver but his body was washed out in the gush of water and only later found in the nearby flooded area, his tank torn off. The possibility of water flow into the pipe occurred to the tender but was discounted by the victim: neither realised the head of force resulting from 26 feet deep water in the dam.

DAM. UNEXPECTED SUCTION INTO OPEN PIPE (26 FT HEAD OF WATER) TRAUMA.

<u>Case SC 83/5</u>

During a weekend cruise the vessel was anchored off a beach to give passengers an opportunity to go ashore or go shore diving. Three, all trained, decided to scuba dive and were rowed ashore. The water was shallow near the beach (15 ft) and when one became short of air and swam back to the vessel on the surface the other pair continued to scuba until the victim also became low-air, when they surfaced together. The victim seemed to be in some mild distress with a cramp like pain at the surface, though calmer after inflating her "compensator". The victim was seen to change to a snorkel then let it loose and go unconscious, mouth submerged. The buddy attempted in-water CPR, which was continued when back on the vessel, without reviving the victim. There was no stated previous ill health but autopsy showed evidence of previous myocardial damage. The "cramp" may in reality have been the pain of a heart attack.

SURFACE DEATH. INFLATED VEST FAILED KEEP MOUTH OUT ABOVE WATER. COLD WATER. PREVIOUS MYOCARDIAL DAMAGE. PROBABLE HEART ATTACK.

<u>Case SC 83/6</u>

Few details are available beyond the fact that the victim dived alone, having waited till his friends finished their dive before being able to borrow a mask, having forgotten his own. Alarm was felt when he failed to return to the boat but a search was unsuccessful. The equipment was later recovered but there was no trace of the victim. Available information is insufficient for proposing any scenario for this incident.

ALONE. EQUIPMENT RECOVERED WITHOUT BODY.

<u>Case SC 83/7</u>

The victim and his son, overseas visitors, were making a day trip to dive on the Barrier Reef. Both were certificated and experienced scuba divers. The vessel also carried competitors for a spearfishing competition who left the dive boat before the two scuba divers entered the water. They checked each other's scuba then entered the water and descended together. Separation occurred near the bottom despite good visibility. After a short search underwater the buddy surfaced, asked those on the boat whether his father had surfaced. He then decided that non-appearance indicated that his father had continued to dive, so decided he also would dive alone. Shortly afterwards the victim was heard yelling a short distance from the boat. Several boys swam to assist him, but he sank before they could reach him. A breath-hold diver soon located him on the sea bed, mask off and regulator lying free. Water depth was 20 feet and the boy was not able to pull him to the surface. A more experienced diver arrived shortly and surfaced the victim by inflating his buoyancy vest. He failed to respond to resuscitation efforts. The buddy surfaced 30 minutes later. No fault was found in any of the equipment, autopsy showed no evidence of ill health. It is not known why he drowned.

TRAINED. EXPERIENCED. SEPARATION ON DESCENT, CONTINUED SOLO. UNKNOWN TROUBLE CAUSED HIM TO SURFACE AND CRY FOR HELP. SANK BUT HAD WORKING BUOYANCY AID. POSSIBLE AIR EMBOLISM.

Case H 83/1

Bravery and "Can Do" are unfortunately inadequate as protection against the several problems inseparably associated with deep diving. In this instance a deep bounce dive was thought necessary to retrieve a TV camera apparently caught on the object of the search, a lost stop-gate deep in a dam. The surface of the dam was at an altitude of 236 m, depth was 185 ft, visibility nil, the water cold, and objects capable of causing entanglement were very probably present. The victim was owner of a small dive company. His employee/friend had made a bounce dive here the previous day to check whether the stop-gate had been located by a scanner. The only problem he reported was an entanglement of his air line which necessitated him ditching his equipment and surfacing using his "bail out" scuba. Before travelling to the dam the victim had phoned a large dive company to discuss the possible need for special equipment, but left a decision till he had checked the need for divers to attach cables when the gate was found. Possibly the presence of engineers from the water authority influenced his impulsive decision to dive to check the underwater object and free the TV camera as he ascended. The dive base was a barge held in position by shore lines and the wind moved it while he was underwater. He fixed his bailout bottle on his chest, an unusual position, before descending. Line calls to his tender indicated ascent started then abruptly ceased. Failure of the diver to respond alarmed the surface party and the standby diver was ordered to descend to give assistance. When he surfaced he reported having found the victim under some ropes and drums, on his back, mask on but no air bubbles escaping, indicating absence of breathing. He attempted unsuccessfully to free the victim then ascended. Some doubt was later cast on the exact situation found, nitrogen narcosis and the nil visibility which limited contact to the use of cold hands being thought to make recollections unreliable. Next efforts were made to pull the diver free, but the line parted. The next search, also using air, failed too. Later a dive using full deep-dive protocol (oxy-helium, hot water suits, wet bell, available RCC) was successful, the body being found floating near the bottom. The TV camera pulled free after the incident. A helmet with voice communication to the surface was available but not used because a little time was needed to rig it for use. During the investigation it was established that the compressor could barely supply enough air at 180 ft depth for light work and certainly insufficient for heavy exertion or a panic or emergency situation. The second diver was lucky not to suffer a lethal mix of anoxia, hypothermia, narcosis and entanglement. No adequate allowance for the altitude was made in initial dives.

DAM. ALONE. ALTITUDE. DEPTH. COLD. NIL VISIBILITY. INADEQUATE AIR SUPPLY. ENTANGLEMENT. NO VOICE COMMUNICATION WITH SURFACE. DIVE BARGE MOVED. INEXPERIENCED AT THAT DEPTH. DIVE UNPLANNED. NARCOSIS. POSSIBLY EXCESS CO_2.

Case H 83/2

Few facts are available of the circumstances of this fatal incident. It is said the victim was using hookah with a scuba set as bail-out bottle. He is said to have dived from his fishing boat to free some nets.

NO DETAILS YET AVAILABLE

Case RB 83/1

The task was to obtain a series of bottom samples in a shipping channel, depth 180 ft. Because of a safety consciousness desire to avoid repeat dives there was a problem of having enough divers so each only dived once daily. The victim was trained but relatively inexperienced and had done no recent deep dives. Choice of flow rate was the responsibility of each diver. His was correct only for light work, with no margin for the unexpected (in the view of the other divers). He gave line calls to his tender which told of an ascent stopped after a few feet. It was imagined he had then realised narcosis had effected the performance of his task so had descended again to complete it before surfacing. However he failed to answer line calls so the standby diver was sent down. He found the victim on his back, full-face mask off, mouthpiece loosely in his mouth, unresponsive. He gave line calls to be pulled up with the victim and endeavoured to supply air to him using the demand valve of his octopus rig. This action was criticised at the court of inquiry because CT scan suggested presence of air embolism: but the "correct procedure" was not promulgated. Resuscitation failed. It was noted that his noseclip was missing: under the influence of nitrogen narcosis he may have removed his mask to replace it.

DEEP DIVE (180 fsw). TRAINED. RELATIVE INEXPERIENCE. LOW AIRFLOW RATE. NO RECENT DEEP DIVES. ALONE. NITROGEN NARCOSIS(?). REMOVED FACEMASK.

DISCUSSION

The three breath-hold fatalities followed dives where breaches of correct diving procedures were no worse than those a multitude of other divers probably commit. The supreme penalty they suffered should be a reminder that luck does not invariably protect those who tempt an unforgiving environment. Those with an interest in water safety should consider the value of identifying areas where swimmers and motorboats may seek to co-exist BEFORE an accident occurs.

The post-hyperventilation blackout syndrome is a problem

AUSTRALIAN DIVING RELATED DEATHS 1983

Case	Age	Dive Victim	Skill Buddy	Diving group	Dive base	Dive purpose	Water sea	Depth incident	Weight Belt On?	lb	Contents gauge	Buoyancy vest
BH 1	33	experienced	experienced	2 separation	land	recreation	20'	surface	off	12	N/A	no
BH 2	N/S	experienced	N/A	solo	boat	training	60'	ascent	N/S	N/S	N/A	no
BH 3	30	some experience	N/S	2 separation	boat	spear fishing	8m	surface	on	17	N/A	no
SC 1	23	trained inexperienced	N/S	2 separation	boat	recreation	<30'	N/S	on	18	J reserve	no
SC 2	31	trained experienced	trained experienced	3 separation	boat	recreation	80'	N/S	on	18	yes	no
SC 3	35	not trained inexperienced	trained experienced	2	shore	recreation	60'	60'	on	21	yes	yes no cylinder
SC 4	21	trained	N/A	solo	land	work (dam)	26'	26'	on	N/S	N/S	yes
SC 5	55	trained experienced	trained experienced	2	shore	recreation	15'	surface	N/S	N/S	yes	yes inflated
SC 6	20	not trained inexperienced	N/A	solo	boat	recreation	8m	N/S	off	N/S	N/S	yes
SC 7	53	trained experienced	trained experienced	2	boat	recreation	20'	surface	on	N/S	N/S	yes
H 1	35	experienced	N/A	solo	boat	work	185'	185'	on	N/S	N/A	N/S
H 2	27	N/S	N/A	solo	boat	work	70'	70'	N/S	N/S	N/A	N/S
RB 1	30	trained. some experience	N/A	solo	boat	work	180'	180'	on	15	N/A	yes

KEY: N/S not stated N/A not applicable

GOOD breath-hold spearfishermen MUST take into account. It is unfortunate it is not painful, for then survivors might learn. The third case illustrates that the conjunction of several factors can be fatal (cold, no fins so greater fatigue, no vest, separation at a critical moment) and that the surface also has danger for divers.

The scuba fatalities are best studied case by case, each one illustrating different potentially critical factors. Included are the unwisdom of taking an untrained, inexperienced diver scuba diving and the lack of value of a buoyancy vest without its inflation cylinder or of one which fails to maintain the wearer's mouth out of the water. The dramatic power of water flowing through a pipe to create an irresistible suction was tragically illustrated, even a rope failing to preserve the victim from harm. Solo diving and separation again appear to be adverse factors in fatal incidents, though many consider themselves their own best buddy. A buddy may not always be successful, but examination of the case reports will show buddy assistance at least offered the victim another chance of surviving. That even the trained and experienced may suffer if safe-diving protocols are breached is illustrated by case SC 83/2.

Both the hookah divers died alone. The dam dive had so many adverse factors that it is fortunate the other diver did not also die. Broken rules prejudice a diver's chances of survival.

That only the thoroughly trained should even consider using a rebreather unit is a truism generally accepted, but even a trained diver needs to keep in practice, particularly when diving at depths where narcosis or decompression

AUSTRALIAN DIVING RELATED DEATHS 1983

Remaining air	Equipment test	Equipment	Wet suit	Significant Factors
N/A	N/A	own	yes	Hit by a motor boat in a near-shore channel. No flag. No warning signs.
N/A	N/A	own	N/S	Breath-hold down weighted line to pearl divers. Seen to start ascent.
N/A	N/A	N/S	N/S	Surface separation when buddy went to recover drifting boat. Cold. No fins.
low	yes	loaned	yes	Separation. Buddy was low on air and surfaced alone. One of other pair first scuba dive. Tanks first seen floating then the body. Pulmonary barotrauma.
low	yes	own	yes	Separation. Started solo ascent up anchor line while buddies continued dive. Vomit in mask.
low	yes	hired	no	Too large to hire wetsuit. Excess weights. Buddy breathing ascent. Surfaced unconscious. Unsuccessful buddy aid. Air embolism?
full	yes	own	yes	Leaking outlet pipe of dam. Dive to find and cover inlet. Unexpected suction.
low	yes	own	yes	Surfaced. Distressed (cramp?). Changed to snorkel. Unconscious. Mouth under water. Angina?
N/S	N/S	hired	N/S	Borrowed mask after others dived. Failed to resurface. Only the equipment was found.
N/S	yes	hired	N/S	Separation on descent, continued alone. Suddenly surfaced, cried out and sank.
N/A	yes	own	yes	Cold, deep, nil visibility, altitude. Impulse dive. Entanglement. Inadequate airflow.
N/A	N/S	own	N/S	Wore scuba and hookah. Retrieving net. Pre-dive Valium? Ill health? Other?
N/S	yes	employer's	yes	Little experience at depth. Using rebreather with low gas flowrate. Nose clip off. Removed his full face-mask. Nitrogen narcosis?

problems need consideration in dive planning. Regulations governing North Sea divers do not allow any "free diving" at the depth of this dive for reasons deserving consideration when planning any deep dive.

This report is presented in the hope that knowledge of these events will increase the level of awareness of factors able to critically effect the safety of the reader's diving.

ACKNOWLEDGMENTS.

This report would not be possible without the generous and continued support of many organisations and individuals, in particular the assistance of the Attorney General's, Justice, or Law Department in every State. Such assistance is a vital element in this ongoing investigation into factors capable of critically influencing the safety of diving.

PROJECT STICKYBEAK

The aim of this investigation is to receive, store, and if appropriate publish and make available for discussion, an accurate record of all types and severities of problems encountered by divers. CONFIDENTIALITY is maintained and no details are published to identify persons involved or sources of information.

Please send reports to:

Dr DG Walker
PO Box 120
Narrabeen NSW 2101

A double fatality in a pond, again due to suction through the outlet, was reported in DIVER December 1984.

INNER EAR BAROTRAUMA - A REPORT OF 31 CASES

OI Molvaer
Norwegian Underwater Technology Center (NUTEC)

The occurrence of inner ear barotrauma with or without peri lymphatic fistula, and with transient or permanent injury to the cochlea and/or the vestibular apparatus has been discussed in this journal previously.[1,2,3] In addition to the 25 cases published by this author,[2] 31 more cases have been collected since 1980.

Case reports

1. A 32 year old male professional diver with previous normal hearing needed 12 minutes to descend to 30 msw due to bilateral equalization problems that were most pronounced on the right side. At the bottom he felt slightly dizzy and he got the impression that the volume was turned down in the right ear phone. After ten minutes at the bottom he started a slow ascent and stopped both at 6 msw and 3 msw. Immediately after getting onboard the boat he experienced vertigo and was unable to walk straight. As soon as he got his band mask off he realized that the hearing in his right ear was markedly reduced.

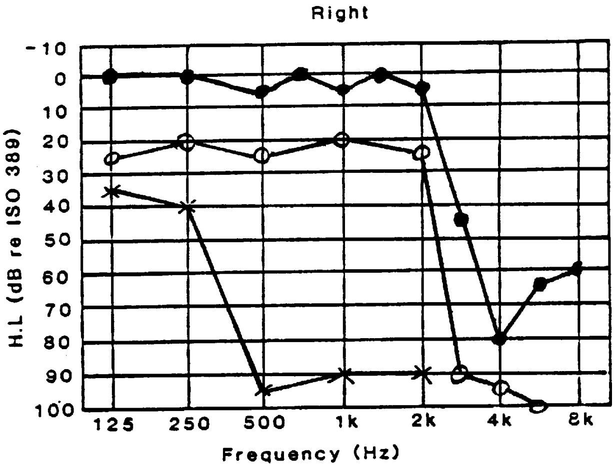
He saw a physician who found slight barotraumatic changes in both tympanic membranes and the diver got decongestant nose drops (oximetazolini chloride) and an oral decongestant (phenylpropanolamini chloride). His hearing was not examined in spite of having a plugged sensation in his right ear. The next morning he did not hear his alarm clock because his left ear lay on the pillow. He saw a diving physician who had an audiogram taken (Figure l) and administered hyperbaric oxygen (HBO) (USN table 5). During the treatment he felt subjectively better but after the treatment his hearing was still reduced and he had ringing in the right ear. A bithermal caloric vestibular test with electronystagmography (ENG) was normal. He received another HBO session and was put to bed. Later his hearing deteriorated dramatically so he was admitted to hospital for surgery four days after the incident, but no labyrinthine fistula could be found. Nevertheless, his hearing gradually improved, but he has a marked, permanent high tone sensori-neural loss and constant ringing in the ear. He had no alternative education and ran into severe economic trouble. After a successful test dive in a dry chamber he was allowed to return to his profession three months after the incident with some limitations added to his licence.

2. A 19 year old male Navy scuba student experienced pressure equalization difficulties to his middle ears due to a common cold in the 10 msw lock of the submarine escape training tower. While performing a forceful Valsalva manoeuvre he felt pain in his right ear and became dizzy. Afterwards both tympanic membranes showed signs of barotrauma, his audiogram was normal, but the ENG disclosed a right canal paresis during bithermal caloric testing of the vestibular system.

3. A 23 year old male commercial diver had trouble clearing his left ear in an open sea dive and experienced spinning vertigo and ringing in the ear. The ringing continued for six months, and he acquired a permanent, sensori-neural high tone loss (Figure 2).

FIGURE 1

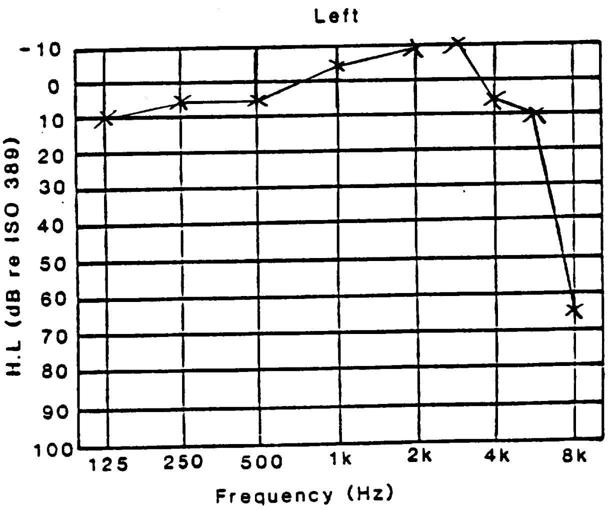
In case one, screening at 20 dB level prior to the dive was normal.



0—0: The day after the incident
X—X: Day three after the incident
•—•: Two and a half months after the incident

FIGURE 2

The hearing in Case Three - seven years after the incident.

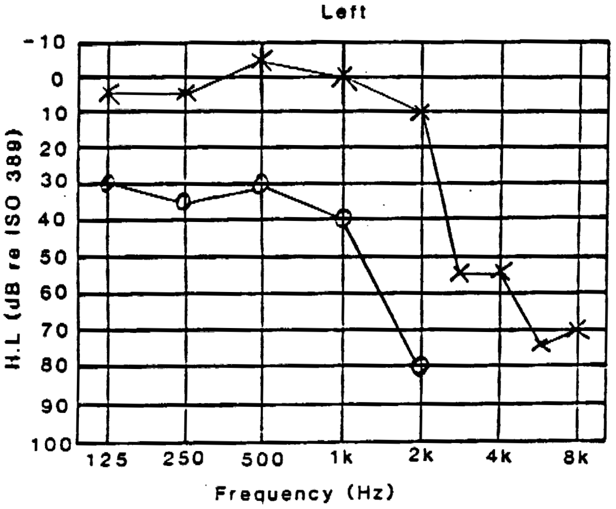


4. A 26 year old female sport scuba diver had, as always, problems with the pressure equalization to her middle ears during an open dive to 20 msw for 15 minutes. During descent she felt pain in her ears, most on the left side, and performed forceful Valsalva manoeuvres. Back onboard the boat she felt unsteady and had to take a broad stance to keep her balance. Her left ear felt plugged with reduced hearing and she had continuous ringing in the same ear. The next day she saw a physician who referred her to an ENT out-patient clinic where marked barotraumatic changes were found in both ears and an

audiogram demonstrated a pronounced sensori-neural hearing loss for all test frequencies in the left ear. Above 2 kHz no hearing was detectable (Figure 3). The speech reception threshold (SRT) corresponded to the pure tone audiogram. Two days later a bithermal caloric vestibular test with ENG demonstrated a left anal paresis. She abstained from further diving, and when last seen five months later she was not dizzy, had no plugged sensation in the ear, the tinnitus (ringing) was reduced and a pure tone audiogram demonstrated markedly improved hearing.

FIGURE 3

The hearing in case 4.



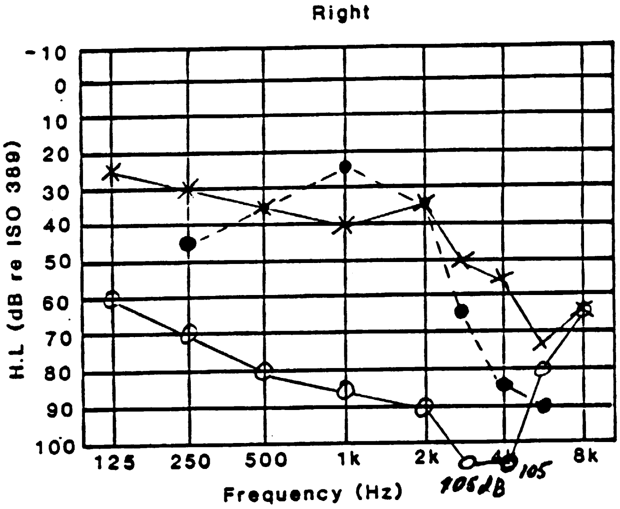
0—0: Two weeks after the dive
X--X: Two months later

5. A 16 year old male sport scuba diver performed an open sea dive in spite of having a common cold. He had equalization trouble and performed forceful Valsalva manoeuvres. He experienced ringing in his right ear, which felt plugged, and he felt dizzy. Since the cochlear symptoms continued he, two weeks later, saw a physician who gave him penicillin and referred him to an ENT out-patient clinic. The tympanic membranes were then normal, but pure tone audiometry revealed a grossly elevated hearing threshold for all tests frequencies in his right ear, and SRT demonstrated a discrimination loss of 60%. Two months later the hearing had improved markedly and there was no longer any discrimination loss. Two and a half years later he was rejected as an applicant for the Navy Academy due to the hearing impairment that by then had again increased. At 8 kHz no hearing could be detected at all (>120dB) (Figure 4).

6. A female sport scuba diver performed, on her 29th birthday, an open sea dive to 19 msw. Prior to the dive she had normal hearing but afterwards she experienced pain and ringing in her right ear and was dizzy. Three days later she saw a physician who found signs of marked barotrauma in her right tympanic membrane and also a severe hearing loss in the same ear (Figure 5). She was given decongestant nose drops. One week after the dive she attended an ENT out-patient clinic where a caloric vestibular test with ENG showed slight directional preponderance to the right. One week later she was admitted to hospital where also a transient, positional nystagmus to the right was detected in the right ear down position. The middle ear was explored surgically and an oval window fistula was detected and closed. Unfortunately the constant tinnitus continued, but both her pure tone hearing and SRT was somewhat improved (20% discrimination loss), also subjectively.

FIGURE 4

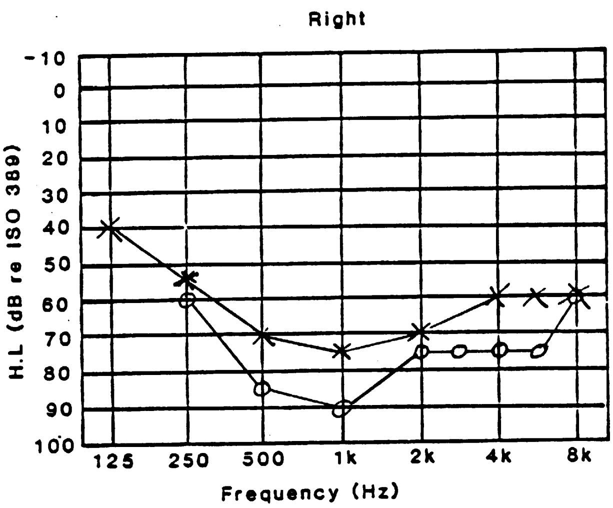
The hearing in case 5.



0—0: Two weeks after the dive
X--X: Two months later
•—•: Two and a half years later

FIGURE 5

The hearing in case 6.



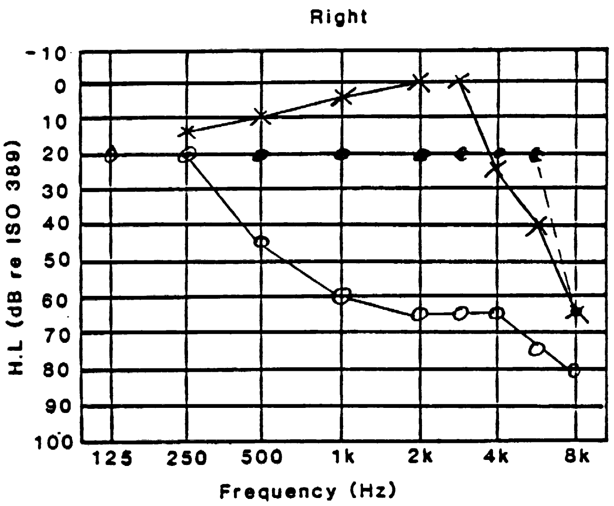
0—0: Three days after the dive
X—X: One month after the dive (12 days postoperatively)

7. A 26 year old male engineer experienced spinning vertigo after playing underwater rugby (breath-hold diving) for one half hour in a 4 metre deep pool. He was unable to fix his gaze which flickered back and forth, he was nauseated and his gait was unsteady. He was seen by a physician and admitted to the hospital ENT department the next morning, where he received an IV drip (500 cc of normal saline). He was discharged symptom free the next evening. He had not experienced any cochlear symptoms, and a control six weeks later showed normal hearing and vestibular function.

8. A 20 year old male Navy diver made a breath-hold dive to 18.5 metres in a submarine escape training tower. During ascent his right ear would not vent properly and upon surfacing the ear felt plugged, the hearing was reduced and he experienced tinnitus. Prior to the dive an audiometric screening at 20 dB level was normal. Three days after the dive an audiogram showed a pronounced hearing loss in the middle and high frequencies (Figure 6) and he was the next day put to bed in the Navy hospital. He had experienced no vestibular symptoms. His hearing improved gradually, and two and a half months after the dive an audiometric screening at 20 dB level was normal except for a drop to 65 dB at 8 kHz. Tinnitus decreased and he was allowed to start diving again. Besides his Navy diving he dived in a civilian scuba club. After four open sea scuba dives, two of them to 50 msw, his hearing was still stable.

FIGURE 6

The hearing in case 8.

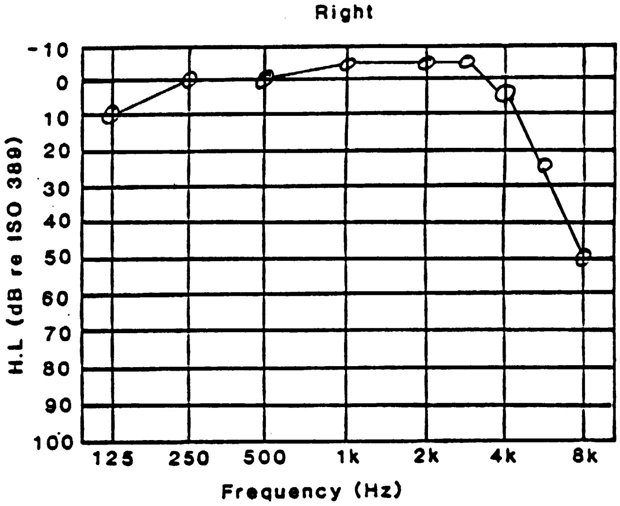


0—0: Three days post dive
X—X: Two months post dive
•—•: Screening at 20 dB level two and a half months post dive

9. A 22 year old male Navy diver, who usually had problems with pressure equalization to his ears in the initial phase of descent, became so eager to chase a fish during an open sea scuba dive that he ignored normal equalization procedures. During the fast descent he felt pain in his right ear, and after coming onboard the boat he had ringing in the ear, which periodically felt plugged, and he was dizzy. He did not see a physician until a week after the dive, and since the cochlear symptoms would not completely clear he was seen by an ENT specialist six months later. Vestibular tests were then normal, but he still had intermittent tinnitus and sometimes a plugged sensation in the ear, and a pure tone audiogram showed a sensori-neural high tone loss (Figure 7). Prior to the dive an audiometric screening at 20 dB-level was normal.

FIGURE 7

The hearing in case 9 six months post dive.

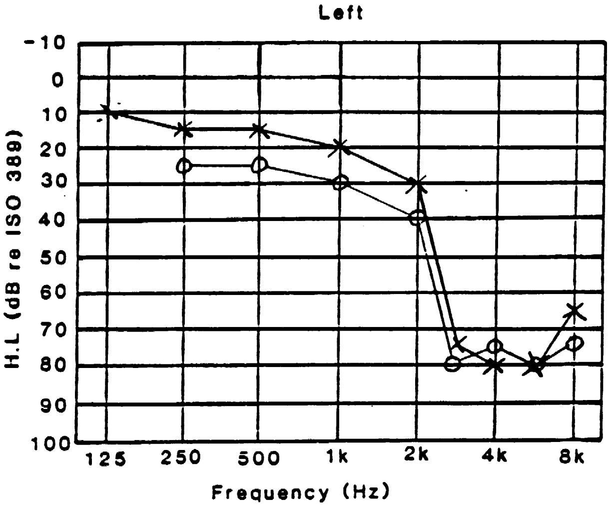


10. A 41 year old male sport scuba diver had his usual trouble with pressure equalization to his left ear when performing three repetitive open sea scuba dives. First he went to 10 metres for a couple of minutes. After five minutes at the surface he descended to 18 metres for 35 minutes. After a couple of minutes at the surface he went to eight metres for 15 minutes. When coming ashore he felt so unsteady he had to hold on to something to prevent falling, and he recognized tinnitus and reduced hearing in his left ear. He did not see a physician until 18 hours later and referral to a diving physician was delayed another day. A severe hearing loss was demonstrated (Figure 8) and he was recompressed according to USN treatment table 6, however without any appreciable effect on his symptoms.

Five days after the dive he was seen in an ENT out-patient clinic where the hearing loss was verified. He was subsequently examined by several physicians who did more extensive audiological and vestibular tests without detecting any new evidence. Three weeks after the dive he had a Stellate ganglion block and received vasodilating medication (Nicotinic acid tablets). This treatment alleviated his balance upset, but had no effect on his cochlear function. His vertigo returned, however, but brain stem evoked response audiometry (ERA) failed to reveal any central lesion. Eventually, two months after the dive, the ear was explored surgically, but no labyrinthine fistula was found. His vertigo fluctuated, but eventually disappeared. His tinnitus also fluctuated, but his hearing loss was permanent and bothered him in conversation with several people simultaneously.

FIGURE 8

The hearing in case 10

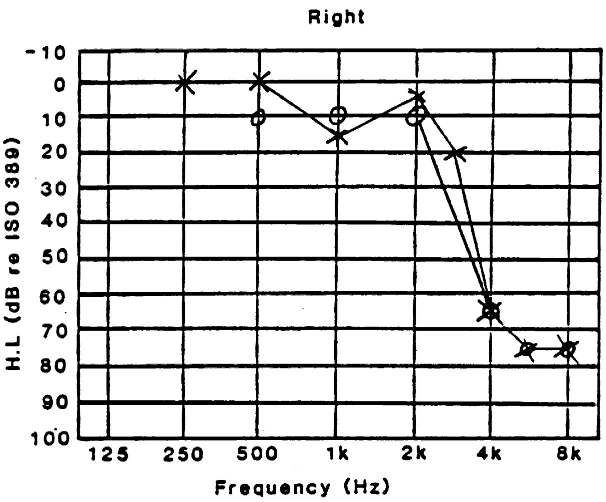


0—0: Two days post dive
X—X: One year post dive

11. A 33 year old man felt dizzy after a dive and had ringing in his right ear. An audiogram revealed a high tone loss which was increased after another dive and became permanent (Figure 9).

FIGURE 9

The hearing in case 11

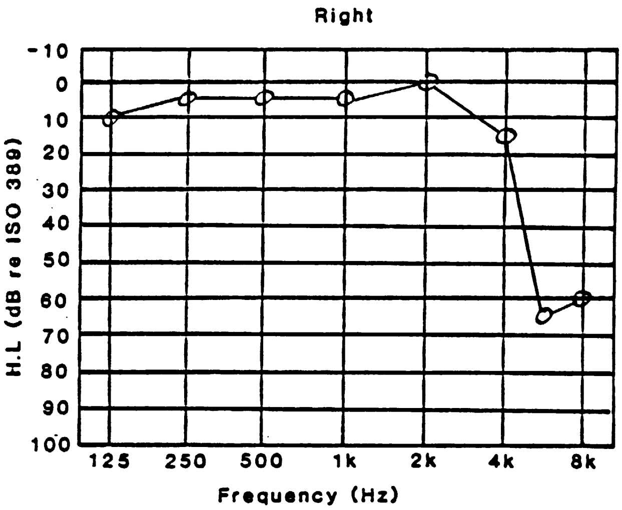


0—0: Second day post dive
X—X: Four years later

12. A 16 year old female sport diver performed an open sea scuba dive to 30 metres. She had problems with the pressure equalization to one of her middle ears due to a slight common cold. She experienced vertigo at the bottom and had problems fixing her gaze. She then ascended slowly, well within the no decompression stop limit, but was unable to climb the ladder to the boat unaided. She experienced spinning vertigo and ringing in her right ear and was unable to walk straight or to keep her balance, and vomited 14 times. The next day she still had spinning vertigo, but was able to walk unaided. She eventually saw a physician who found one of her tympanic membranes red. In spite of admitting that he bad no experience with diving casualties he did not seek advice or refer her to a specialist. Several weeks later she dived again, and again experienced vertigo. After that she stopped diving but still had constant tinnitus. Not until nearly four months after the first mentioned dive was she admitted to an ENT specialist. Her only complaint then was the tinnitus. Caloric vestibular tests were normal, but pure tone audiometry revealed a sensori-neural high tone loss in her right ear (Figure 10).

FIGURE 10

The hearing in case 12 four months post dive



13. A 19 year old male sports diver student performed an open sea scuba dive to 10 m depth. He performed a very forceful Valsalva manoeuvre during descent and experienced severe, spinning vertigo during ascent after a few minutes at depth. He also felt pain in both ears and became nauseated. When coming onshore he was unable to stand and vomited repeatedly. He was recompressed in a chamber without any effect on his symptoms, which actually became worse during decompression. He was then put to bed in an ENT department. Upon admission he was very ill with barotrauma of both middle ears (haematotympanum) and a brisk nystagmus which subsided after bed rest. Audiometry two days after the dive disclosed a sensori-neural high tone in his right ear (Figure 11). He was dismissed from hospital, subjectively well, after three days.

14. A 26 year old male (foreign) Navy diver performed an uneventful chamber dive to 50 msw on air. The day after the hearing in his left ear deteriorated seriously, and three days later he felt unsteady, he was unable to walk straight and bumped into things. He also experienced a high pitched tinnitus in his left ear. He did not see a physician until a week after the dive and was referred to an

ENT specialist who found normal tympanic membranes, but audiometry showed a pronounced sensori-neural hearing loss in his left ear (Figure 12). At 8 kHz the hearing was not measurable with the available equipment (ie. the loss exceeded 110 dB). A caloric vestibular test showed less reactivity on the left side as in a slight canal paresis. He preferred to go back to his homeland for treatment (bed rest, Rheomacrodex IV, medical treatment) and his hearing improved significantly. Decompression sickness could be a differential diagnosis in this case. One can not either completely rule out the possibility of the pure coincidence of a spontaneous hearing loss and the dive.

FIGURE 11

The hearing in case 13 two days post dive.

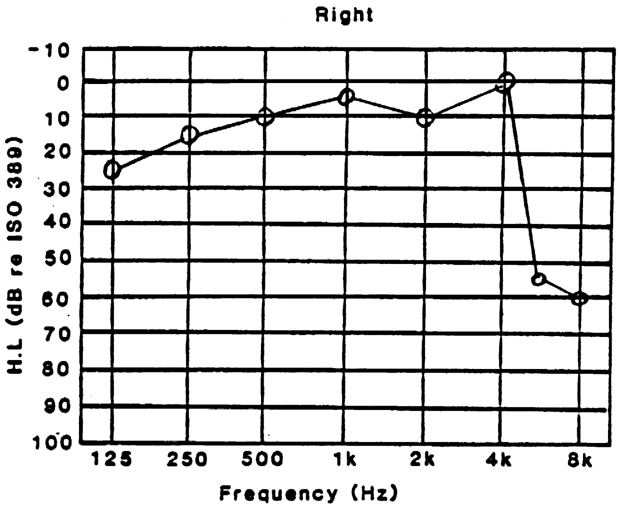
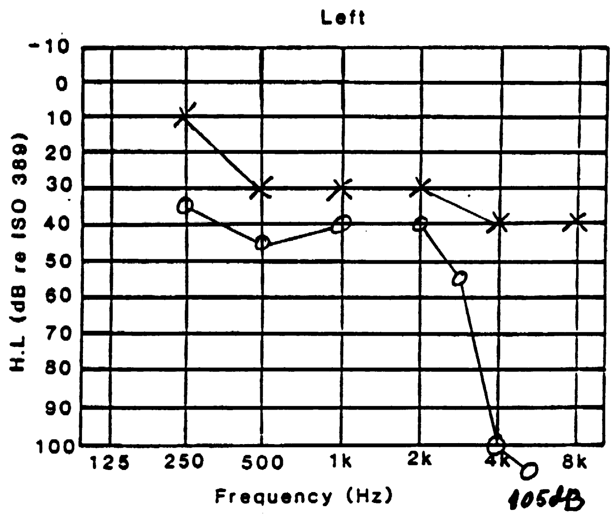


FIGURE 12

The hearing in case 14.

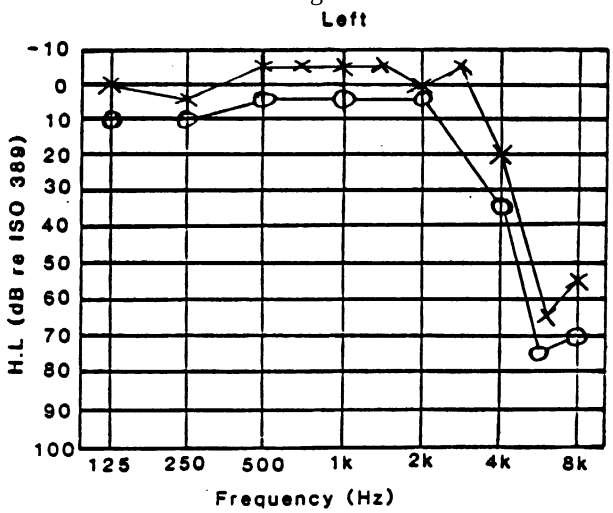


0—0: One week post dive
X—X: Six weeks post dive

15. A 23 year old male sport diving student made an open sea scuba dive to 8 metres. He had difficulties with the pressure equalization to his ears, especially the left, and performed forceful Valsalva manoeuvres. He felt pain in his left ear during descent and ringing in the ear as soon as he took off his gear after surfacing. He had no vestibular symptoms. Since the tinnitus continued, he saw an ENT specialist two days after the dive. The tympanic membranes were normal, as was a bithermal, caloric vestibular test, but pure tone audiometry revealed a sensori-neural high tone loss in his left ear (Figure 13). Prior to the dive he had, audiometrically verified, normal hearing. One month after the dive the hearing had improved slightly, but the tinnitus was unchanged.

FIGURE 13

The hearing in case 15



0—0: Two days after the dive
X—X: One month after the dive

16. A 25 year old male Air Force helicopter pilot performed escape training from a capsized helicopter simulator in the upside down position in a pool at approximately three metres depth. He was under water holding his breath for approximately 30 seconds, and was unable to equalize the pressure to his middle ears. Afterwards he felt pain in his left ear which also felt plugged. He also experienced tinnitus and slight vertigo. Aspirin alleviated the pain, but even after a week the vertigo was bothersome and the tinnitus lasted for two weeks. A couple of weeks after the incident he was seen in an ENT out-patient clinic where his tympanic membranes were found normal. A transient positioning nystagmus to the left on head rotation to the right was recognized, but hot water (44°C) stimulation gave symmetric response. Unfortunately cold water stimulation was not performed nor audiometry. After a few weeks the pilot became symptom free. I think the cause of his cochleo-vestibular symptoms was inner ear barotrauma.

17. A 19 year old male professional diver, who doubled as a sport scuba instructor, performed ten consecutive open sea dives to 10 metres, each of approximately nine minutes duration, in order to familiarize student divers

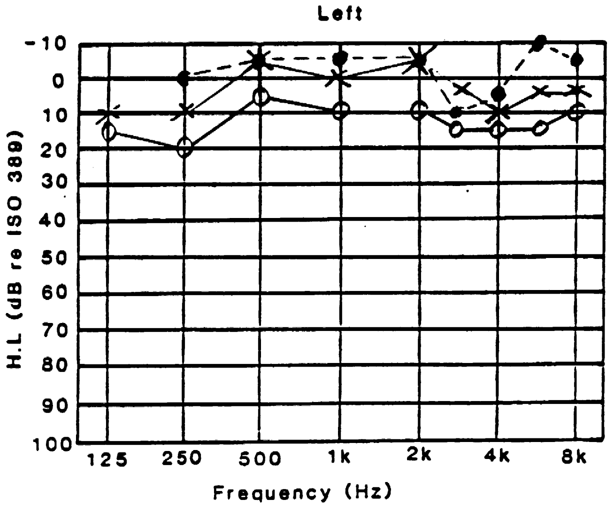
with open sea diving. The tenth ascent was rather fast, and at three metres depth he suddenly experienced a sharp pain in his left ear. He loosened the line to the student who surfaced while he himself returned to the bottom. There the pain was somewhat relieved, but he felt slightly dizzy. After a few minutes he ascended slowly without any change in the symptoms, but he had to sit down as soon as he came ashore due to vertigo.

He was also slightly nauseated. Later he felt a tender swelling behind the left ear and describes crepitation on pressing against it. The skin over the swelling was red. The ear felt plugged and subjectively his hearing was reduced on that side. The next day he saw an ENT specialist who examined him otologically and otoneurologically. There was a slight elevation of the hearing threshold as compared to a recording taken six months previously. The loss was sensori-neural. (Figure 14. Bone conduction has been omitted for clarity). Bithermal caloric vestibular tests revealed a left sided canal paresis. A chest x-ray was negative. He was reported sick for two weeks and put to bed with elevated head. The only medication was aspirin. He was not allowed to dive until after a control two months later. He then felt fit and healthy and the vestibular control test suggested an overcompensation of a peripheral injury on the left side. After a supervised pressure chamber test he was allowed to resume diving.

Three and a half months later he performed two repetitive open sea dives to 12 metres for nine minutes and to approximately 27 metres for 20 minutes (in the "wrong" sequence: shallowest first) in spite of having a common cold. The first dive was unremarkable, but during the second descent he felt pain in his sinuses and his left ear

FIGURE 14

The hearing in case 17



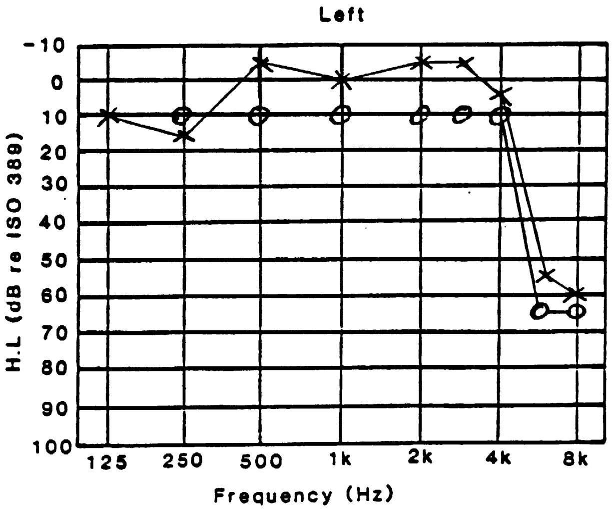
•—•: Pre dive
0—0: First day post dive
X—X: One week post dive

cleared less easily than the right. On the bottom he experienced spinning vertigo, and after surfacing he felt nauseated and vomited once. The next day he vomited four times and saw a physician familiar with diving. He received decongestant nose drops and tablets plus anti-vertiginous tablets and was put to bed. Three days later he still felt slightly dizzy and vestibular testing showed a left canal paresis, slightly worse than after the first injury. He had no cochlear symptoms and an audiogram was close to the pre-dive recording. He was prohibited from diving for six weeks and advised not to dive during respiratory tract infections.

18. A 20 year old male Navy scuba diver experienced barotrauma of his left ear while wearing a full face mask in an open sea dive. In connection with the injury he felt a slight ringing in the ear. Prior to the dive his pure tone audiogram was normal, but a routine control five months later revealed a sensori-neural high tone loss in the affected ear, where he still experienced a high pitched tinnitus while in silent surroundings (Figure 15). Three years later he was accepted as a student at the Norwegian Government Diver Training School in order to get into commercial deep diving, in spite of the fact that his reported high tone hearing loss was permanent. The hearing in his right ear was normal, though.

FIGURE 15

The hearing in case 18.



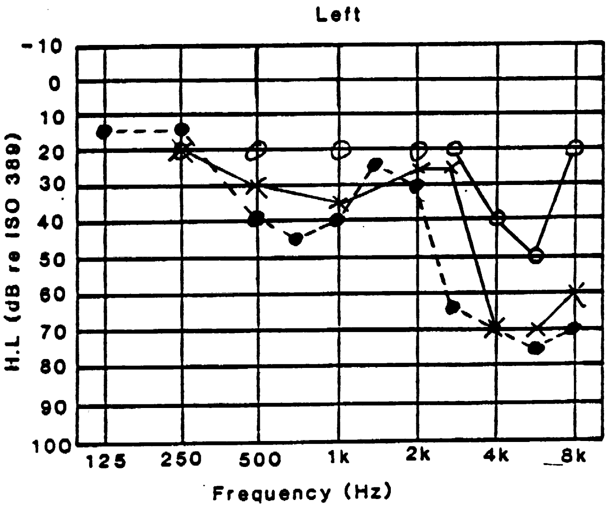
0—0: Five months after the dive
X—X: Three years later

19. A 22 year old man attending a course in sport scuba diving as a leisure time activity during his military service performed an open sea dive to 15 metres for eight minutes. As usual he had equalization problems and performed forceful Valsalva manoeuvres. He did not experience anything peculiar until he came into the silence of his bedroom nine hours after the dive when he recognized a high pitched ringing in his left ear. The tinnitus fluctuated for some days, but eventually became constant. Nine days after the dive he finished his compulsory military service.

A medical examination in that connection revealed an elevation of the hearing threshold in his left ear as compared to the audiogram taken when he entered the military services, but even then the 20 dB level screening had revealed a, probably noise induced, high tone notch in the audiogram from his left ear. He was referred to an ENT specialist who confirmed the hearing loss as sensori-neural, but except for that found normal ears, including vestibular function as tested with bithermal stimulation and ENG. At that time bed rest was tried, but seven months later the hearing had deteriorated significantly at 3 kHz (Figure 16).

FIGURE 16

The hearing in case 19.



0—0: Pre dive screening at 20 dB level
X—X: Nine days post dive
I—I: Seven months later

20. A 37 year old male engineer using diving in his work for underwater inspection and non-destructive testing (NDT) performed an open sea dive to 15 metres depth in spite of having a common cold. He had equalization problems and thus had to perform equalization manoeuvres forcefully and more frequently than usual. After the dive his left ear felt plugged and the hearing in that ear was much reduced. He saw a local physician who reassured him, and when he saw an ENT specialist six months later a sensori-neural hearing loss in the left ear was confirmed (Figure 17).

21. A 29 year old male Navy physician performed a 90 metre dry chamber dive on air. He had difficulties with the pressure equalization to his ears and performed Valsalva manoeuvres repeatedly. Prior to the dive he had no cochlear symptoms and an audiometric screening at 15 dB level was normal. After the dive his left ear felt plugged with reduced hearing and tinnitus, and an audiogram demonstrated a most unusual pattern with a predominantly low tone sensori-neural loss, indicating a lesion in the apical part of the cochlea (Figure 18). He still has tinnitus and hearing loss in that ear 16 years after the incident.

FIGURE 17

The hearing in case 20 six months post dive.

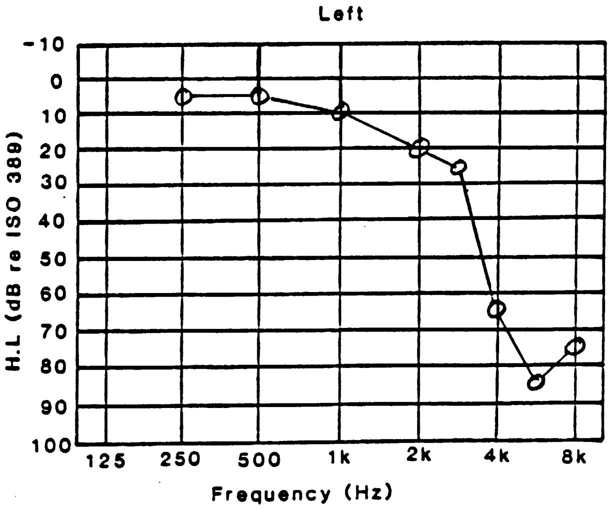
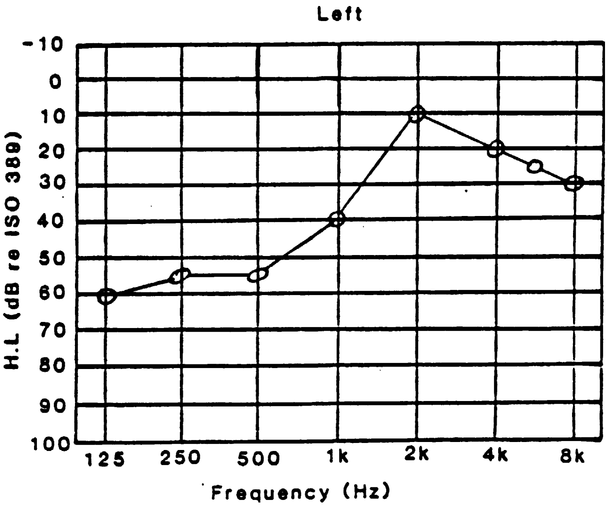


FIGURE 18

The hearing in case 21 immediately after the dive. Pre dive screening at 15 dB level was normal.

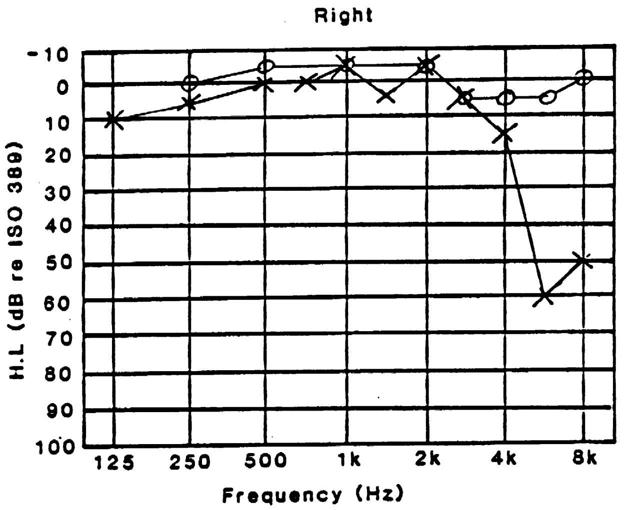


22. A 28 year old male commercial diver lost consciousness at 45 metres depth in the sea wearing a band mask and breathing nitrox. The reason for the loss of consciousness was probably oxygen poisoning. He was taken to the surface and recompressed in a chamber where he woke up at 50 metres depth without any cochlear symptoms. During the decompression his right ear would not vent properly and became painful. He also experienced tinnitus in that ear. Prior to the dive an audiogram demonstrated normal hearing, while a control six months after the dive disclosed a sensori-neural high tone loss (Figure 19). He then still had tinnitus.

During a dive several years before the described incident he had pressure equalization problems to his ears and experienced tinnitus in both ears for a month after the dive. That episode resulted in a permanent, sensori-neural high tone loss in his left ear (Figure 20).

FIGURE 19

The hearing in the right ear of case 22

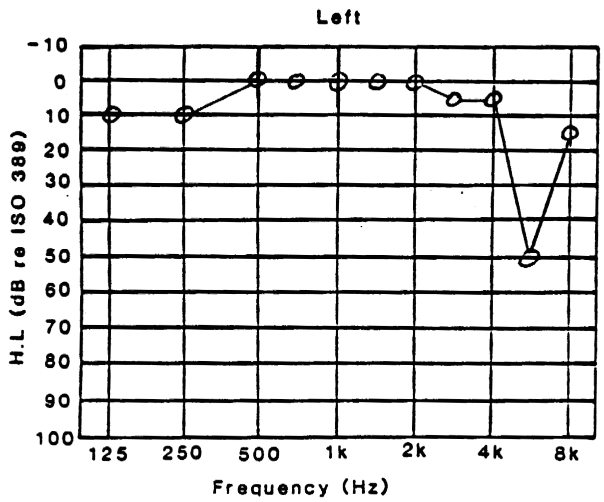


0—0: Prior to the incident
X—X: Six months after the incident

23. A 24 year old male scuba diving student performed a breath-hold dive to 7 metres in the sea. He had pressure equalization trouble, and since he was wearing too little lead he had to use his arms to swim down and was thus unable to perform equalization manoeuvres. A painful

FIGURE 20

The hearing in the left ear of case 22 several years after a different dive.



pressure differential built up in his ears. Three hours after the dive he became dizzy. The next morning he felt fine and did a scuba dive to 12 metres. He descended slowly and managed to equalize the pressure to his ears. A couple of hours after the dive he again felt dizzy.

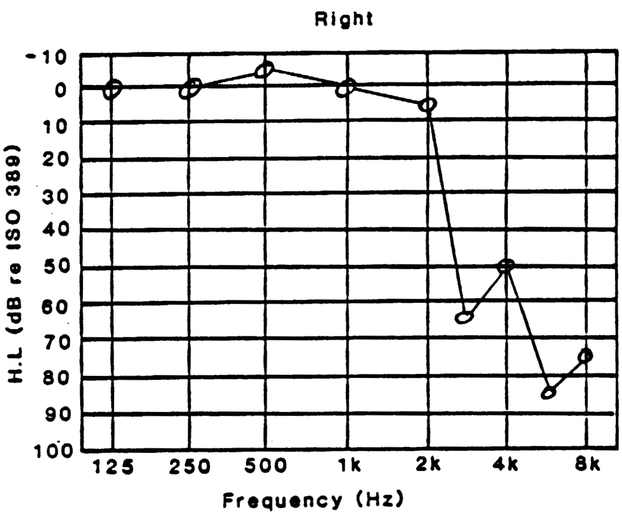
The next morning he again was fine and performed another scuba dive to 12 metres. He had some equalization trouble and experienced vertigo immediately after the dive of the same nautical nature as on the two previous days, but stronger. The symptoms fluctuated for the next couple of days, so he saw a physician who referred him to an ENT specialist on the third day after the last dive. Throughout he had had no cochlear symptoms. Barotraumatic changes were observed in both tympanic membranes, but a bithermal caloric test and ENG did not at that time detect any vestibular injury. Nevertheless the fluctuating symptoms continued and interfered significantly with his university studies. This was still the case two months after the dive, which is the last available information on this case. Eleven years before he had experienced vertigo and intolerance to high sound levels after head trauma. It is difficult in this case to pinpoint a possible lesion to the inner ear, but there seemed to be a definite connection between the diving and the symptoms, and the barotraumatic changes in the tympanic membranes were unmistakable and thus suggestive of inner ear injury as the cause of his symptoms.

24. A 36 year old male sport scuba diver performed approximately seven dives to a maximum of 15 metres in the sea within an hour. Before the dive he had no cochlear symptoms, but the morning after he woke up with a high pitched tinnitus in his right ear. Since that did not clear, he saw a physician a couple of days later, and the next day recompression to 50 metres in a chamber was tried without effect on his symptoms. He was seen regularly by an ENT specialist for months, but even eight years after the incident he still had constant tinnitus and sensori-neural high tone loss (Figure 21). In this case the relation to diving could be just coincidence since spontaneous hearing loss of this kind occurs now and then. A realistic differential diagnosis in this case is also decompression sickness.

25. A 27 year old male Navy diver performed approximately 20 consecutive breath-hold dives to about seven metres as an instructor in a submarine escape training tank. He did not care much about pressure equalization to his ears, and after a while he experienced a plugged

FIGURE 21

The hearing in case 24 eight years after the incident.



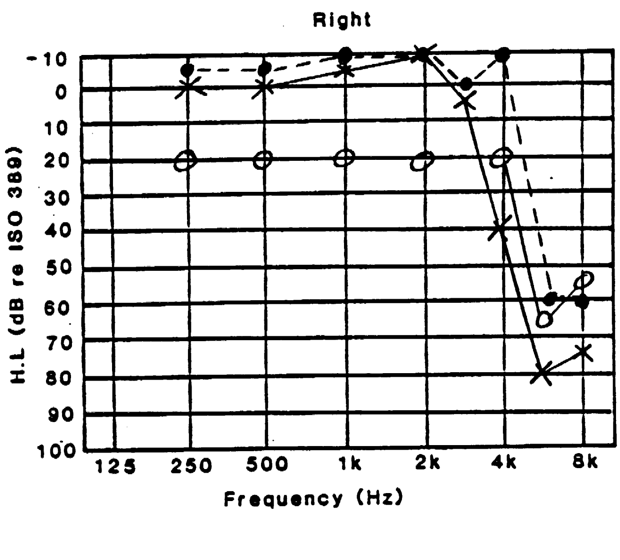
sensation, reduced hearing and tinnitus in his right ear, plus vertigo. Nevertheless he continued until the class was through, but afterwards he was unable to walk straight. One year previously he had experienced similar phenomena from his left ear after he had swum 50 metres underwater, but in that case all symptoms cleared gradually. He now expected the same course and thus postponed his visit to a physician until the next day.

He was then immediately referred to an ENT specialist who found spontaneous nystagmus of the second degree to the left, indicating the right sided canal paresis. The patient complained of pulsation and constant tinnitus in the ear, and an audiogram revealed an up to 20 dB deterioration of his high tone hearing on the right side where the hearing already was substantially reduced in the high tone range, probably due to gun-fire (Figure 22). No sign of middle ear barotrauma was found. He was put to bed and gradually improved. Five days later he had no vertigo, nor nystagmus, and the hearing had improved both subjectively and by audiometric evaluation, at 4 kHz as much as 50 dB. The high tone loss was back at pre-dive level, but caloric vestibular tests and ENG now indicated a stronger cupulo-ocular caloric response on the right than on the left side. Six months after the incident the diver was symptom free, the hearing had stabilized at pre-dive level, and bithermal caloric vestibular tests with ENG showed symmetrical and normal responses. He was then allowed to resume diving provided that he did not dive during common colds and that he was very conscientious with the pressure equalization to his ears. He was also advised not to take the surface position during submarine escape training runs.

Three weeks later he violated all the above mentioned precautions and performed 45 breathhold dives from the surface to approximately seven metres, in spite of having a common cold. Afterwards he experienced severe vertigo and nausea. He was pale and could not walk straight. He

FIGURE 22

The hearing in case 25



0—0: Predive screening at 20 dB level
X—X: One day post dive
•—•: Six days post dive

felt a forceful pulsation in his right ear and slight, periodic tinnitus, but no subjective hearing impairment. When seen by an ENT specialist the next day his only complaint was the pulsation in his right ear. There was no significant change in hearing and no nystagmus. It was speculated that he might have a weak spot in one of the labyrinthine windows where a fistula would open when the pressure differential reached a certain level. He was reported sick for a week and taken off diving for a couple of months. A bithermal caloric vestibular test with ENG was then symmetrical and normal.

26. A 23 year old male sport scuba diver with approximately five years diving experience performed an open sea dive to 25 metres for 25 minutes. As usual he had pressure equalization trouble to his ears, mostly in the right, and performed Valsalva manoeuvres. During ascent he experienced pain in the back of his neck and head. Ten to 15 minutes after coming onboard the boat he experienced spinning vertigo and had to hold on to something to keep his balance even while sitting. The symptoms increased, he was unable to fix his gaze and vomited all the time. When he came ashore 20 minutes later he saw a local physician in general practice who referred him to the neurology department in the local hospital where electroencephalography (EEG), and skull X-ray were negative.

He was the next day transported by helicopter while breathing oxygen to the nearest Navy base where he was recompressed after a prolonged US Navy Table 6. Subjectively the treatment had no effect. Nevertheless he recovered gradually, but was unable to walk straight for one week after the dive. He had no cochlear symptoms. When seen by an ENT specialist two months later he was symptom free, but would still experience some unsteadiness in certain situations, such as when heading the ball during football games. He had not been diving since the incident. Otological and otoneurological examination was unremarkable, except for a sensori-neural high tone loss in his left ear that was present prior to the dive, which was probably caused by gunfire. He recalled having had tinnitus for a couple of days after hunting some time before. Bithermal caloric vestibular tests with ENG were normal.

27. A 45 year old male marine biologist, previously a Navy diver, now a sport scuba diver, performed approximately ten scuba dives to 5 msw for a total duration of approximately half an hour. He had to perform Valsalva manoeuvres to equalize. Immediately on coming onshore he experienced vertigo and unsteadiness and he was slightly nauseated. He also had a constant tinnitus in his right ear. When seen by an ENT specialist one and a half hours later he walked unsteadily, would grasp onto objects to secure his balance and tended to keep his neck in a fixed position. In spite of this overt symptomatology nothing objectively could be found on otological and otoneurological examination. Even a bithermal caloric vestibular test with ENG was judged to be within normal limits (even though a slight right-sided canal paresis could not be completely ruled out). The only difference in the audiogram compared to one done a year previously was a marginal 10 dB drop at 8 kHz (from ten to twenty dB). He was put to bed and the tinnitus disappeared the same evening. He kept his bed

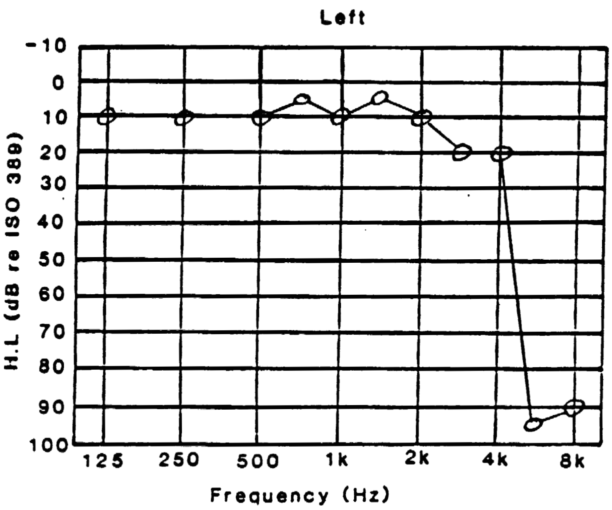
for a few days but had fluctuating tinnitus for periods and fluctuating nausea for approximately three weeks. He was symptom free when seen six weeks after the incident. A caloric test was then completely normal. He then recalled that he had been dizzy after a similar dive seven years previously and also nauseated after that type of diving four years earlier. Maybe a case with such sparse objective signs should not be classified as inner ear barotrauma, but I believe that more sensitive test methods might have proved the diagnosis.

28. A 19 year old man practised underwater rugby (breath-hold diving) in a four metre deep pool. After the game his left ear felt plugged and he had tinnitus for six months. When seen by a physician some days after the incident blood and fluid was found in the left middle ear. His audiogram was normal when reporting for military service prior to the incident, but when entering the military services after the dive a high tone loss was found in his left ear. The sensori-neural loss was confirmed four years later (Figure 23).

29. A 25 year old male scuba diver with six year's experience had for a couple of months experienced severe spinning vertigo for five to ten minutes after diving. Once it caused him to swim in a circle to the left when trying to swim ashore. Eventually, after a dive to four metres depth

FIGURE 23

The hearing in case 29 four years after the dive.



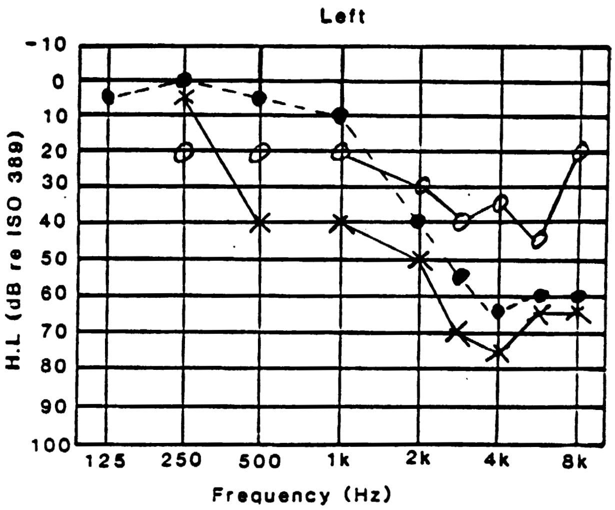
he was unable to stand up when coming ashore. He fell to the ground, was unable to fix his gaze, was very nauseated and all but vomited. He had no cochlear symptoms. The symptoms subsided in a couple of hours, but when seen by an ENT specialist two months later a bithermal caloric vestibular test with ENG revealed a centrally compensated left-sided canal injury. Except for that, an otological examination was normal, including audiometry. At that time he was symptom free. Inner ear barotrauma, possibly with a transient peri lymphatic fistula, could explain the described symptoms.

30. A 29 year old male sport scuba diver with nine years experience participated in breathhold underwater rugby and descended repeatedly to five metres depth without caring about pressure equalization to his ears. In the middle of the game his left ear suddenly felt plugged and he experienced a continuous, high pitched tinnitus. There was no vertigo. He had experienced middle ear barotrauma on the same side twice before and thought this was the same thing. But his hearing deteriorated substantially in the course of the following two days. Then the condition started to improve and he did not see a physician until a week after the incident and was immediately referred to an ENT specialist who found his hearing significantly reduced compared to pre-dive audiograms (Figure 24). He was admitted in hospital and put to bed in the ENT department where his hearing continued to improve, so he was released after a week. Three weeks after the incident the improvement seemed to have halted, and more than four months after the incident the hearing threshold was still significantly higher than prior to the incident. Even a year later he had constant tinnitus and some problems with holding a conversation in background noise. Nevertheless he wanted to take up diving again. His marked pre-dive hearing loss was probably caused by noise exposure in mechanical industry where he had worked without using ear protectors.

31. A 30 year old male sport scuba diver performed an open sea dive to 26 metres for 26 minutes. The dive was unremarkable and he had no pressure equalization trouble to his ears. Four hours after the dive his right ear felt plugged, and the next day an ENT specialist found a

FIGURE 24

The hearing in case 30.



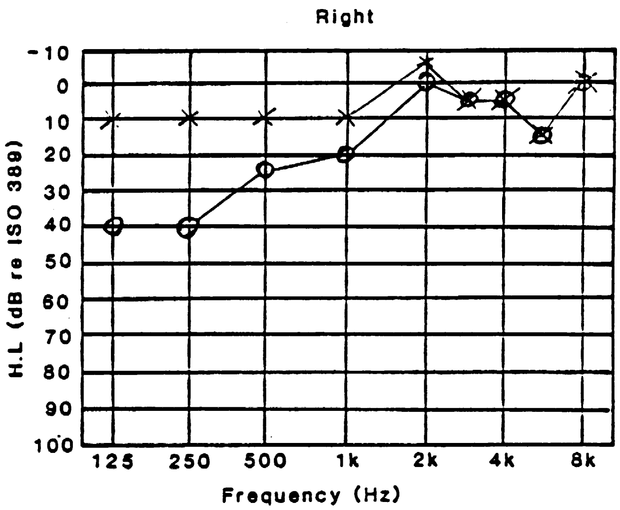
0—0: Pre incident screening at 20 dB level
X—X: One week post dive
•—•: Four months after the dive

sensori-neural low tone loss in that ear (Figure 25). The day thereafter he also experienced tinnitus in his right ear and the next day (3rd day after the dive) he received hyperbaric oxygen therapy (HBO) (USN Table 6) which had no subjective effect, while the audiogram showed some improvement. The next day he got more HBO (USN Table 5) which traded his tinnitus for slight vertigo, but again the audiogram indicated improved hearing. The day after his ear felt plugged again after a lot of shouting as a coach for a volleyball team, and the tinnitus was back. Two days later the hearing had normalized subjectively and audiometrically and he had no more tinnitus or vertigo.

This man was diving a lot so one may argue that the described symptoms occurred in time relation to a dive only by chance and that there was no causative relation, especially since he had no equalization trouble. Thus it could have been an early manifestation of Meniere's

FIGURE 25

The hearing in case 31.



0—0: First day post dive
X—X: One week post dive

disease (hydrops labyrinthi), completely unrelated to the dive, or precipitated by it. Decompression sickness (DCS) is unlikely, but cannot completely be ruled out. On the other hand, diving can cause considerable pressure differentials in the ears before a diver cares about equalizing, especially if he is distracted or busy with other tasks. So even if it may be speculative I think that also this case can be classified as inner ear barotrauma.

DISCUSSION

Even though information about dive depth is missing in five of the cases, we can conclude that this injury most often occurs at shallow depth, not surprisingly since that is where most of the diving is taking place, and also where the relative pressure/volume differences are greatest. As might be expected, the majority of the cases (45.2%) were sport scuba divers, who also perform the majority of the diving in shallow waters. Commercial divers comprised 16.1% of the total and Navy scuba divers 6.5%. The rest were divided between breath-hold and chamber diving, and in one case the information was missing. In 61% of the cases the incident happened during air scuba diving, but data were missing in two cases. The sex difference, 90.3% male and 9.7% female cases, probably more reflects the composition of the diving community rather than anything about sex difference in susceptibility.

The delay before the diver sought medical advice varied from hours to "indefinitely", which means that the diver did not seek advice but was found by chance during routine examinations months after the incident. More than 70% reported problems with pressure equalization to the ears and close to 20% admitted to have dived in spite of a common cold when the incident occurred. Close to 26% reported to have performed forceful Valsalva manoeuvres (but data were missing in approximately 13%).

No consistent way of therapy can be traced in this material. More than 35% received no therapy at all, and data are missing in three cases. In the rest, several types of treatment were tried; bed rest, medication, IV fluid, HBO, Stellate ganglion block and ear surgery.

The lay off time before diving again varies considerably and information is missing in several cases, but in only four cases is positive information available that they stopped diving permanently. Approximately 65% experienced permanent cochlear injury, leading in one case to rejection by the Naval Academy. In a commercial diver the incident caused severe economic problems. Ten of the cases were only cochlear (two right, seven left and one bilateral), five only vestibular (one on each side and three probably bilateral) and 16 both cochlear and vestibular (ten right, six left). Thus 13 were only right-sided 14 only left-sided and four bilateral. Only three of the ears were explored surgically (two right, one left) and in only one of them was a fistula found, in the right oval window.

SUMMARY

Barotrauma of the inner ear in 31 cases (35 ears) is reported. Inner ear barotrauma is no rarity, but is under-reported. It most often occurs in shallow water. Permanent cochlear injury is common, which can seriously affect a person's choice of profession and cause social and economic problems. Of those who resumed diving some had recurrences while others were stable throughout the observation period.

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ATYPICAL DECOMPRESSION SICKNESS

THE DIAGNOSTIC CHAMBER

Harry F Oxer

A 23 year old female, otherwise very fit, was scuba diving on Sunday 11th November in 80 feet of water for 30 minutes. She was spear fishing, not very energetically, the water was warm but with poor visibility, no current, and she was diving alone. She had logged about 25 dives since her training course. Her ascent was slow and controlled, slower than the bubbles.

Some 5 to 6 hours after the dive she complained of abdominal pain which became progressively more severe, and was described as cramping and deep inside the abdomen. Some days later, she described that the pain had in fact been in circular, fairly narrow bands around the upper abdomen, but this was not the story listed at the time.

The pain lasted for the rest of that day and most of the night. The next day (Monday), she developed a right sided stiff neck, with increasing muscle spasm, as though she had slept badly. Later that day she developed a severe headache, described as a tight band rotating around her head. She put up with this for the whole of the day, but presented at a major hospital emergency department at 2300 hours.

On examination there were no positive findings except this history. Following a telephone consultation to Fremantle Hospital, the centre for management of diving cases in Western Australia, she was transferred there and seen at 0100 hours on Tuesday morning.

There were no abnormal neurological or other findings, except that the patient had a stiff neck and some headache. Because of the long delay before any symptoms came on, the relatively short dive, the non-specificity of the symptoms, and the fact that they were improving, it was decided to observe the patient overnight. Later that morning, Tuesday 13th November, in working hours, she still felt unwell and still had a stiff neck and headache. There was now no abdominal pain. It was decided that this could be decompression sickness and she was transferred for a trial of recompression at HMAS Stirling.

She was recompressed on a Table RN62, and her pain and discomfort were halved by arrival at depth, and by ten minutes there was little pain. She was totally normal after two hours. She was treated with an extended Table RN62, intravenous Hartmann's solution totalling 5 litres, and the Royal Australian Navy routine of Dexamethasone, 16mgms IV stat, then 8mgms eight hourly. She complained of a very unpleasant whole body sensation of pruritus "from the inside" immediately following each injection of intravenous Dexamethasone. On completion of recompression, she was given 100% oxygen for alternate hours overnight.

On Wednesday 14th November she was returned to Fremantle Hospital at 1800 hours, complaining of some chest discomfort and tightness, almost certainly due to the high oxygen dosage. Her IV therapy was continued until late evening, then discontinued, and the final dose of Dexamethasone was given. By the next morning she was fully fit and was discharged with a certificate for work, and advised to give up diving as she appeared to have a sensitivity to, even by average standards, a small nitrogen load.

To our surprise, the patient returned the same evening with a recurrence of the pain in the right side of the neck, and the headache. She was recompressed that night at 2200 hours on RN Table 61, and the symptoms resolved slowly during that recompression.

The next day (Friday 16th November) she had a minor recurrence of symptoms and was recompressed, again on RN Table 61, when the symptoms cleared.

She was discharged home on Saturday 17th November 1984, with no further symptoms.

Thursday 22nd November, the patient presented with a recurrence of pain across both shoulders, which she described as similar to the previous pain. She was recompressed at 1700 hours on RN Table 62, with initial good, but not complete, resolution of discomfort. However, on decompression to the surface, her pain recurred in the back and shoulders.

On Friday 23rd November, now 12 days after the original incident, she was recompressed to 5 metres of sea water on 100% oxygen for two hours, with complete resolution of the pain. There has, to date, been no recurrence.

COMMENT

This case was unusual for several reasons. The symptoms occurred following a relative small exposure which was within the tables (80 feet of water for 30 minutes). It was not a particularly energetic dive, and one that she had done a number of occasions before. The symptoms did not come on for 5 to 6 hours, and were not very typical. With hindsight, any symptoms following a dive, and occurring up to 24 hours, are decompression sickness until proved otherwise.

However, her symptoms were so non-specific, and could have been associated with intercurrent viral illness which was prevalent at the time, and were getting better. When this was added to presentation at 0100 hours, the decision to await recompression until the morning seemed reasonable at the time.

It is well known that if the management of decompression sickness is not commenced until a considerable time after the onset of the problem, treatment is more difficult and recurrence more likely. This patient did not present until almost two days after the dive. Her recurrences stretched unusually long and though her symptoms were very non-specific, they did respond to recompression.

Perhaps this case again illustrates the fact that anything, after a dive is decompression illness until proved otherwise, and you may never be able to prove otherwise! Symptoms that respond to recompression, almost by definition, confirm the diagnosis.

UNSCRAMBLING HELIUM SPEECH

Microelectronics Group Implements Prototype in Bit-Slice Technology

Offshore operations need divers, and there is no prospect of divers being totally replaced by subsea completion systems or ROV's for many years to come. At depth, divers have to breathe gas at a pressure equivalent to the hydrostatic pressure of the water around them. Unfortunately, beyond about 50 m depth, oxygen and nitrogen would be lethal in the proportions in which they are normally found in air, so most of the divers' breathing gas mixture consists of the biologically inert gas helium.

As we might expect, using helium also has its drawbacks. Among the most serious of these is that the gas seriously distorts speech sounds; 'Donald Duck with a bad cold' is how divers' speech is usually described. Unfortunately, this is not simply an aesthetic problem; when intelligibility falls to 10-20%, as it does at 300m and below, both safety and efficiency are seriously compromised.

The Norwegian Underwater Technology Center (NUTEC) has implemented a mathematical model of the processes needed to 'unscramble' the distorted sound of speech in helium. This model has been successfully used in test dives at NUTEC. Running this algorithm, however, requires a great deal of computing power, and the computer itself takes up a lot of space. NUTEC therefore engaged the microprocessor applications group at CMI to develop a microprocessor-based real-time hardware version of the algorithm. Additional support for the project was provided by Vesta, a major Norwegian life insurance company.

To handle this project, CMI installed a VAX 11/730 mini-computer with 165 Mbyte disc capacity, A/D and D/A boards, and a bit-slice 1/0 processor to increase sampling speed. The large disc capacity is needed because sampling at high frequency demands huge amounts of memory. A special software package called ILS was also acquired to perform the functions of signal editing, digital filtering, signal processing, etc.

With this combination of hardware and software the CMI team have simulated and modified the NUTEC model, tried out new algorithms, and simulated (in Fortran 77) the hardware version while this was being developed. The long series of operations performed by the model is too complicated to illustrate here. In principle, the first stage is to reverse the sound distortions in the vocal tract which are caused by a given gas mixture under given atmospheric conditions; thereafter a normal vocal tract response is simulated. This of course is simpler to describe than to do.

Following simulation and testing, the microprocessor team designed a hardware system with the same performance as the hardware model but capable of unscrambling speech in real time. The system is designed according to a master/slave principle; the master administers the processing routines and 1/0, while the slave will be a series of bit-slice elements operating as a fast number-cruncher which performs complex processing on commands from the master. A 16-bit data format is used, except in the A/D and D/A converters, where 12-bit precision is acceptable.

The hardware unscrambler is being constructed on a set of three four-layer printed circuit boards, which because of their complexity had to be laid out by computer. Components are now being put into the boards, and the unscrambler will be debugged and tested on an advanced microprocessor development system. When completed the prototype will be tested using recorded speech from deep dives at NUTEC.

This unscrambler will be the result of cooperation by specialist groups at two applied research institutes, a university department of phonetics, and a manufacturer. It will be produced by the Norwegian electronics company Data Instrument A/S.

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MEDICAL PROBLEMS OF SATURATION DIVING

A four-year study of medical problems during a major diving company's saturation diving operations is summarized below. The favourable figures are attributed to aggressive control of the environment (pre-dive checks that rule out incipient infection, etc.; frequent and regular diving complex cleansing) and the encouragement of early reporting of any unusual symptoms or signs of illness or disease. The saturation storage system remained under pressure for up to six months at a time.

COMPLAINT	No. of Cases Reported	Isolated and Decom-pressed
Skin infection (spots, boils)	19	0
Indigestion/nausea/vomiting	14	2
External Otitis	14	1
Hot water suit burns	12	0
Headache	12	0
Pulmonary Oxygen Toxicity	11	0
Athlete's Foot	10	0
Laceration	8	0
Upper Respiratory symptoms	7	3
Dental pain	5	0
Skin infection (rash)	5	0
Diarrhoea	5	0
Abrasion	4	0
Friction burns	4	0
General malaise	4	0
Hypoxia	3	0
Low back pain	2	0
Jellyfish stings	2	0
Puncture wound	2	0
Urinary tract symptoms	2	1
Elevated $CO_2$2	0	
Decompression sickness (post sat. decomp.)	2	N/A
Decompression sickness (post excursion)	1	N/A
Sinus pain	1	0
Total	**151**	**7**

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DIVING SAFETY MEMORANDA

Department of Energy,
Diving Inspectorate,
Thames House South,
Millbank, London,
SW1P 4QJ.

DIVING SAFETY MEMORANDUM 5/1984
DIVING SYSTEMS/CONTAMINATION

27 July 1984

Your attention is drawn to the problem of contamination of the divers' environment which can result from the failure to completely purge the system after cleansing on commissioning.

For example, freon, which is often used as a cleansing agent, can collect in pockets within a diving system and if disturbed by such as ship movement can contaminate the divers' environment.

Experience has shown that it is unlikely that an entire system can be cleansed and purged of the cleansing agent in one operation without leaving pockets of the cleansing agent. It is advisable to cleanse a system in sections.

The Association of Offshore Diving Contractors (AODC) will shortly issue technical guidance covering this problem.

DIVING SAFETY MEMORANDUM 6/1984
MAN RIDING TUGGER WINCHES - MINIMUM CRITERIA

August 1984

The following criteria should be applied by the competent person testing or examining man riding winches.

1. Winches in this category must be certified by a competent person as suitable for the task

2. The winch operating lever should automatically be returned to neutral on release in any operating position.

3. An automatic brake should be fitted that will apply automatically whenever the operating lever is returned to neutral or on loss of power.

4. In the event of failure of the automatic brake mechanism a secondary means should be provided to prevent the load from falling. This may be manual in operation and should be simple in design.

5. A clutch capable of disengaging during operation should not be fitted.

6. Where reasonably practicable, devices should be fitted to avoid the winch from over riding (pulling the wire end attachment through the head block) or under riding (running away and stripping all the winch wire off the drum). Alternatively, specific written instructions must be prepared and followed in order to project personnel when man riding. These instructions should be approved by the competent person and should take into consideration means of communication between the winch operator and passenger.

7. Winches deemed unsuitable for man riding by the manufacturer should not be used for this purpose.

DIVING SAFETY MEMORANDUM 7/1984
DIVING GASES - SUPPLIERS STANDARD

August, 1984

The following minimum purity standards for diving breathing gases, AS SUPPLIED, has been agreed with the AODC and the gas companies.

Specifications for Oxygen, Helium and Nitrogen

Gases, whether pure or mixed, should not contain any impurity at concentration likely to cause toxic or harmful effects when breathed under pressure.

Maximum permissible level of contamination in each supply gas component (ppm by volume of water phase)

CONTAMINANT	OXYGEN	HELIUM	NITROGEN
Nitrogen	1000	200	—
Oxygen	—	50	50
Carbon Dioxide	10	10	10
Carbon Monoxide	1	1	1
Neon	10	10	10
Argon	4000	25	25
Hydrogen	10	10	10
Methane	25	5	5
Other hydrocarbons	3	1	1
Moisture	25	25	25

Oil mist should not exceed 1 mg/m3.

This list is not all inclusive and no other contaminants should be present in concentrations likely to cause toxic or harmful effects when breathed.

Percentage of tolerance in mixture should be restricted to plus or minus 5% of the minor components of the mixed gas.

It is stressed that the above standards are for gas AS SUPPLIED from the gas companies and not the standards for chamber atmosphere purity or for helium reclamation systems. Research aimed at establishing the maximum acceptable level of contaminants in a chamber atmosphere and reclamation system is progressing but until this information is available diving contractors should take advice from the Health and Safety Executive recommendations on occupational exposure limits published in a guidance note obtainable from Her Majesty's Stationery Office entitled "Guidance Note EH40 Occupational Exposure Limits 1984". The necessary correction for pressure and exposure time must be applied.

DIVING SAFETY MEMORANDUM 8/1984 EVACUATION UNDER PRESSURE OF MEDICAL CASUALTIES

10 September 1984.

The Diving Operation at Work Regulations 1981 specifically refers to the responsibilities of every diving contractor and every other person involved, and, in the case of:-

(i) an offshore installation, the owner;
(ii) a proposed offshore installation, the concession owner;
(iii) a pipeline, the owner; and
(iv) a proposed pipeline, the person who will be the owner when it is layed.

The above are required to ensure so far as is reasonably practicable that these regulations are complied with.

The regulations go on to spell out in more detail that every diving contractor shall so far as is reasonably practicable ensure that emergency services are available and in particular when diving:-

(i) using saturation techniques; or
(ii) at a depth exceeding 50 metres; facilities for transferring the divers safely under a suitable pressure to a place where treatment can be given safely under pressure.

Paragraph 103 in the Guidance Notes suggest the requirement for a transfer under pressure facility for certain operations, but does not specify who makes this provision. Practical consideration may indicate advantage if a person (other than the diving contractor) having a general duty under Regulation 4, provides such facilities. The diving contractor then has the duty of ensuring transfer lock compatibility with his own related equipment. (The diving contractor has the responsibility to ensure the provision of a means of transferring diving medical casualties under pressure for all saturation diving operations and any other diving operation deeper than 50 metres. The client has the responsibility to ensure that the diving contractor conforms to the legislation. However, there is no objection to the client providing such a service to be used by the diving contractor when required).

At present the offshore facilities for carrying out a transfer under pressure of a medical casualty are supported and maintained by a group of oil companies who participate in a joint funding project. Subject to chamber transfer locks being compatible with the transfer under pressure system diving companies operating under contract from a participant member are considered to be fulfilling the legislative requirements.

However, in circumstances where the “client” is not a participant in the joint funding project, contingency plans must be made for the evacuation of medical casualties to a place where treatment can be given safely under pressure. The plans need not involve the system run by the Participant Group already mentioned, but should a contractor wish to call upon those services in the event of an emergency, prior arrangements must be made in writing with the Participants Group.

Diving contractors not wishing to involve the system run by the Participants Group must clearly define in their contingency plans the alternative system to be used.

Documentary evidence showing that satisfactory contingency plans were made prior to commencement of diving operations, will be required by the Diving Inspectorate when carrying out routine inspections.

DIVING SAFETY MEMORANDUM NO. 9/1984 DIVING FROM DYNAMICALLY POSITIONED VESSELS

9 October 1984.

Your attention is drawn to the following:-

In a recent incident on the UK continental shelf a dynamically positioned diving vessel failed to remain on station whilst a diving operation was in progress due to a failure in the electrical system supplying power to the thrusters. An investigation into the failure was carried out by the operators and the DP vessel was returned to service without the fundamental cause of the power failure being determined. As a result, further losses of power were experienced including a loss of DP during another diving operation.

Only after this second incident did the necessary comprehensive investigation into the system malfunction by competent engineers reveal the fundamental causes of the failures.

Operators of such vessels should ensure that any system malfunction or failure is properly assessed by the relevant competent person. Any diving operation, which could place a person’s health or safety at risk if such a malfunction or failure re-occurred, must be held in abeyance until such time that the competent person has satisfactorily established the cause of the malfunction or failure and has taken proper measures to prevent the re-occurrence.

DIVING SAFETY MEMORANDUM 11/1984 QUALIFICATION OF DIVERS

19 November 1984

Regulation 10(1)(b) of the Diving Operations at Work Regulations 1981 states that the certificate of training required by legislation shall be valid only if the person or body issuing the certificate (the Health and Safety Executive) is satisfied that the diver has attained a satisfactory standard of competence (whether by training, experience or a combination of both).

Under no circumstances does the Department of Energy Diving Inspectorate accept sport diving as “experience”.

The above Diving Safety Memorandum reminds us of the need for properly conducted training for commercial divers. The Underwater Training Centre at Morwell (pages 6-8) is the only place in Australia set up to carry out this training.

DIVING SAFETY MEMORANDUM 12/1984 CERTIFICATION OF DIVING PLANT AND EQUIPMENT

26 November 1984

Regulation 13(4) of the Diving Operations at Work Regulations 1981 states that "The Diving Contractor shall enter in, attach to or insert into a register kept for the purpose, the certificates and information required by para (1)(b) and (c)". Para (1)(c) of this regulation requires that a certificate is issued by a competent person in accordance with para (2) which at (a) states the information that is required on the certificate.

Diving contractors are to ensure that certificates for each major item of equipment comply with the above requirements. It is however appropriate that where there are a number of similar items such as gas cylinders etc. that one certificate may contain the serial numbers of all these items. One certificate covering a number of different types of plant or a blanket certificate for the whole system is not acceptable.

The certificates are to be contained in the register in a logical sequence so that they can be readily identified and compared with the serial number and description of the plant to which they refer. The register is to be kept on board the diving platform and be available to the Diving Inspectors from this Department on request.

DIVING SAFETY MEMORANDUM 13/1984 FIRES IN COMPRESSION CHAMBERS

30 November 1984

It has been reported to the Diving Inspectorate that a pillow started to smoulder after coming into contact with a light bulb in a saturation deck compression chamber.

Diving companies are to ensure that all lights and other sources of heat which could cause ignition are adequately guarded. Where practicable, flammable items should be prohibited from entering compression chambers and all bedding, towels etc. should be manufactured from fire retardant materials.

Commander SA Warner
Chief Inspector of Diving

WORK STARTS ON LIFEBOAT TRAINING FACILITY

Construction work has begun at the Norwegian Underwater Technology Centre (NUTEC) in Bergen with the world's first permanent facility for training offshore platform personnel to use freefall lifeboats.

Due to be completed by summer 1985, the NOK 22 mill installation comprises a 40-metre-tall tower positioned on the shore close to NUTEC and a lifeboat.

Training courses are scheduled to begin on 1 September next year, involving crew for the production platform being built by Norway's State Oil Company Statoil for its north sea Gullfaks field.

This platform will be the first offshore unit fitted with lifeboats specially designed to drop vertically into the sea from a height of 28 metres.

Developed by the Harding Company of Norway, these steel-hulled craft incorporate several features which enhance evacuation safety from an offshore installation.

The 68-seat boat accordingly hangs from a single hook, avoiding the problems posed when Davits get stuck, and a rapid launch procedure means that orders for launching can be delayed by comparison with conventional craft.

Once dropped into the sea, a special hull configuration ensures that the Harding vessel's own weight and inertia will propel it some 200 metres away from the platform without engine power.

The course will aim to train offshore workers in freefall lifeboat launch procedures, and overcome any reluctance they may feel about the prospect of being dropped from 28 metres.

In the latter context, a lower launch height of only 15 metres is being incorporated in the tower to give trainees a chance of adjusting to the idea.

Further information from:

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N-5034 Ytre Laksevåg.

EVEN PIRANHAS NEED CLEAN HANDS AT FEEDING TIME

A businessman had as his slogan "Our new (video recorder) sets won't cost you an arm or a leg", and a good idea to draw customers. He offered large discounts to anyone brave or stupid enough to snatch a coin from a fish tank full of piranhas. Among the resultant throng were, naturally, RSPCA Inspectors. But they were not after the coin. No! They were after the businessman, intent to discover whether the animal protection laws could be extended to include such events, for this occurred in Stockton. This is a town 320 km north of London, where the river does not contain schools of these fish waiting for the opportunity to attack and devour any animal they can reach. In their native habitat there are probably few willing to contribute to RSPCA funds for their protection, and this is not only because they are neither furry nor cuddly. But in the UK things are different. The reason for the RSPCA's concern? Yes, they feared lest eager shoppers, after a bargain, would omit to clean their hands adequately and the piranhas would get food poisoning from the contamination. It is hoped that any antipodean entrepreneur wishing to avail himself to this advertising gimmick will remember to place a washbasin, soap and a clean towel ready by the tank. And plenty of bandages.

Based on a news item in the Sydney Daily Telegraph, 2 April 1984.

DIVER MEDICAL TECHNICIANS BEWARE OF DEXTRAN

Dextran is often stocked, as the standard intravenous fluid for the treatment of decompression sickness and other accidents, in the medical kits of commercial diving operations. There are problems with dextran which prompted the National Association of Diver Medical Technicians to publish, in its newsletter "TRIAGE", the article which appears below. We are grateful to NADMT for permission to republish. (FDA stands for the US Food and Drug Administration.)

The FDA Drug Bulletin, Volume 13, Number 3 (November 1983) describes adverse reactions to the intravenous administration of dextran, and is reproduced below.

FDA has learned of 12 adverse reactions to dextran, including 3 deaths, that occurred during the first half of 1983. Other adverse effects included dyspnoea, flushing, rash, fever, oliguria, cardiac arrhythmia and, most frequently, a decrease in blood pressure. Such reactions followed administration of either Dextran 40 ("Low Molecular Weight Dextran") or Dextran 70. Usually the first untoward reaction begin in the first few minutes of the infusion. In all 3 cases that resulted in death, less than 10 millilitres of the product was given.

Because the total distribution of dextran has increased only slightly in recent years, it is not known whether these adverse drug experiences represent a true increase in reaction frequency or only improved reporting. FDA received 5 reports of adverse reactions to Dextran 40 and 70 occurring in 1981, and 7 (including at least 1 product-related death) in 1982. Examination of the manufacturing and testing records revealed no product aberrations. These reports were reviewed at a recent meeting of the FDA Blood Products Advisory Committee, which recommended that the medical community be apprised of this experience and the potential for side effects when dextran is used.

Two types of dextran products are marketed in the United States. The low molecular weight material, Dextran 40, is prepared as a 10% solution in either 0.9% sodium chloride or 5% dextrose. The higher molecular weight preparations, Dextran 70 and Dextran 75, are sold as 6% solutions in 0.9% sodium chloride or a sugar solution. (The numerical designation, eg. 40, is simply the weight-average molecular weight divided by 1000).

Labelled Indications and Adverse Reactions

The current approved labelling for Dextran 40 includes a total of 3 indications (not all of which are claimed by each manufacturer of the product): as a plasma volume expander in the treatment of shock; as a priming fluid during extracorporeal circulation; and for prophylaxis against venous thrombosis and pulmonary embolism in surgical procedures associated with a high incidence of thromboembolic complications. Dextran 70 and Dextran 75 have only one approved indication: for plasma volume expansion in the treatment of shock.

The labelling also notes the following adverse reactions: nausea, vomiting, fluid overload (and, for products in sodium chloride, hypernatremia and oedema), renal tubular vacuolization and renal failure, fever, several haemostatic abnormalities including decreased Factor VIII levels, interference with platelet function, prolonged bleeding time and increased bleeding tendency, as well as urticaria, wheezing, chest tightness, hypotension, and cardiac and/or respiratory arrest. The latter symptoms are indicative of an anaphylactic or anaphylactoid reaction. Such reactions can be severe and, as emphasized by the reports described above, fatal.

Steps To Decrease Incidence of Adverse Effects

Anaphylactic/anaphylactoid reactions are most likely to occur when dextran is administered to a patient who has a high plasma concentration of dextran-reactive antibodies. These antibodies evidently arise in response to dietary or bacterial polysaccharides, not as a result of previous dextran administration. Working on the principle of antibody neutralization by haptens, one manufacturer has recently developed a very low molecular weight product known as Dextran 1. In clinical trials it was found that injecting 20 millilitres of Dextran 1 intravenously 2 minutes before infusion of Dextran 40 or 70 significantly decreased the incidence of severe (but not moderate or mild) side reactionn

It is expected that Dextran 1 will be distributed with Dextran 40 and 70 in the near future. It will be of interest to learn whether the results achieved in the trials are duplicated in clinical practice. Therefore, clinicians and pharmacists who encounter adverse reactions to dextran are encouraged to report their findings to the National Center for Drugs and Biologics, 8800 Rockville Pike, Bethesda, MD 20205; telephone (301) 443-5410.

IS FUGU FUN FINISHED?

The Japanese Ministry of Health has issued rules which prohibit the serving of fugu (puffer fish) even in the restaurants presently specially licensed to prepare and sell this danger-laden fish. Although many European palates may find the taste lacking in interest, to many Japanese the chance of dying/not dying adds that something extra to the meal. Skilled chefs are licensed to prepare the fish, removing the most poisonous parts. It is said that if the customer is alive half a day after the meal he will not be in any danger. In 1975 a famous Japanese Kabuki actor died after he had consumed four portions of fugu: the chef received an eight year suspended sentence and the restaurant paid out compensation. To reduce the chance of errors occurring, only 22 of the over 50 species of fugu are allowed to be prepared for eating. It is said that about a dozen fatalities occur yearly from this food, chiefly from meals prepared by amateur chefs. The law is thought by some to be something of an over-kill and likely to be given the Japanese equivalent of the Nelson (blind) eye.

CONFERENCES

1985 JOINT CONFERENCE UNDERSEA MEDICAL SOCIETY ANNUAL SCIENTIFIC MEETING AND THE TENTH ANNUAL CONFERENCE ON CLINICAL APPLICATION OF HYPERBARIC OXYGEN

11 - 14 JUNE 1985
The Hyatt Regency Hotel
Long Beach, California, USA.

THE SOCIETY
Founded in 1967, the Undersea Medical Society is actively engaged in the advancement of undersea medicine, clinical hyperbaric medicine, and support sciences. The Society provides an international forum for professional scientific communication among individuals and groups involved in basic and applied studies concerned with the undersea environment, and the treatment of specific diseases with hyperbaric oxygen therapy.

THE HYPERBARIC OXYGEN CONFERENCE
The Annual Conference on the Clinical Application of Hyperbaric Oxygen is directed towards updating the clinician with the state of the art applications of hyperbaric oxygen therapy. During its nine year history, the HBO Conference has become international in scope and an important source for introducing new clinical applications of HBO therapy.

THE UNDERSEA MEDICAL SOCIETY ANNUAL SCIENTIFIC MEETING
This year, the Undersea Medical Society Annual Scientific Meeting and the Hyperbaric Oxygen Conference are combined in order to provide a program that will complement the goals of each, and offer the attendees an expanded program of diving and HBO information. The joint UMS/HBO meeting is open to individuals throughout the world who are interested in the exchange of information on all aspects of research in undersea biomedical sciences and hyperbaric oxygen therapy.

LOCATION AND DATES
The 1985 joint Undersea Medical Society and Hyperbaric Oxygen Conference will be held in the Hyatt Regency Hotel, Long Beach, California. The Opening Reception is scheduled for Monday evening, 10th June 1985, and sessions will be scheduled from Tuesday morning through noon Friday, 14th June 1985.

PROGRAMME PLAN
Preliminary plans include a programme of symposia, poster sessions, original papers and discussion. A special one day session for the Associates of the UMS will run concurrently with the joint UMS/HBO Meeting on Wednesday 12th June 1985. The Annual Business Meeting of the UMS is scheduled following the noon luncheon on Thursday 13th June 1985. The Suzanne Kronheim Memorial Lecture will be given Friday morning, 14th June 1985.

Plans include a diving trip and tour of the Catalina marine facilities, visits to the Baromedical Department at Memorial Medical Center and a tour of the College of Oceaneering. The Second Symposium on Decompression Procedures Across the Pressure Continuum will be chaired by RW Hamilton, Jr, PhD.

The annual Awards Banquet is scheduled for Thursday evening, 13th June 1985. The Albert R Behnke Award, Oceaneering International Award, Stover-Link Award, Charles W Shilling Award, Craig Hoffman Award and Boerema Award will be presented during the banquet to individuals who have made special professional contributions to the field of diving and hyperbaric medicine.

REGISTRATION AND HOUSING
Registration cards and housing forms are available on request.

Registration Fees	*In Advance*	*On-Site*
Members	$110.00	$125.00
Non members	$130.00	$145.00
*Students, Associates of UMS and EUBS Members	$80.00	$90.00
Spouse/Guest	$35.00	$45.00
Associate Luncheon (Wednesday June 12, 1985)	$12.50	$15.00

*Any student working toward a degree in the biochemical sciences may obtain the reduced rate by providing a letter verifying eligibility, signed by a Department Head or Research Advisor.

The registration fee includes receipt of the badge, Program/ Abstracts book, opening reception, refreshments during breaks, local tours,** and ticket for the UMS/HBO Awards Banquet. The spouse/guest fee includes receipt of the badge, opening reception, banquet, and local tours.** (**Baromedical Unit at Memorial Medical Center and College of Oceaneering.)

To register in advance write to:

1985 Joint UMS/HBO Meeting
Attn: Ms Marilyn Dale
c/o Baromedical Department Memorial Medical Center
PO Box 1428
Long Beach, CA 90801-1428 USA.
before May 1, 1985.

THE 4TH WORLD CONGRESS ON INTENSIVE AND CRITICAL CARE MEDICINE

Will be held in Jerusalem from 23 June to 28 June 1985. It will be followed by

AN INTERNATIONAL SATELLITE SYMPOSIUM ON HYPERBARIC OXYGEN IN CRITICAL CARE MEDICINE

to be held in Eilat from 30th June to 2nd July 1985.
The Chairman, Scientific Programme, will be

Dr Yehuda Melamed
Director, Israeli Naval Hyperbaric Institute
PO Box 8040 31080 Haifa
Israel

WORLD UNDERWATER FEDERATION (CMAS) INTERNATIONAL CONGRESS 1985

We reproduce (as received) two letters from CMAS about the international Congress to be held in Miami in April 1985.

Confederation Mondiale des Activites Subaquatiques
World Underwater Federation
34, rue du Colisee, 75008
PARIS, FRANCE

Paris, October the 19th 1984

-:- CIRCULAR LETTER TO CMAS DOCTORS -:-

Dear Colleague,

As you know, the next CMAS General Assembly will take place in Miami from 22nd to 28th April 1985.

The medical and prevention commission, like other CMAS commissions and committees, wished organize there its general assembly and medical colloquy.

We should be obliged if you could tell us before 15 November, if you intend to go to Miami.

Cheap travel prices are studied with Pan Am.

Please find here enclosed a reservation voucher from Hotel Hyatt Regency, chosen by the organizers, which offers 50% discount.

Book your room before 22 March 1985 to beneficiate of this price.

Awaiting your reply, I remain

Yours sincerely

Marcel BIBAS
Vice-President of CMP

Paris, 31 October 1984.

CIRCULAR
TO ALL MEMBERS OF THE EXECUTIVE BUREAU
TO ALL AFFILIATED FEDERATIONS

Dear Sir

We hereby confirm that the next CMAS international congress will be held in Miami (USA) 22-28 April 1985.

We hope that you will take advantage of this exceptional occasion to get to know Florida and to make use of the very interesting rates that we have been given by Hotel Hyatt Regency (chosen by the American organizing committee), as well as by the travel agency which has made us a remarkable offer for groups with departures from Paris or Marseille.

Hotel Hyatt Regency - Miami:

single room (1 person)	$60 (instead of $120)
double room (1 bed)	$60 (instead of $120) (270 FF/person.)
double room (2 beds)	$60 (instead of $120)
room for 3 people (2 beds)	$75 (instead of $150) (225 FF/person.)
room for 4 people (2 beds)	$90 (instead of $180) (203 FF/person.)

FLIGHT PARIS - MIAMI (via Madrid) IBERIA - FF 2.200 (instead of 9.350 FF)

You can find numerous places to eat in small restaurants and fast food stores at a very reasonable price.

Dives will also be organized on remarkable sites but we are still waiting for details.

We would like to inform you that there will be many other activities at the same time as our General Assembly:

scientific symposium
photo competition
evening entertainment with films and conferences
discussions (round table) with diving pioneers
etc.

Don't forget that the "Ocean Week", 22-28 April 1985 is organized in Miami by "Ocean Realm Magazine" with the participation of "International Oceanographic Foundation", Pan Am, and National Car Rental.

An entertaining program is planned: exhibitions, conferences, fashion shows, music, sea food

We are sure that all these activities will attract you, but before making definitive arrangements with the airline company we would like to consult you for the last time and could therefore like to ask you to return the attached question form duly filled and signed to us before 15 November 1984.

Thank you in advance for your cooperation.

Yours sincerely

Pierre PERRAUD
General Secretary.

SPUMS NOTICE

REPRINTING OF ARTICLES