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CONTENTS

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To publish a journal.
To convene members of the Society annually at a scientific conference.

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1 Anderson T, RAN medical officers' training in underwater medicine. *SPUMS J* 1985; 15: (2) 19-22
2 Lippmann J, Bugg S. The diving emergency handbook. Melbourne: J.L.Publications, 1985

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EDITORIAL

Two papers in this issue report on the significance to divers of having an incompletely sealed Foramen ovale in the heart. As this condition is demonstrable in about 7.5% of the population and has been noted particularly frequently (when it has been looked for) in those suffering neurological decompression sickness the reports have undoubted relevance to any discussion concerning assessment of fitness to dive. As the risk of a right to left side passage of blood within the heart is increased by performing the Valsalva manoeuvre, wearing a wet suit and immersion, it is obvious that the diving medical of the future should perhaps include testing for this condition. If that should happen many will be found Unfit to Dive when the condition is demonstrated. In this case because of the expense, and occasional morbidity, far fewer will pursue an initial interest in diving and scuba diving will return to being a recreation which only those who are exceptionally fit will undertake, and then only after very strict medical assessment. This would be a far cry from the view that diving is a sport for everyone, which has led to the current boom in diving. There will be a lot of resistance to such an investigation being included in a diving medical from the diving industry, many of whose members feel that the price of an adequately performed diving medical inhibits prospective customers. However in these days of increasing willingness to resort to litigation it is not beyond the bounds of possibility to suppose that some lawyer will claim that investigation to exclude this condition is mandatory in any assessment of fitness to dive. Such is the price we may pay for seeking a better understanding of the physiology and pathology of diving.

Had Cousteau and Gagnan been aware of even a small part of what we now know about the physiological problems to which the development of the Aqualung would expose millions of scuba divers it is possible that they would have been rather more cautious in the popularising of their product. If they had been told of the significance and frequency of an imperfectly sealed foramen ovale in the hearts of healthy people they themselves might have held back from venturing underwater. On the other hand they might have remembered the "experts" who confidently predicted that travel at 30 mph in the new fangled trains would be fatal, that the humble Bumble Bee was aerodynamically unsuitable for flight, that Mount Everest could never be climbed without using cylinder-supplied oxygen and that men would never reach and walk on the moon. So probably the real importance of these papers is that they help us to understand the mechanism of unexpected clinical presentations. They naturally leave unanswered the question of why recogniseable morbidity is so infrequent, and usually only occurs when our simple rules for safe diving are broken significantly. The human body is obviously better at safeguarding us than was ever predicted. But NEVER take it for granted that it can protect you from every mistake !

Now may be an appropriate time to analyse the significance of asthma and other medical conditions in relation to actual causes of morbidity and mortality. But for this there will first need to be a determined (and successful) effort to collect relevant data, a raw material in far shorter supply than opinions on the subject.

The description of the medical planning undertaken to safeguard workers during the tunnelling work for the Singapore Mass Rapid Transit System is an invaluable insight into that brother activity to diving, working in an hyperbaric air environment in a caisson. This has a particular relevance as it was the engineers who were undertaking the deep sinking of bridge foundations using caissons in the late 19th century, and the doctors contracted to look after the workers on these projects, who laid the foundations of our understanding of the management of hyperbaric related problems.

Both the Aviation Bends case and the case of the Seven Green Divers show the clinical acumen of the people first involved in treatment. The paramedics who recognised that the co-pilot was suffering from high altitude decompression sickness especially deserve commendation. Unfortunately the medical management showed a lesser awareness of the need for early recompression treatment. The limitations which still exist in the provision of adequate recompression facilities in some centres is noted in relation to two facilities, neither of which was involved in the National Safety Council of Australia (Victorian Division) debacle. The claims that there are cases which require hyperbaric oxygen treatment but are denied it because there are inadequate facilities is one which should provoke a response from the State Health Departments involved. Perhaps there may be a place after all for the litiginous patient, that great stimulator of action, though not necessarily of medically correct action.

Litigation is a problem more likely to increase than disappear so there is little comfort to be gained from bemoaning the fact that there may be no unassailably correct decision to hand down when asked to decide on medical fitness to dive. Hence the practice by doctors of certifying an applicant as either meeting or failing to meet criteria promulgated by some official organisation and so avoid offering an opinion. This may be cowardice but is hardly unreasonable. However, as has been shown recently, even official standards can be challenged in court. Lawyers acting for some air pilots were able to claim successfully that it was unreasonable to regard colour blindness as a reason for refusing them permission to fly. It is said that there are bumper stickers in the U.S.A which read "Make your child a doctor and support a lawyer". Perhaps one day we will

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ORIGINAL PAPERS

FORAMEN-OVALE, DECOMPRESSION SICKNESS AND POSTURE FOR ARTERIAL GAS EMBOLISM.

D F Gorman and S C Helps

The significance and prevalence of a patent foramen-ovale in divers and in particular in divers with decompression sickness (DCS), and the importance of these findings to the preferred posture for divers with suspected arterial gas embolism (AGE) is now of concern to both divers and physicians because of a study recently reported in the Lancet[1]. This study used two-dimensional echocardiography to show an increased prevalence of patent foramen-ovale in divers with severe DCS (11 out of 18). In another study of 6 divers, the 5 who had early onset neurological disease had a patent foramen ovale[2]. The suggestion is that these severe episodes of DCS result from the shunting of bubbles from the right to the left side of the heart and hence that AGE occurs. This is supported by another recent observation, that in 23 divers with neurological DCS who were investigated by SPECT scanning, perfusion defects (indicative of AGE) were demonstrated in all of their brains[3]. The significance of these defects is reinforced by the good correlation shown between the regions of reduced brain blood flow and the functional neurological deficits in the divers. The concern then is two-fold; should all prospective divers be screened for a patent foramen ovale and is the head-down posture advisable in divers with suspected AGE? This posture, especially if maintained at 30° to the horizontal and associated with leg-elevation will cause an increase in venous return and hence any right to left shunting of gas bubbles[4].

Although bubbles were detected by doppler ultrasonic monitoring to pass from the heart and aorta to the carotid arteries of head-down dogs[5], there is still a good argument to place divers suspected of AGE head-down. In another series of dogs, gas injected into the pulmonary vein always caused embolism of the cephalic circulations of head-up dogs but never in head-down dogs[6]. In our own experiments in rabbits, gas bubbles travelled "backwards" up the aorta in head-up animals against blood flow, and while we could always see these gas bubbles in the pial arteries of head-up animals, none were ever seen in head-down animals[7,8]. Also, in our clinical experience a posturally-induced relapse in a diver with AGE is usually lethal. The majority of divers with AGE experience some spontaneous resolution of symptoms and signs[9]. This recovery is almost certainly due to bubbles passing through the brain capillaries and being cleared by the veins allowing a restoration of brain blood flow[7,8]. Unfortunately, many subsequently relapse. Such relapses could be due to re-embolism[10], progressive brain oedema causing tissue to shift[11], or most likely is due to increased resistance to brain blood flow because of the effects that gas bubbles have on endothelial cells and blood constituents[12-14]. Over recent years a head-down posture has been frequently employed, and often abandoned without incident. However, in 4 divers with AGE that we have been involved with and who had largely recovered spontaneously, a change from a head-down to a head-up posture resulted in a sudden and dramatic loss of consciousness, and only 1 of these survived. The only reasonable explanation of these events is that new bubbles (from a patent bronchovenous fistula or from a gas reservoir in the heart or great vessels) have distributed to the brain circulation. The poor outcome is most likely due to these bubbles entering an already compromised circulation[12-14].

It follows that the head-down posture is an important part of the first-aid management of some divers with AGE. As it is impossible on the basis of their initial presentation to predict who will subsequently relapse[10], it is probably advisable to employ a head-down posture for all divers suspected of AGE. This posture does have costs which include an increased hydrostatic pressure in the head and the potential for increased right to left shunting of gas bubbles in divers who in fact (or who also) have DCS.

The distinction between AGE and DCS is often difficult. The convention has been that AGE in divers presents as neurological symptoms and signs within 5 minutes of reaching the surface. Although this is usually true, evidence of AGE can occasionally be delayed for 10 to 15 minutes, and in some divers cardiac involvement predominates[10]. It is reasonable to assume that any diver who develops neurological or cardiac symptoms and signs within 15 minutes of reaching the surface has AGE until proven otherwise. Symptoms and signs of DCS can, however, arise during a decompression, and fulminant and life-threatening DCS will also usually present within minutes of the decompression. In animals, the more fulminant the DCS, the shorter is the time between decompression and the development of overt disease[15]. Cerebral DCS in particular has a relatively short latency before onset[16]. A modest head-down posture will probably not disadvantage (and if it is true that a patent foramen-ovale and AGE underlies many of the serious cases of DCS may help by limiting embolism of the brain circulation) a diver with such DCS. However, it is now clear that this posture must be modest (head lower than chest which is lower than the abdomen and pelvis) and care must be taken to avoid a significant increase in venous return and hence minimise right to left shunting of gas bubbles across any patent-foramen ovale by not elevating the divers' legs[4].

The need to survey potential divers for a patent foramen-ovale is not established by these recent studies[1-3]. Certainly, neurological DCS is still a relatively rare phe-

nomena amongst sports divers (about 300 episodes of neurological DCS in Australia annually from an active diving population of more than 400,000) and yet between 15 and 30% (60,000 - 120,000) of these divers can be expected to have a patent foramen-ovale[17]. The technique used to detect a foramen-ovale is not inexpensive, will only identify between 30% and 50% of those divers who do have a patent foramen ovale[1,17], and is not without risk. For example, microbubbles are injected into the circulation to help to identify any foramen[1,2], and yet if the results obtained in experiments in rabbits just completed in our laboratories can be extrapolated to humans then such microbubbles can have a significant effect on both cerebral blood flow and brain function. A decision to screen all divers should await further data. There is no doubt however that the 2 current issues of most interest in the pathophysiology of DCS is the role of a patent foramen-ovale and AGE[1-4] and the importance of complement protein activity in animal models of DCS[18].

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PATENT FORAMEN OVALE

David Davies

This paper was originally written in response to an enquiry from the Training Department of the National Association of Underwater Instructors (NAUI) which was concerned about the incidence of patent foramen ovale amongst trainee divers and the possibility of these people having a diving accident attributable to the defect. In addition the paper has formed the basis of a report to the Australian Sports Medicine Federation, many members of

which do medical examinations for divers, before being offered to the SPUMS Journal.

There has been some concern over recent months in diving circles about the relation between cerebral decompression sickness, cerebral gas embolus and the presence of a patent foramen ovale.

In the foetal circulation, the foramen ovale enables oxygenated blood from the placenta to cross from the right to the left side of the heart thus by-passing the lungs. At birth, with the onset of normal breathing, pressure changes in the heart close the flap value arrangement of the foramen and the shunt ceases. Obviously in a small percentage, this shunt persists and, depending upon its size, can be readily detected and, if necessary, treated by surgical means.

There is a significant proportion of the population in which the foramen ovale persists but is functionally closed and is asymptomatic. However, if there are significant intrathoracic and intracardiac pressure changes then the foramen may reopen and a right to left shunt occur. In an autopsy study of 965 normal hearts, Hagen[1] reported that the incidence of patent foramen ovale is 15-30% in the population so that of the 400,000 divers in Australia 60-120,000 of these can be expected to have a foramen ovale.

In 1988 Lechat[2,] in the New England Journal of Medicine, and Webster[3,] in The Lancet, reported on the association between stroke and the presence of a patent foramen ovale. Webster showed that a patent foramen ovale was present in 30% at rest but this increased to 50% during a Valsalva manoeuvre in the stroke patients. This was compared with normal controls who had an incidence of 7.5% at rest which increased to 15% during a Valsalva manoeuvre. Lechat's figures were very similar. Wilmshurst[4,] in the British Medical Journal, reported a case of cerebral gas embolism in a diver with a previously undiagnosed foramen ovale. This patient dived to 38 metres for 15 minutes and ascended at 15 m/minute, a dive close to the no decompression limits of the RNPL/BASC tables and, within two minutes of surfacing, developed profound cerebral symptoms.

In an article by Moon[5] reported in the Lancet, 30 patients with a history of decompression sickness were examined for the presence of a patent foramen ovale. 37% had a right to left shunt at rest compared with 5% in healthy volunteers. In a subset of 18 patients who had serious signs and symptoms, 61% had evidence of intra cardiac shunting.

It has been well shown that venous gas emboli occur in most divers diving within the no decompression limits and these bubbles result from the release of inert gas from tissues during decompression. Normally these bubbles are filtered out in the lungs and remain asymptomatic. However the presence of a patent foramen ovale may permit the passage of these bubbles into the arterial circulation. These gas bubbles may also 'seed' susceptible tissues and precipitate symptoms of severe decompression sickness.

This evidence then suggests that diagnosis of patent foramen ovale is essential during the medical examination. However Webster[3] showed that clinical examination by a cardiologist, radiology and electrocardiography was unrewarding. Definitive diagnosis of a patent foramen ovale is made by bubble contrast two dimensional echocardiography, a technique available only in some specialist cardiac laboratories which requires injection of small bubbles into the circulation and their detection in the heart by a type of sonar apparatus. This technique has a significant morbidity and not inconsiderable cost to the patient. It cannot be recommended as a routine screening procedure on all prospective divers, especially as the incidence of neurological decompression sickness is still relatively uncommon. Should clinical evidence of a septal defect be detected at a diving medical examination whether or not there is evidence of intracardiac shunt the prospective diver should be advised of the risks associated with the condition and be referred to a cardiologist.

There are factors other than just the Valsalva manoeuvre which can precipitate a right to left shunt across the atrial septum. Arborelius[6] reported that immersion in water may raise the right atrial pressure by 12 mm Hg. Similar pressure changes may be obtained by tilting the patient's head down, by elevating the legs, by wearing a tight wetsuit or by coughing. In the diver therefore these could have potentially disastrous consequences.

First aid for the diving casualty who is suspected of having cerebral gas embolism is then a matter of weighing up advantages and disadvantages. It has been traditional to recommend a head down tilt of 30° with the patient lying on the left hand side. There is good evidence from Gorman[7] and others that gas bubbles will move against the blood flow towards the most elevated part so that the head down position will tend to preserve the cerebral circulation from bubble overload. It is suggested that the left lateral position serves to keep bubbles away from both the pulmonary outflow tract and the interatrial septum. However, if the patient requires external cardiac massage or expired air resuscitation then he needs to be supine. In order to limit, as much as possible, the rise in right atrial pressure the head down tilt should be restricted to 10-15°. Other first aid measures include maintenance of the airway, 100% oxygen, intra venous fluids and urgent recompression. Gorman[8] has reported several incidents of re-embolisation after apparent recovery when the patient was placed head up. It is therefore essential that the patient remain head down until recompressed.

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SEVEN GREEN DIVERS HANGING FROM A LINE

A case reported to Project Stickybeak

Douglas Walker

There was nothing to indicate that this was to be anything other than an uneventful dive. The divers were taking an Advanced Diver Course under the care of an instructor and in the dive boat there remained two men with coxswain qualifications. There was 15 m (50 feet) visibility underwater, the dive was to be to 27 metres for 20 minutes, and there was to be a decompression stop at 5 metres, this being a routine precaution even when the proposed dive was within no-decompression limits. The "stop" was to be taken on a line suspended from a 20 litre plastic buoy with the group remaining together at all times. To prevent uncontrolled fluctuation of diver depth during the decompression stop due to the surge at the surface, the divers were advised to be slightly negatively weighted rather than aiming for neutral buoyancy at the surface.

The dive progressed as planned and the group of eight then clustered on the shot line at 5 metres for the 5 minutes planned. It was only after the instructor had twice needed to equalise his ears in the four minutes they had been hanging on the line that a suspicion rose in his mind that this was somewhat unusual so he checked his depth gauge. It read 15 metres. He looked up and saw that the buoy had been pulled underwater by their weight: it was now visibly indented by the pressure and was consequently now providing even less buoyancy lift to the eight divers. Though he at once started trying to indicate to his pupils that they had to let go of the line this took time, and after obeying this order they lost the buoyancy it had provided so sank deeper until some, if not all, returned to the sea bed.

One of the divers, A, now found that he was low on air and started breathing from the octopus regulator of diver B after indicating his need. The instructor saw that they were connected only by the air hose and put them into the correct, and safer, hands-on contact position before they started their ascent. He then saw another pupil swimming over the sea bed unable to reach the inflator hose and apparently lacking sufficient buoyancy to start ascending. After ensuring the vest's inflation he ascended with this diver and surfaced uneventfully.

On surfacing the instructor found that all was not well as divers A and B had surfaced in distress and were requiring an urgent resuscitation management. At this time there was still one diver not surfaced but fortunately this diver soon returned after completing the planned, but interrupted, 5 minute stop at 5 metres. Witnesses had seen A and B surface then float quietly face up and unresponsive. When the boat reached them one was able to make some response but the other was ash-grey faced and blood was seen in his face mask. They were quickly taken from the water and into the dive boat where A was noted as cold, breathless, and ash-grey faced, with chest pain and tingling fingers, and he was immediately placed in the Trendelenberg position and oxygen commenced. He had these symptoms when the boat reached harbour and he was airlifted with his buddy to a hospital having a hyperbaric unit. However as he appeared to have recovered by the time he reached the hospital and his buddy was similarly well they were both allowed to return home after a short period of observation.

These divers had apparently made an out-of-air ascent from an unstated depth (possibly 5 metres) because the octopus had soon exhausted the buddy's remaining air. They were observed to "pop to the surface". Diver B admitted that "he had held his breath" as he ascended. At the surface he was breathless and felt anxious but far less effected than was diver A. He had retained his weight belt. His scuba diving experience is unknown but diver A had been diving for one year.

When he got home diver A felt very tired and this tiredness was still present the next day after a good sleep so he went back to the recompression unit and on this occasion it was agreed to recompress him. This completely removed his feeling of fatigue and it did not recur.

Discussion

This incident commenced because the instructor made a decision intended to increase the safety of the group of divers as they waited out the decompression stop together on a shot line. The need to check that the buoy provided sufficient buoyancy when eight divers hung on the line was not recognised. Compression of the buoy as it was pulled underwater aggravated the situation. There was an acute and unexpected worsening of the situation when the dive group followed instructions and released themselves from the line and sank deeper as soon as the uplift it gave was lost. The group was no longer facing an expected situation and may then have been scattered to some degree, although the good visibility enabled the instructor to see what was happening. In these circumstances this instructor exercised as effective control as anyone could expect, but the group was too large for a single person to fully control. He ensured that divers A and B established effective hands-on contact before commencing their ascent and could not have altered their risk of running out of air because at that time most of the divers were probably down to a similar remaining-air and his assistance was required by the diver whose vest inflation control was floating out of reach.

The response of the persons in the dive boat when they saw divers A and B "pop" to the surface and then float motionless was completely correct. Possibly less so was the medical decision to discount the incident history, which suggests that cerebral air embolism may have complicated a lung-overpressure episode, as soon as the two divers appeared to be fully recovered. The development of decompression sickness by diver A, indicated by the excessive fatigue he felt, might have been clinically suspected while he was still at the recompression facility had there been a higher index of suspicion applied.

This incident indicates the rapidity with which any diving situation can change from the uneventful to the potentially fatal after a single additional adverse element in a dive already containing several "silent" adverse factors. In retrospect it is clear that the surface float provided inadequate buoyancy, that one instructor cannot control seven pupils should a problem arise involving more than one of them, and that it remains true that careful divers should know enough to diagnose their own diving-related problems and be prepared to maintain such opinions if necessary even in the face of dismissive medical opinions. The incident could have led to two fatalities. The triad of Archimedes, Boyle, and Murphy must never be forgotten when using a "sky anchor" for a shot line.

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witness the ultimate in justice, lawyers being sued for the effects of their case law medicine on the management of a patient. But do not hold your breath waiting for this to happen.

Until that day there is the risk of being sued by a disgruntled diver either because an "ordinary" long bone survey was performed instead of scintigraphy, or on the grounds that it was totally unreasonable to refuse to issue a certificate of fitness, because some bone changes had been found, thereby depriving the diver of his livelihood. Naturally failure to act on a finding of such changes will be actionable also. This threat to practitioners of diving medicine can only be contained when we have a database containing far more information than is now available, as decisions can then be defended from a secure foundation of case histories. Lawyers have a great respect for precedent but it will be up to us, the medical and diving community, to collect case histories and to define the areas of uncertainty where suppositions reign.

Among articles reprinted are what to do when embraced by an overinflated buoyancy compensator. Some regular divers will recognise the feelings of the occasional diver whose musings appear on page 202. And those who entrust their bottom time to a computer may get a tip or two from Bill Lovin's sad story on page 201.

SOUTH PACIFIC UNDERWATER MEDICINE SOCIETY
18th ANNUAL CONVENTION

PALAU PACIFIC RESORT
31 May - 9 June, 1990

Members wishing to present papers should contact the organiser of the Scientific Meeting.

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SPUMS ANNUAL SCIENTIFIC MEETING 1989

MEDICAL SUPPORT OF COMPRESSED AIR TUNNELLING IN THE SINGAPORE MASS RAPID TRANSIT PROJECT

Jimmy How

Summary

The Mass Rapid Transit System in Singapore has been a unique experience for the many who have worked in its development. The Republic of Singapore Navy, it was given the exciting task of providing medical support to the compressed air phase of the project. Decompression sickness (DCS) is a recognised occupational hazard faced by compressed air workers, divers and aviators. In the Singapore Mass Rapid Transit Project (MRT), 11 km of underground tunnels were built using compressed air. This report deals with the planning and the set up of the medical support infrastructure in support of the contract. It also highlights the findings obtained during the medical examination. Medical problems faced by workers in the project are discussed.

Introduction

Let me first pay tribute to Major (Dr) Vijayan, Captain (Dr) Wong Ted Min and a team of National Service doctors, nursing officers, and medical orderlies who have contributed to the successful completion of the Singapore Mass Rapid Transit Project.

Some of you may wonder why a tiny place like Singapore needs a Mass Rapid Transit system. Singapore is an island 24 miles (38.4 km) East to West and 16 miles (25.6 km) North to South, about 400 square miles in area. No longer do people live in kampongs as they did in the 1960s and 1970s. 75% to 80% of people are housed in flats in high rise buildings. It is so built up that it is a concrete jungle choked with cars. Already we have 3 million tourists a year and they are building a second international airport at Changi, with the second terminal to open in 1990. We had to have tunnels going underneath the city to transport residents and tourists because there is not sufficient road space on top for more cars. We chose to have trains running in the tunnels rather than cars.

Use of caissons and tunnelling in compressed air

Denys Papin first mentioned the idea of using compressed air to displace ground water from a working tunnel in 1691.[1]

In 1830 the English engineer, Cochrane (later Lord Dundonald) took out a patent for using compressed air to keep water back from tunnels.[1] However, it was left to the celebrated French engineer, Triger, to solve the practical problems. In 1839, Triger was able to successfully sink a tunnel into quicksand to reach a bed of coal at Haye-Longne.[2]

COMPRESSED AIR WORK IN THE UNITED STATES

In the United States of America compressed air was first used in 1869 during the construction of a railroad bridge over the Pee Dee river between Wilmington and Colombia. In the same year, the foundations of the bridge over the Mississippi at St. Louis were built using compressed air.[3] Decompression schedules used in the 1879 Hudson River Project required men to work at 32 psig (2.18 Bar gauge) for 8 hours out of 24, taking half an hour for lunch at the working pressure or at a slightly reduced pressure. At pressures higher than 32 psi (2.18 Bar gauge), the men worked in shifts of 3 hours with 3 hour rest intervals. Workers working between 40 psig (2.72 Bar gauge) and 42 psig (2.82 Bar gauge) spent 3 hours on shift with a 3 hour rest interval between shifts at normal pressure.[4] There was a high incidence of DCS in this project because the workers decompressed at the same rate regardless of the duration or pressures they were exposed to.

F.L. Keays[5], Medical Director for the contractor in charge of the construction of the East River Tunnels for the Pennsylvania Railroad in 1909, reported 3,692 cases of DCS arising out of 557,000 decompressions, with 20 deaths. The New York Tables (1912), which were a revision of the decompression tables used in the Hudson River Project, were formulated in connection with the Public Service Commission Tunnel Project. These tables were revised in 1922. However, the 1922 Tables were inadequate and Thorne[6] reported 300 cases of DCS. The New York Tables 1955-57 used in the Lincoln Tunnel Operation were yet another revision of the 1922 Tables, in an attempt by the New York State Department of Health and the Port Authority to minimise disabilities arising from dysbaric osteonecrosis.[6]

In 1961, Dr Leon Sealey, Medical Consultant to the Municipality of Metropolitan Seattle and Metropolitan Engineers, organised a committee to formulate decompression tables regulating work in compressed air in connection with the major sewage tunnel project through Seattle.[7] The new tables were subsequently adopted by the States of Michigan, New York and California. It was later observed that these new tables again failed to prevent disabling dysbaric osteonecrosis in compressed air workers. Kindwall et al[8] re-examined the current Occupational Safety and

Health Agency (OSHA) decompression schedules and concluded that these tables permitted the development of dysbaric osteonecrosis when used in the recommended pressure ranges. Until the development of a new set of schedules, an interim set of decompression schedules, with longer decompression times was adopted. An oxygen version of this table was also designed to reduce the decompression times considerably.

COMPRESSED AIR WORK IN ENGLAND

In 1852 caissons were first used in Britain by Hughes during the construction of the foundations of a bridge at Rochester in Kent[9] and shortly afterwards by Brunel for the Saltash bridge between Devon and Cornwall. DCS and dysbaric osteonecrosis were great problems for diving as well as for caisson work and in 1906 the British Admiralty appointed a committee which included J.S. Haldane to develop safe decompression schedules[10]. In 1907, Haldane described the now classic principles of staged decompression. Based on his experiments, he believed the pressures could be reduced in a 2:1 ratio without bubble formation. The decompression schedules described were used to some extent by tunnel and caisson workers, but it later became apparent that the 2:1 ratio proposed was too rapid for prolonged and high exposures to pressure.

In 1935, a British committee appointed by the Institution of Civil Engineers developed a set of decompression tables for compressed air workers working for varying periods up to 50 psig (3.4 Bar gauge), using the principle of staged decompression. These tables were widely used until new decompression tables were compiled by the Compressed Air Committee of the Institution of Civil Engineers and the Ministry of Labour (United Kingdom). These tables were first used in 1948 in the construction of a tunnel under the River Tyne and were subsequently adopted in the Compressed Air Special Regulation of the Ministry of Labour (United Kingdom), which came into force in 1958.[11]

At about the same time, new tables were produced based on Hempleman's theory on "Diffusion-Limited Gas Uptake" of tissues. The new tables were first used in Blackpool in 1966. The Blackpool Tables, with the code of practice prepared by the Medical Research Council Decompression Sickness Panel and published by the Construction Industry Research and Information Association (CIRIA), is the currently accepted standard governing compressed air work in the United Kingdom[12]. One of the more recent large scale tunnelling projects requiring compressed air, was undertaken in Hong Kong for the Mass Transit Railway, using the Blackpool Tables.

Hazards in compressed air work

With the advent of compressed air work, more and more medical problems were encountered. Besides the usual hazards of noise, dust, vibration and construction related accidents faced by construction workers everywhere, other significant health hazards of work in the compressed air environment are decompression sickness (DCS), pulmonary barotrauma and dysbaric osteonecrosis. DCS, also known as bends, caisson disease, compressed air illness or dysbarism, is a condition which results when there is an overly abrupt and extensive reduction in environmental pressure. Compressed air workers, divers, medical workers in hyperbaric environments and aviators are similarly susceptible to DCS and dysbaric osteonecrosis.

Although the symptoms of DCS were first described by Triger[2] in 1841, it was only in 1854 that Pol and Watelle[13] noted these afflictions occurring only in workers leaving the tunnel and not whilst entering or remaining in the compressed air environment. Signs and symptoms of DCS with painful joints or with disturbances of the cardiovascular, respiratory and nervous system were described. They rightly recommended recompression as a therapeutic modality but it was left to others to develop proper decompression schedules and therapeutic tables.

Pathogenesis

DECOMPRESSION SICKNESS

A reduction in ambient pressure causes dissolved nitrogen to form nitrogen bubbles in tissues. The exact mechanism of bubble formation, even after 100 years of research, is still unclear. Various theories of bubble formation, de novo nucleation, supersaturation, tribonucleation and in vivo cavitation, have been suggested as possible causative factors of the bubbles implicated in decompression sickness.

As early as 1670, Boyle[14] observed bubbles in tissue and blood samples of animals decompressed in hypobaric chambers. Paul Bert[15], in a series of experiments with goats and other small animals, established the role of nitrogen bubbles in DCS. Many other workers[16-18] showed that gas bubbles arose both intravascularly and within tissues. Intravascular bubble formation can lead to embolisation and mechanical obstruction of blood vessels. This was the earliest proposed mechanism explaining the observed symptoms and the findings of ischaemic changes in the various organs. The fact that bubbles can be detected by histology, direct observation with an operating microscope and by doppler ultrasonography[19], indicate that nitrogen bubbles are the causative agents in DCS.

Further studies since the 1930s have found that bubble-blood interactions occur in vivo and may account for some of the clinically observed symptoms like inflammation around joints and relapsed symptoms, and biochemical changes in the blood. Concurrent work done by Swindle[20] and End[21] showed sludging of red cells with the formation of

emboli and petechial infarcts in spinal cord and brain in DCS. Subsequent work done by numerous researchers have shown that bubbles produced changes in the blood and tissues with both morphological and metabolic consequences. These include alteration in platelet function, changes in the coagulability of blood and in catecholamine, plasma lipid, plasma protein and enzyme levels. Leitch and Hallenbeck[22] showed that the pathology may also be caused by arterial gas embolism leading to peripheral vascular obstruction by gas. Cord segments involved showed varying degrees of haemorrhage and occasionally vascular congestion. Microscopic petechiae were present in both the grey and white matter. These appearances were compatible with hypoxia or embolic episodes. In 1987 Thorsen et al[23] showed, with the help of scanning electron microscopy, activation of human platelets by nitrogen micro bubbles.

DYSBARIC OSTEONECROSIS

The other hazard of compressed air work is that of dysbaric osteonecrosis. Bassoe[24], in a paper to the Chicago Neurological Society, described chronic joint pain and stiffness in 11 out of 161 caisson workers. Bornstein and Plate[25], also described 3 cases of joint disease among some 500 bends cases associated with the construction of the Elbe Tunnel at Hamburg. Bassoe suggested a relationship between initial joint "bends" and subsequent development of bone atrophy.

This condition is included in this paper as many workers consider dysbaric osteonecrosis as a chronic form of DCS. The aetiological basis of this disease is similar. Dysbaric osteonecrosis has been observed following caisson work at a pressure as low as 17 psig (<12 msw), and also for as short an exposure as 7 hours at 3.38 ATA.

Dysbaric osteonecrosis may appear several months, or even years, following inadequate decompression from compressed air environment. The victims may or may not have had a past history of DCS. It has been proposed that dysbaric osteonecrosis occurs as a result of bone infarction caused by occlusion of capillaries by nitrogen bubbles or sludge-formed elements of the blood. Once blockage occurs, the osteocytes in the affected bone die if the ischaemia is not reversed within 12 hours. Common sites for dysbaric osteonecrosis are the head, neck and shaft of long bones, especially in the femur, tibia and humerus. Dysbaric osteonecrosis is seen radiologically about 4 months after the initial insult. Severe cases of dysbaric osteonecrosis may have marked sclerosis and collapse of the bony trabeculae, resulting in disruption of the overlying joint.

Use of compressed air tunnelling in Singapore

In Singapore, compressed air work was first used in sewage tunnelling in 1982. In 1984, compressed air tunnelling began on the Singapore Mass Rapid Transit (MRT) project.

The idea of a Singapore Mass Rapid Transit system first surfaced in the early 1970s when the State and City Planning Study examined land use and transportation in the light of the Government's developmental policies. This study, completed in 1971, confirmed that it would be physically impossible and environmentally unacceptable to build roads to accommodate the demands placed by the automobile.

Thus the Mass Transit Study (MTS) was carried out in 3 phases from 1972 to 1980. In the meantime, an MRT review team from Harvard headed by the late Kenneth Hanson, studied the transportation needs and recommended an all bus network. This proposal was later examined in the Comprehensive Traffic Study (CTS) in 1981 which confirmed previous forecasts that an all bus system would not provide comparable service to a rail network.

In 1982, the Government finally announced its decision to go ahead with the MRT Project. The Protem Committee of the MRT Project subsequently approached the Republic of Singapore Navy (RSN) in August 1982 to assist in providing the overall medical support for the project. The RSN was chosen as it had the experience and facilities to examine and treat divers and compressed air workers.

In 1984 the Ministry of Defence gave approval to the request from Mass Rapid Transit Corporation (MRTC) for the Diving and Hyperbaric Medical Centre (DHMC) to provide the medical support for the compressed air phase of the project. DHMC assisted the Ministry of Labour, in 1984, to draft regulations pertaining to compressed air work. The regulations were adopted by MRTC and the Blackpool Tables were used by the contractors. The manpower and doctors from DHMC enable a comprehensive management and documentation of DCS cases treated.

The Singapore MRT system comprises 41 stations along a 65.8 km route. A total of about 20 km were underground tunnels of which about 11 km were constructed using compressed air. Compressed air was used by 6 contractors for tunnel construction from 21 September 1984 to 17 April 1987. Six contracts were drawn up with the contractors to formalise the agreement to provide medical support. These contracts were:

Tobishima-Takenaka Joint Venture (TTJV)
Contract 104
Bocotra Construction Pty. Ltd (BOC)
Contract 105
Kajima-Keppel Joint Venture (KKJV)
Contract 107
Taisei-Shimizu-Marubeni Consortium (TSM)
Contract 108
Ohbayashi-Gum/Okumura Joint Venture (OOJV)
Contract 109

Nishimatsu-Lum Chang Joint Venture (NLJV)
Contract 301

In addition, the Mass Rapid Transit Corporation, Gammon-Antara Koh Joint Venture, the Industrial Health Division, Ministry of Labour and the Singapore Fire Service have also used the services of the centre.

Methods employed in the construction of the MRT tunnels

The MRT tracks run underground in the central business district. Two construction methods were employed:

TUNNELLING

This method was chosen for construction of the MRT tunnels across the central business district as it minimises the disruption to traffic flow. When the tunnel was below the water table or the soil was unstable compressed air tunnelling was employed. Non compressed air tunnelling was employed in soil and rock which was above the water table. Tunnelling involved the use of various shields. The Full Faced Mechanised Shield consists of a steel cylinder which precisely fits the diameter of the tunnel. The cutting face rotates and removes the earth from the face of the tunnel. As each section is completed, the shield is moved forward by powerful hydraulic rams. Prefabricated concrete liners are then placed inside the tunnel. The shield supports the tunnel face and protects the men working inside. Other variations of this method that were utilised were the Greathead Shield, the Semi-Mechanised Shield and the Fully Closed Shield and the New Austrian Tunnelling Method.

Jet grouting was used to stiffen the soft marine clay encountered in the Kallang region where the tunnels had to go. This process involved the drilling of holes into the ground to a certain depth. The grout, a mixture of cement, chemicals and water, was injected into the soil through high pressure jets which extend horizontally out of the drilling shaft into the surrounding ground. The displaced soil was pumped to the surface and carried away in sludge trucks. This process reduces the surface settlement of the ground and makes the ground more uniform.

CUT AND COVER

This method is a 3 step process. An excavation is first made, then the tunnels are laid in place and finally covered over. This method is much cheaper than bored tunnelling especially if the depth of the tunnels are less than 10 metres below ground level, but it has the disadvantages of noise, dust and disruption to traffic.

For the construction of the tunnel across the Singapore River and Marina Bay, a special method of cut and cover was used with the help of cofferdams. Sheet piles were driven into the river bed on either side of the proposed tunnel line, creating a dammed up central portion, the coffer dam. The water trapped between the two walls was pumped out and a cut and cover method was employed to construct this tunnel.

Planning phase

A study team, led by myself, was sent overseas to study the medical support concept that existed in various countries.

In planning for medical support, we realised that there were two main hazards in tunnelling operations. These were:

1. The hazards of exposure to hyperbaric and closed environments; and
2. The general hazards of tunnelling, industrial and construction work.

Thus our objectives were:

(a) To assist in the preparation of proper legislation for tunnel worksites and compressed air workers.
(b) To provide a comprehensive medical examination for all compressed air workers.
(c) To provide training of personnel working with compressed air.
(d) To provide preventive care and medical treatment to all personnel working in the compressed air environment.
(e) To supervise and cultivate safe work habits and to perform safety inspections and ensure all regulations were adhered to.
(f) To be prepared for all emergencies with the establishment of a 24 hour emergency service with doctors, medics and ambulance service in readiness to meet the exigencies.
(g) To centralise all documentation and data collection so as to expedite data processing and information retrieval.

Preparation

Legislation was incorporated to ensure the safe conduct of construction and tunnelling. Besides the building code which the contractors had to comply with, they had to ensure that all potential compressed air workers underwent a medical examination at DHMC prior to commencement of work in compressed air. The compressed air workers had to attend training courses and be aware of the dangers of working inside the tunnels.

Legislation also detailed the need for medical selection of compressed air workers, periodic clearance, chest

and long bone X-rays, training and proper medical certification of compressed air workers. Regulations pertaining to use of compression facilities by man-lock and medical-lock attendants were also included.

The preparatory phase included the establishment of DHMC as the key Medical Operations Centre and various satellite Medical Centres in the various worksites which provided local cover for the compressed air operations.

Diving and Hyperbaric Medical Centre

DHMC was subdivided into the Clinical and Medical Selection Department, the Operations Room and the Therapeutic Centre. More medical officers were recruited through the Ministry of Defence and were put through a Ministry of Labour approved course to enable them to examine and treat dysbaric osteonecrosis with dysbaric illnesses and to certify them fit for compressed air work.

The senior medical orderlies from DHMC, who were experienced in dealing with diving cases, were also put through the Manlock and Medical Lock Supervisor's course. This enabled them to conduct training for compressed air workers seeking work as medical and manlock attendants.

A new, larger and more advanced hyperbaric chamber was purchased and installed at DHMC to support the treatment of cases of decompression sickness. Medical drugs and various other equipment were also purchased to assist in the therapy and health care of the compressed air workers, e.g.. spirometer and audiometer.

An operational set up with 24 hour manning was established to enable the recall of duty medical personnel at any time of the day to treat cases requiring recompression. Pagers were issued and emergency contact numbers were disseminated to all the worksite offices to facilitate the recall of personnel to the site of the decompression incident. The Operations Room also held the medical records of all the personnel examined for clearance to work in compressed air. It also served as the centre for monitoring the appointments for periodic medical clearance, X-rays, blood and other investigations. Data was collected from the various tunnels, with regard to tunnel pressures, temperatures, humidity, periodic inspections for noise, oxygen, carbon dioxide, carbon monoxide and other contaminant levels. These data were stored in a microcomputer and monthly progress reports were compiled and sent to the relevant authorities.

Medical arrangements at the various worksites

To ensure safety and to provide emergency medical support to compressed air workers, a medical centre was established at each of the sites under the supervision of DHMC. Medical and emergency equipment were in readiness at the centres to cater to exigencies. The set up of the medical centre included a consultation room with couch, drugs, equipment including audiogram, vitalograph and resuscitation equipment. A double lock medical recompression chamber (RCC) was mandatory at worksites which had to work with pressure exceeding 1 Bar gauge. These RCCs were equipped with emergency backup power supply. These were housed in an enclosed area.

Charts of tunnel pressures, humidity, temperatures, gas contents, shift timetables, and various safety/warning messages were also put up in the medial centre. A trained medical officer was present daily when the tunnels were pressurised above 1 Bar gauge. At all times, there was a medical lock attendant on duty to administer immediate medical treatment to workers suffering from pressure related diseases.

Medical Selection

Medical selection of compressed air workers started in April 1984 with the commencement of compressed air work at the Shan Road worksite.

Compressed air workers were divided into those who engaged in manual labour doing shifts of specified duration, supervisory personnel and those with special skills (e.g.. electricians, pyrotechnic experts), who were usually non-shift employees. In addition the men of the Fire Service and Industrial Health Division also had to be examined for suitability to enter into compressed air.

Fitness to work in Compressed Air

The labourers were usually young. Enforcing stringent criteria on age, degree of body fat and freedom from pulmonary and ENT pathology was not a problem. However, the experienced supervisors were usually older and they harboured the usual physical impediments of this age group. In addition, if these supervisors had previous experience with compressed air, they would also have a greater risk of having dysbaric osteonecrosis of the long bones.

Paton and Walder[26] showed that men over 40 had a greater risk of DCS. Age restrictions were imposed. The compressed air workers had to be fit individuals of 18 to 40. Those over 40 were cleared on a case to case basis with stress testing to ensure their fitness as well as the limitations of their exposure to compressed air.

Nitrogen is 5 times more soluble in fat than in water. A man whose body weight comprises 30% fat will have 2,000 ml more nitrogen than a lean man who had only 10% body fat for every 1 atmosphere change in pressure. This predisposes the obese to a greater risk of decompression sickness on account of the large amount of nitrogen gas

released from the tissues during decompression. Therefore, compressed air workers were required to be lean individuals of less than 20% body fat, as measured by the skin fold method. Individuals who were between 20 and 24% body fat were given limitations on the duration and pressure at which they could work.

A comprehensive history was taken to exclude any conditions, especially asthma or ENT conditions, which could place the worker at greater risk of getting barotrauma. Experienced compressed air workers were also screened for past histories of decompression sickness as well as problems of dysbaric osteonecrosis.

The height, weight and body fat were measured. The workers were examined for any ENT condition which might predispose them to sinus, or aural barotrauma. They were assessed on their cardiovascular health and an audiometric examination was performed.

Investigations included their full blood count, urine testing, eyesight testing, serum lipids and cholesterol, chest and long bone X-rays. Stress testing on a bicycle ergometer was required for a worker over the age of 40.

Entrapment of air under pressure within the lungs due to secondary lung pathology can result in serious pulmonary embolism. Congenital cysts, scar tissues, vesicles and emphysematous bullae are possible sites of air entrapment. Many physicians feel that the stethoscope is not efficient in the detection of small lesions. The routine examination, therefore, included spirometry and the measurement of peak expiratory velocity or equivalent quantitative tests of pulmonary function. Chest X-rays were taken prior to commencement of work in compressed air and were repeated yearly. The workers were also given a recompression chamber run to ensure that they were able to equalise the pressure in their ears, sinuses and lungs.

A total of 2,392 potential compressed air workers were seen for pre-employment medical examination. Out of these, 1,737 (72.5%) passed and 655 (27.4%) failed the medical examinations.

Among the failures, ear problems stood out as the commonest cause, accounting for 43.5%. These included perforations of the tympanic membranes and chronic infection of the ears. Heart and lung problems together contributed 31% to the failures. Common pulmonary conditions were asthma and pulmonary tuberculosis. Hypertension and valvular heart conditions were the common cardiovascular problems.

It is interesting to note that 3 cases were rejected because of dysbaric osteonecrosis. All these 3 cases had previously worked in compressed air in other parts of the world. Other causes that disqualified candidates from compressed air work can be seen in Table 1.

TABLE 1

REASONS FOR FAILING MEDICAL EXAMINATION

Condition	Number Failed	%
Ear	285	43.5
Cardiovascular	102	15.5
Respiratory	102	15.5
Chamber Test	36	5.5
Nose	32	4.9
Sinus	19	2.9
Endocrine	14	2.2
Musculoskeletal	10	1.5
Dysbaric Osteonecrosis	4	0.6
Others	51	7.8
Total	**655**	**100.0**

Training of various personnel

To provide medical coverage at all the worksites daily, doctors, nursing staff and medical orderlies had to be trained in management of compressed air illnesses. Training was also administered to man-lock and medical-lock attendants who had the great responsibility of looking after workers who worked in the compressed air tunnels. Other lectures had to be given to safety officers, firemen, ambulance officers, industrial health nurses and compressed air workers on the medical aspects and hazards of compressed air exposure. Some of these courses were administered and approved by the National Productivity Board but the training of the personnel was done by DHMC. The courses conducted are shown in Table 2.

TABLE 2

COURSES CONDUCTED BY DHMC

Courses	Number	Number Trained
Medical-lock Attendants' Instructors (MRT) Course	1	5
Medical-lock Attendant Course	7	36
Man-lock Attendant Course	10	51
Medical-Lock Conversion Course	1	5
MRT Construction Safety	7	55
Compressed Air Course for SAF Medical Officers	3	23
Safety Officer's Course	3	45

Prevention and health care

ROUTINE MEDICAL CONSULTATION

The fluctuation of pressures during entry and exit from the tunnels can potentially cause injury to the air cavities of the body. In particular, the sinuses, middle ear, lung and even air cavities in the teeth can be affected.

In order to prevent compressed air workers from suffering from these pressure related problems or barotrauma, consultation at the worksite was made available. This facilitated early treatment of colds and coughs which may occlude the airways and sinuses of these workers thus predisposing to barotrauma. It was noted that the high humidity in the confined working chamber and dampness from the soil caused more respiratory tract infections than usual.

There were problems of high humidity (almost 100%) and high temperatures (approaching 40 degrees on some occasions) which caused workers to suffer from dehydration, heat exhaustion and an increased risk of decompression sickness. There were also many cases of dermatological problems related to the humid and hot environment.

Regular periodic follow-ups were made compulsory for all compressed air workers. New starter clearance was also conducted at the worksite. This ensured that all compressed air workers remained fit and that any problems were tackled early on.

MONITORING OF WORKSITE AND TUNNEL SAFETY

The medical officer at the worksite was required to monitor the tunnel pressures and the man-lock register daily. The tunnel pressures were recorded on barographs and daily checks were performed to ensure that appropriate decompression was carried out by the man-lock attendant for the workers on the man-lock register. Immediate action was taken to ensure workers did not exceed the Blackpool Tables, which required compressed air workers to spend at least 12 hours at ground level between shifts.

All man-lock attendants had to abide with the proper decompression procedures. They were fined when wrong decompression schedules were used. Those compressed air workers in the man-locks found tampering with the emergency exhaust valves to release themselves early, were also fined.

An MO inspected the worksite and inside the tunnels fortnightly to ensure that there were no unsafe work practices and to reduce the number of accidents occurring through carelessness or ignorance.

Inspection of the man-locks revealed that some man-lock chambers leaked, especially during the last 0.3 bar of pressure. This was due to poor seals and bad alignment of the doors. This problem was further compounded as some man-locks were not level. This was a problem as it meant that the workers were suddenly decompressed when the doors opened unexpectedly. As there was a real danger that the compressed air workers would get DCS, measures were taken by the contractor to modify the chamber doors by including various locking devices to hold the doors shut until decompression was completed. Leaks which required rectification were also found at some of the piping.

With the high humidity and temperatures in some of the tunnels, the contractors were told to install water coolers inside the tunnels to prevent dehydration in the compressed air workers.

Cigarette butts were found on some occasions within the compressed air tunnels, which was alarming. The high partial pressure of oxygen in the tunnel air can cause fires or explosions, especially with naked flames. Immediate action was taken to notify the contractors and engineers to stop such unsafe practices.

However, in spite of the checks, two major accidents still occurred in Contracts 104 and 105 when runaway railway cars smashed into the mud-lock doors of the tunnel causing an explosive decompression. At Contract 105, (tunnel pressure 1.88 bar gauge), 15 men suffered from aural and sinus barotrauma and 4 men had to be treated at DHMC. At Contract 104, (pressure 1.45 bar gauge), all the 15 men inside the tunnel had to be treated simultaneously for DCS at DHMC.

There was a problem with ammonia fumes produced by jet grouting of the tunnel face causing a disruption of the ammonia equilibrium in the soil. This caused the ammonia level to rise to 36 ppm at one stage, resulting in some workers complaining of irritation of the throat, smarting and tearing of the eyes. The problem eventually controlled when the company took steps to wash the tunnel face and to spray the soil with mild acids.

Decompression Sickness

Of special interest in the entire project was the management and treatment of decompression sickness suffered by the compressed air workers.

The population at risk of suffering from DCS was a cohort of 1737 workers who were a multinational lot. There were Thai, Korean, Japanese, Chinese, Indian, Malay and Caucasian workers of various age groups. Both shift and non-shift workers were involved in the contract. The non-shift workers included engineers, supervisors, electricians and fitters who entered the tunnel to perform tasks. They spent variable times in the tunnel and had to be decompressed accordingly.

TABLE 3

SITE OF TYPE I SYMPTOMS

Site	Pain	Skin Rashes	Lymphatic Manifestations
Head	-	1 (Macular Rash)	-
Shoulder	19	-	-
Elbow	16	-	-
Wrist	2	-	-
Abdomen	-	2 (Papular Rash)	-
		1 (Marbling Rash)	1
Inguinal Region	-	-	1
Hip	4	-	-
Knee	108	-	-
Lower Leg	-	1 (Itchy, Macular)	-
Ankle	5	-	-
Total	**154**	**5**	**2**

For ease of reporting and presentation, the cases of DCS were classified into two types after Golding et al.[27]

Type I (mild) Symptoms and signs were mild and present as musculoskeletal pain or swelling due to lymphatic obstruction and skin involvement.

Type II (serious) Symptoms and signs were severe and attributable to disorders of the nervous, pulmonary and cardiovascular systems.

Various factors were analysed including the host characteristics, duration of onset, age, length of exposure, pressure, number of episodes of DCS, overall incidence, relationship to shift-work and physical environment of the worksite, treatment methods and outcome.

Data on the incidence of dysbaric osteonecrosis was obtained by review of the long bone X-ray reports performed yearly as required by legislation. Factors with regard to pressures of exposure and other host characteristics like obesity, race and number of episodes of DCS were analysed.

SYMPTOMATOLOGY

There were 164 cases of DCS. 160 were of the milder Type I category while 4 were of the more serious Type II category.

Type I

The commonest presentation of Type I DCS in this series was pain (154 cases or 96.3%). The commonest site of pain was around the joints. 84 cases (55%) presented with monoarticular pain while 70 cases (45%) presented with polyarticular pain. In 76% of the cases the joints of the lower limb were involved. The knee joint was the commonest joint involved (108 cases) followed by the shoulder joint and the elbow joint (Table 3).

The characteristic nature of the pain noted was that it was deep joint pain, aggravated by movement. There was also a limitation of the range of joint movement.

There were 5 cases (3.1%) of cutaneous DCS. Two cases presented with papular rashes over the abdomen. Two cases had macular rashes, over the forehead and over both shins respectively. One case presented with a marbling rash over the abdomen. One case of lymphatic DCS was seen involving the inguinal lymphatics and presenting as swelling of the penile skin (Table 4).

Type II

Table 5 illustrates the clinical presentation of the 4 Type II cases seen in the MRT project. Three of them were exposed to compressed air for periods exceeding 8 hours. All of them presented within 2 hours of decompression. Case number 4 developed pulmonary DCS following an explosive decompression of the tunnel when a runaway locomotive smashed open the mud-lock door.

ONSET OF SYMPTOMS

94.5% of the Type I and II cases developed symptoms within 12 hours of decompression (Table 6).

TABLE 4

CLINICAL PRESENTATION OF TYPE I DCS

Symptomatology	No. of Cases
Pain:	
Deep Pain	133
Superficial	7
Constant	24
Throbbing	5
Radiation of Pain	3
Limitation of Movement	16
Rashes:	
Erythematous, Papular, Itchy	2
Erythematous, Macular, Itchy	2
Marbling Rash over abdomen	1
Lymphatic Swelling	2
Itching	5
Warmth Around Joints	8
Numbness	16

EPISODES OF DCS

Of 1,737 people who worked in compressed air from September 1984 to April 1987, 136 (7.83%) suffered from DCS. Of these 136 men, 114 (83.5%) suffered from a single episode of DCS while 22 (16.2%) had two or more episodes of DCS. Table 7 shows the distribution of DCS episodes amongst the 136 affected. The maximum number of episodes of DCS in any one man was 4.

INCIDENCE WITH PRESSURE AND DURATION OF EXPOSURE

A total of 188,538 man decompressions were performed in the MRT project. There were 160 cases of mild (Type I) DCS and 4 cases of severe (Type II) DCS, giving the overall incidence of DCS at 0.087%. There were 64,059 man decompressions over 1 Bar gauge, with 154 cases of DCS, giving an incident rate of 0.240%.

A study of the compressed air exposure time of the 164 cases showed that the majority, 125 cases (76.3%), occurred after exposure times exceeding 8 hours as seen in Table 8.

TABLE 5

CLINICAL PRESENTATION OF TYPE II DCS

Case Number	Working Pr (Bar)	Exposure Time	Onset of Symptoms Following Decompression	Clinical Presentation	Treatment
1 (N S)	1.75	8 hrs 5 mins	1/2 < 1 hr	Pain - left knee and hip joints. Loss of sensation L3, 4 bilateral. BP 110/70. 2 previous episodes of Type 1 DCS.	CIRIA 2
2 (A Y)	1.53	8 hrs 23 mins	1/2 < 1 hr	Pain - right knee. Loss of sensation to pinprick over right half of body.	CIRIA 2
3 (K P)	1.45	8 hrs 32 mins	1/2 < 2 hrs	Felt weak & giddy 70 min after leaving manlock. Noted to be staggering & vomiting but no nystagmus, visual or auditory symptoms. BP 110/70. Relapsed with giddiness and low BP of 90/50 & vomiting on sitting.	CIRIA 2 and TABLE 62
4 (I H)	1.4	6 hrs 25 mins	< 1/2 hr	Sudden decompression accident. Pain - Both knees. Aural barotrauma bilateral. Chest pain & dyspnoea. BP 140/80 Pulse 84.	TABLE 62

TABLE 6

ONSET OF SYMPTOMS OF DECOMPRESSION SICKNESS

Time	**< 1 Hr**	**1 < 4 Hrs**	**4 < 6 Hrs**	**6 < 12 Hrs**	**12 < 24 Hrs**	**> 24 hrs**	**Total**
Number of Type I	32	68	32	19	1	8	**160**
Number of Type II	3	1	0	0	0	0	**4**

TABLE 7

NUMBER OF EPISODES OF DCS

No. of Episodes of DCS	1	2	3	4	Total
No. of Men Affected	114	17	4	1	136
Percentage of Men Affected	83.8	12.5	2.9	0.8	100%

TABLE 8

INCIDENCE OF DCS WITH PRESSURE AND DURATION OF EXPOSURE

Maximum Working Pressure (Bars Gauge)	**DCS Type**	**Decompression Sickness Incidence In % (brackets show absolute figures)**			**DCS Incidence (by Type of DCS %)**	**Total Incidence %**
		< 4 Hours	**4-8 Hours**	**> 8 Hours**		
</1 Bar	Type I	0.00 (0)	0.00 (0)	0.027 (10)	0.008 (10)	**0.008%**
	Type II	0.00 (0)	0.00 (0)	0.00 (0)	0	
No. of Man-Decompressions.		60,976	27,080	36,423	12,4479	
1-2 Bar	Type I	0.016 (4)	0.310 (32)	0.359 (98)	0.215 (134)	**0.221%**
	Type II	0.00 (0)	0.001 (1)	0.011 (3)	0.006 (4)	
No. of Man-Decompressions.		24,596	10,423	2,7331	62,350	
2-3 Bar	Type I	0.00 (0)	1.786 (2)	1.724 (14)	0.936 (16)	**0.936%**
	Type II	0.00 (0)	0.00 (0)	0.00 (0)	0.00 (0)	
No. of Man-Decompressions.		785	112	812	1,709	
TOTAL	**Type I**	**0.005 (4)**	**0.090 (34)**	**0.193 (122)**	**0.085**	**0.087%**

The incidence of DCS at less than 1 Bar gauge exposures was 0.008%. There were 10 cases in 124,479 man decompressions. This unexpected finding will be discussed in another paper.

The incidence of DCS at pressures between 1 to 2 Bar gauge was 0.218% with 138 cases in 62,350 exposures. Between 1-2 Bar, for durations less than 4 hours, the results again were significantly lower ($p < 10\text{-}6$) than the mean values. At exposure times of greater than 4 hours however, the number of cases of DCS observed were significantly higher than the expected values for the exposures of longer than 4 hours duration (both p values < 10-6) (Table 8).

The incidence of DCS occurring at pressures greater than 2 Bar gauge was 0.936%, with 16 cases in 1,709 exposures. Above 2 Bar gauge pressure, with duration of exposure less than 4 hours, there were no significant variations between the observed and the expected number of DCS cases. However, the observed increase in the number of cases of DCS was statistically significant for exposures between 4-8 hours ($p < 0.03$) and exposures greater than 8 hours ($p < 10\text{-}6$) (Table 8).

INCIDENCE OF DCS AND HEAT AND HUMIDITY

The incidence of DCS did not appear to correlate significantly with the observed high temperatures and humidity of some tunnels. The highest temperatures were recorded at Contract 105, where temperatures reached a high of 42 degrees. Most tunnels had relatively high humidity above 80%. At Contracts 107 and 108, there were occasions where humidity reached 100% .

DCS AND OBESITY

Few of the compressed air workers were obese. However, there were 3 cases of DCS amongst the 26 personnel who had more than 30% body fat during initial medical clearance (Table 9).

DCS AND OCCUPATION

The distribution of cases among the various occupations is shown in Table 10.

INCIDENCE OF DCS BY CONTRACTS

The maximum working pressure in the various contracts varied, the lowest being 1.43 Bar gauge in Contract 105, and the highest, 2.35 Bar gauge in Contract 301. The incidence of DCS by contracts is given in Table 11.

SHIFT-WORK

The incidence of DCS was evenly distributed among the 3 shifts.

DCS BY RACE

There appeared to be a slightly higher incidence of DCS occurring in the Malay population as compared to the other races. However, in view of the small number of man decompressions undertaken by this group of compressed air workers compared with the other groups, the findings were not significant ($p > 1.0$) (Table 12).

Treatment

Treatment primarily consisted of recompression with supportive drug therapy. Recompression in most instances were carried out at the medical-locks of the various work-sites. Following the recompression therapy, 100% surface oxygen was administered ensuring no relapse of symptoms (Table 13).

The CIRIA air therapeutic tables were used in most (73.9%) of the cases. The CIRIA table for Type 1 DCS involves returning the patient to the original working pressure, then a stepwise 0.1 Bar decompression every two minutes. When the pressure has been reduced to half the working pressure the rate of ascent is reduced to 0.1 Bar every 25 minutes. The table for Type II DCS involves returning to the original working pressure. If the symptoms are not relieved at this pressure, pressure is increased in increments of 0.1 Bar. Normally with an extra 0.6-0.7 Bar the patient loses his symptoms. He is then held at that pressure for 15 to 30 minutes before pressure is reduced by 0.1 Bar every 25 minutes. The patient is held at 1 Bar gauge for 6 hours and then decompressed at 0.1 Bar every 45 minutes. There is a 90 minute hold at 0.5 bar gauge after which decompression is continued at the same rate. The CIRIA tables were used as not all the medical-locks at the work-sites were equipped with built-in oxygen breathing systems. In resistant and relapsed cases, Workman and Goodman oxygen therapeutic tables were used by bringing oxygen breathing apparatus into the chamber or by transporting the patient to DHMC.

Of the 164 cases treated, 4 (2.44%) cases relapsed after the first treatment and had to be treated with hyperbaric oxygen. None of the 39 (23.78%) cases treated with hyperbaric oxygen had a relapse (Table 13).

Dysbaric osteonecrosis

Of the 1,737 compressed air workers who were certified fit to work in compressed air, 32 had previous type B dysbaric osteonecrosis lesions while 11 had other benign lesions such as bone islands. The compressed air workers with type B dysbaric osteonecrosis were allowed to work in compressed air, with exposure limited to not more than 2 Bar gauge. Follow up yearly long bone X-rays showed no new

TABLE 9

DCS AND PERCENTAGE OF BODY FAT

Percentage of Body Fat	Number of DCS Cases	Number of Man Decompressions	Incidence
< 20 %	156	181,813	0.086%
24 - 30 %	5	5,586	0.090 %
> 30 %	3	1,139	0.262%
Total	**164**	**188,538**	**0.087%**

TABLE 10

INCIDENCE OF DCS BY OCCUPATION

Occupation	Number of DCS cases	Number of Man-Decompressions	% Incidence
Compressed air worker	110	110,943	0.099
Engineer	15	51,050	0.029
Supervisor	23	12,883	0.179
Others	16	13,701	0.117

TABLE 11

INCIDENCE OF DCS BY CONTRACT

Contract	Maximum Pressure (Bar)	Number of DCS cases			Number of Man Decompressions		Incidence of DCS					
							Overall %			Over 1 Bar %		
		Type I	Type II	Total	Total	Above 1 Bar	Type I	Type II	Total	Type I	Type II	Total
104	1.50	36	1	37	79,363	39,064	0.045	0.001	0.046	0.092	0.002	0.094
105	1.43	3	0	3	27,976	937	0.011	0.0	0.011	0.320	0	0.320
107	1.60	31	0	31	8,757	2,679	0.354	0.0	0.354	1.157	0	1.157
108	1.95	26	1	27	19,520	6,666	0.133	0.005	0.138	0.390	0.015	0.405
109	1.50	28	2	30	38,110	5,550	0.073	0.005	0.078	0.505	0.036	0.541
301	2.35	36	0	36	14,812	9,163	0.019	0.0	0.019	1.746	0.044	1.790
TOTAL	**-**	**160**	**4**	**164**	**188,538**	**64,059**	**0.085**	**0.002**	**0.087**	**0.250**	**0.006**	**0.256**

TABLE 12

INCIDENCE OF DCS BY RACE

Race	Number of DCS cases	No. of Man-Decompressions	% Incidence
Chinese	39	45,008	0.0867%
Malay	14	15,849	0.0884%
Indian	34	39,064	0.0870%
Japanese	20	23,035	0.0868%
Korean	18	20,539	0.0876%
Thai	35	40,239	0.0870%
Caucasian	4	4,804	0.0834%
Total	**164**	**188,538**	**0.0870%**

TABLE 13

TREATMENT USED

Recompression Table	Contracts 104	105	107	108	109	301	Total
CIRIA 1	35	-	26	21	19	14	115
CIRIA 2	1	-	1	2	(2)	-	4(2)
TABLE 61	-	3	4	3	9	1	20
TABLE 62	(1)	-	-	-	-	18	18(1)
CIRIA 1 followed by Table 61	-	-	-	-	-	1	1
CIRIA 1 and 2 followed by Table 62	-	-	-	(1)	-	2	2(1)
Total	**37**	**3**	**31**	**27**	**30**	**36**	**160(4)**

Numbers in brackets denote cases of Type II DCS. There were 160 cases of Type I and 4 cases of Type II DCS

cases of dysbaric osteonecrosis and those compressed air workers who had pre-existing dysbaric osteonecrosis did not have any further change seen. Only 643 compressed air workers had exit long bone X-rays done at the end of their contract (Table 14).

Incidence of DCS in Singapore

The incidence of DCS in the Singapore MRT project was low when compared with compressed air work done elsewhere in the world. (Table 15)

The main reason for the low incidence is the generally low pressures that were used. The highest pressures were at Contract 301, which had a maximum tunnel pressure of 2.35 Bar gauge.

64.63% of our 164 cases of DCS occurred with exposures exceeding 8 hours. A higher incidence of DCS was also noted with increasing pressures (Table 8). This was observed with the incidence of DCS rising from a low of 0.008% to 1.61% for exposures exceeding 8 hours. With the increase in duration of exposure, there was an overall increase in the DCS incidence. For exposures greater than 2

TABLE 14

ABNORMAL LONG BONE X-RAYS OF COMPRESSED AIR WORKERS

	Abnormal Entry Long Bone X-rays		Abnormal Exit Long Bone X-rays	
Contract	**Type B**	**Benign Orthopaedic Conditions**	**Type B**	**Benign Orthopaedic Conditions**
MRT Task Force*	1	2	1	2
109	0	0	0	0
108	0	0	0	0
104	0	0	0	0
107	2	2	0	0
MRTC	8	0	3	0
105	2	1	0	0
301	19	6	5	6
Total	**32**	**11**	****9**	****8**

** Only 643 compressed air workers had done their exit Long Bone X-rays.
* MRT Task Force comprised firemen from the Singapore Fire Service.

TABLE 15

COMPARISON OF VARIOUS COMPRESSED AIR CONTRACTS

Contract	**Period of Compressed Air (Months)**	**Total No. of workers**	**Maximum Pressure (Bar)**	**No. of Man-Decomp.**	**No. of DCS Cases (Over-all)**	**DCS Incid. Over-all**	**DCS Incid. (>1 Bar)**
East River Tunnel New York							
1914-21	84	-	3.26	1,360,000	680	0.05%	-
Howrah Bridge India 1938	6	509	2.72	12,400	353	2.8%	-
Lincoln Tunnel NY 1955-56	18	704	2.31	138,000	44	0.03%	0.07%
Dartford Tunnel 1957-59	24	1200	1.90	122,000	685	0.56%	0.97%
Blackwall Tunnel 1960-64	44	1536	2.65	8,100	863	1.1%	1.09%
Tyne Road Tunnel 1960-64	38	650	2.86	44,800	711	1.6%	1.74%
Hong Kong Islandline							
1982-85	36	3966	2.85	443,430	2003	0.46%	0.52%
Singapore MRT 1984-87	**31**	**1737**	**2.35**	**188,538**	**164**	**0.087%**	**0.26%**

Bar absolute, due to the exigencies of the project, we had to increase the decompression time as the Blackpool Tables do not indicate decompression times for exposures greater than 8 hours. This had in a way, prevented more cases of DCS from occurring.

The higher incidence of DCS noted at the extremes of exposure indicate that far greater risks are associated with long exposures with the use of the Blackpool Tables in spite of adequacy of control. It is likely that with longer working hours, the workers have absorbed grater amounts of gases and have also been subjected to greater stresses involving the use of vibrating tools and lifting heavy loads over long distances in the tunnels. The cumulative factors resulted in the development of DCS in some men who appeared to be more susceptible to developing DCS than their peers who

had been similarly exposed to the same pressures and performed the same type of work.

The overall incidence of the 4 cases of Type II DCS was 0.002% compared with 1 case (0.001%) reported by Lam's Hong Kong MTR series out of 93,509 man decompressions[28]. Three of the Type II cases developed DCS after more than 8 hours exposure at 1.45 Bar and above. The fourth case developed DCS soon after suffering from sudden decompression, when the mud-lock doors were smashed open by the runaway rail cars. Three out of four developed symptoms within 1 hour of decompression, while the fourth case had symptoms within 2 hours of decompression.

The incidence of DCS among the obese compressed air workers was higher than in the other compressed air workers. Nitrogen had been shown to be 5 times more soluble in fat than in lean tissues like muscle. With long exposures, it is expected that there is near saturation of the fatty tissue by nitrogen in the obese person. This results in a 5 times greater gas load during decompression. Therefore it was not surprising that despite conditionally passing the obese compressed air workers and allowing them to work at a limited exposure time and pressure, they still had a significantly higher incidence of DCS.

The supervisor category of personnel had an overall greater incidence of DCS when compared with the other categories of workers. This is related to the nature of work and the fact that supervisors were generally of an older age group. They were required to enter and exit from the tunnels up to five times a day, increasing their risk of DCS. The compressed air workers, in comparison, although were performing heavier work, exited only once per shift.

The incidence of DCS among the various races were fairly similar, with exception of the Caucasians, who had a lower DCS incidence. This is due to the fact that the Caucasians held specialist appointments like engineers and inspectors and had short exposure times in the course of their work.

Reasons for low incidence

MEDICAL STANDARDS

No compromise was made with regard to the selection of men working in compressed air. Workers who were susceptible to DCS were certified unfit or conditionally cleared to work in compressed air. This included the obese (> 24% body fat by skinfold measurement), those with a high incidence of DCS in the past and compressed air workers with established dysbaric osteonecrosis. The obese (over 24% body fat), and those with Type B dysbaric osteonecrosis were given a conditional clearance. They were allowed to work at a limited pressure and for a limited duration.

TRAINING

The man-lock attendants were required to attend certification courses conducted by DHMC in conjunction with the National Productivity Board and Ministry of Labour. In addition, all compressed air workers were thoroughly briefed on the safety aspects of compressed air work. They were taught the importance of proper decompression and the signs and symptoms of early DCS. Some compressed air workers found tampering with the emergency exhaust valves in the man-locks to release themselves early, were disciplined. The strict control of the decompression procedure paid off and prevented DCS occurring due to negligence.

LEGISLATION AND CONTROL OF DECOMPRESSION PRACTICE

The legislative framework determined the requirement for the control of the time of exposure. In addition, we believed that a period of acclimatisation for new starters did much to reduce the incidents of DCS. This was incorporated in the legislation for all new compressed air workers as well as those who have been away for more than 12 consecutive days. Paton and Walder[26] noted a high incidence of DCS in newly introduced workers, but with acclimatisation, the incidence fell. As a result of the regulations, we did not see any cases of DCS in new starters.

The Blackpool Tables, designed by Hempleman, were well tested in the UK and Hong Kong with a DCS incidence rate of less than 2%. For exposures less than 1 Bar gauge, compressed air workers were decompressed to the surface at a rate of not exceeding 0.4 bar/min. However, in the Singapore MRT project, a stop at 0.2 Bar gauge for 5 minutes was included into the regulations. This stop was included to reduce the rate of ascent further as we believed that it would have been to risky to ascend immediately to the surface after more than 10 hours of exposure.

Compressed air workers exposed to pressures more than 1 Bar gauge had to follow the decompression schedules according to the Blackpool Tables. These tables required compressed air workers to remain at the surface for at least 12 hours in every 24 hours. This was because at the end of decompression, residual nitrogen still remains in the body. This will accumulate without an adequate rest period at atmospheric pressure.

Multiple entries into the compressed air tunnels were allowed for the supervisors. By law, they were allowed to enter the chambers only 5 times in a 24 hour period at pressures not greater than 2 bar gauge, for not more than half an hour on any one occasion and with a minimum surface interval at normal pressure of more than 1.5 hours.

There were instances where compressed air workers were exposed to compressed air exceeding the time limits

imposed by the Blackpool Tables, either by misinformation or the exigencies of work. This posed a problem of decompression as the Blackpool Tables were calculated with a maximum of 8 hours exposure. In order for them to work at these long hours, extension of the Tables were required and additional stops had to be included as a safety measure. The incidence of DCS in the extended part of the Tables was lower than the incidence following decompression with the Blackpool Tables.

Clinical presentation

The commonest presentation of Type I DCS in our series was that of joint pain (Table 3). This occurred in 96.3% of cases with 76% of cases having pain in the lower limbs. Pain was mostly around the joints of the long bones. 84 cases (55%) presented with monoarticular pain while 70 cases (45%) presented with polyarticular pain. 76% had pain in their lower limbs compared with 87.82% reported Lam in the Hong Kong MTR project.[28] The knee was most commonly affected in our series (108 cases or 70.1%), followed by the shoulder joint (19 cases or 12.3%) and the elbow (16 cases or 10.4%).

A detailed analysis of the onset of symptoms revealed that 82.9% of cases had symptoms within 6 hours of decompression, with 95.1% of cases presenting before 24 hours. This is comparable with Lam's Hong Kong MTR series with 93.6% presenting within 6 hours.[28]

There were 5 cases of Type 1 DCS presenting as rashes. These cases proved difficult to diagnose. Recompression confirmed the assessment. Two cases presented as swelling of the lymphatics, one of who had lymphatic swelling over the inguinal region which also subsided with recompression. In the Hong Kong series, 4 cases had skin mottling, 39 had non-specific symptoms of headache, nausea and vomiting. The other 94.6% (749) cases presented with joint pains.

Treatment

Early recognition of symptoms and prompt treatment was ensured by making it compulsory for compressed air workers to remain at the worksite for 2 hours following decompression from the tunnel. This probably accounted for the fairly low relapse rate of 2.44% (4 cases). By comparison, 8.2% (64 cases) required a second treatment and 11 (1.4%) required a third treatment in the Hong Kong series.

Adjuvant therapy including fluid replacement was used. Fluids included Dextran in addition to Normal Saline and Hartmann's Solution. Aspirin was not given to the compressed air workers because of the possibility of masking the symptoms.

The objective in fluid therapy was to replace depleted blood volume, to restore haematocrit and to prevent blood sludging and to improve tissue perfusion. It is common to find that patients with acute DCS have reduced blood volumes, and many animal studies have confirmed this finding. Dextran has advantages for restoring intravascular fluid lost by increased capillary permeability during DCS[29], although there are those who are more concerned with the potential of Dextran for creating an acute volume overload and further lung congestion. Our experiences with Type 1 DCS and delayed Type II DCS has shown us that judicious use of Dextran, especially in the young, robust compressed air workers or fisherman divers, reaped benefits in the improvement of symptoms in our patients. We advocate the combined use of fluid therapy with colloids and crystalloids, aspirin, dexamethasone and recompression therapy in DCS therapy.

Accidents

Two major accidents occurred in Contracts 104 (Bocotra at Orchard Road) and 105 (TTJV at Novena Station) when runaway rail cars smashed the mud-lock doors of the tunnel causing an explosive decompression. At Contract 105, (tunnel pressure : 0.88 Bar gauge), 15 men suffered from aural and sinus barotrauma and 4 had to be admitted and treated at DHMC.

At Contract 104, the tunnel pressure was 1.45 Bar gauge. There were 15 men inside the tunnel while 2 men were working around the mud-lock. The men had been working for seven and a half hours. Five men developed symptoms of DCS and the operations centre at DHMC was thrown into full alert. Ambulances were sent to the site and doctors were deployed to three recompression chambers treating the patients simultaneously. One of the workers working around the man-lock was thrown 10 metres by the out-rushing air. He was evacuated to hospital with head and body injuries. The number of cases of DCS would have been less (159 instead of 164) except for this accident.

The future

The future of compressed air tunnelling will see the use of oxygen for decompression. Oxygen decompression was suggested as early as 1878 by Paul Bert[15], and 1905 by Ham and Hill[30]. The advantages that oxygen decompression offer are reduction of the decompression times and perhaps a lower DCS rate. Oxygen tables are available for decompression and, while they reduce the amount of time spent decompressing, they have inherent disadvantages. Man-locks will have to be modified to incorporate oxygen built-in breathing systems (BIBS) and an oxygen overboard dump. This will incur increased costs. High pressure oxygen, being a fire hazard, will require special care during the decompression. No flammable articles can be brought

into the chambers. Proper use of the masks for oxygen breathing during decompression will have to be ensured if DCS was to be avoided. The workers may develop CNS oxygen toxicity, and a doctor will have to be at the man-lock to supervise the decompression. The Japanese have had bad experiences with oxygen decompression, as fire and deaths have occurred in their man-locks.

The Blackpool Tables and the current American OSHA tables are still not perfect. Kindwall et al are currently evaluating the use of oxygen tables to supersede the current United States Occupational Health and Safety Agency (OSHA) Decompression Schedules. Oxygen tables are currently being used by the French and Germans for decompression from tunnelling work, but these have not been widely adopted elsewhere. These tables adopt profiles similar to those of dive tables, but incorporate oxygen in order to shorten the decompression times.

Automation and robotics may be featured more prominently in future, where tunnels may be dug using unmanned devices. Alternatively, the compressed air worker may adopt a lightweight armoured suit as used by the deep sea diver, where the worker can remain at 1 atmosphere pressure and perform tasks without the need for decompression. Saturation compressed air tunnelling could be another method which may be adopted for the future.

Conclusion

Decompression sickness is a preventable condition in compressed air work. The prevention of this illness is to a large extent dependent on the recognition of the hazards involved and in the application of recognised medical and environmental control measures. The DCS incidence of 0.087% in the Singapore experience was low and the medical team involved in the project can look back at the months of compressed air work with much satisfaction. The attention to detail during planning and the adoption of strict medical standards in selection of men and in safety control ensured that no case of DCS ever occurred out of ignorance or poor compliance with safety regulations. The preventive measures undertaken enabled a fairly good safety record to be achieved.

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PROJECT STICKYBEAK

This project seeks to document all types and severities of diving-related accidents. Information,which is treated as being CONFIDENTIAL in regards to identifying details, may be sent to:

Dr D. Walker
P.O. Box 120, Narrabeen,
N.S.W. 2101,Australia.

ASSESSMENT OF THE PNEUPAC HC HYPERBARIC VENTILATOR

A.J. Gibson, F.M. Davis and A.R. Wilkinson

Introduction

The PneuPac HC Hyperbaric Ventilator (Figure 1) has been specifically designed to ventilate patients within hyperbaric chambers over a range of pressures up to 10 ATA (atmospheres absolute). It is a standard pneumatically controlled, time-cycled ventilator providing independent control of inspiratory time, inspiratory flow rate and expiratory time. The range of these parameters has been significantly extended to allow compensation for the changes in ventilator performance with different chamber pressures.[1] Following the manufacturer's advice, we carried out calibration of the PneuPac HC ventilator to derive a series of calibration tables for its clinical use.

Description of PneuPac HC ventilator

The HC Ventilator consists of a pneumatic control module operating a remote patient valve housed in the patient connection block. These are linked by a small diameter flexible hose which can be separated to allow sterilisation of the patient valve. The control module is operated from compressed air, oxygen or a helium/O_2 mixture which is delivered from a regulator within the chamber, set at 400-1000 kPa gauge pressure. A simple schematic diagram of the PneuPac HC is shown in Figure 2.

The control module has three control knobs; one each for the inspiratory and expiratory times which are each arbitrarily graduated from one to nine, and an inspiratory flow control which completes eight and a half revolutions between its minimum and maximum settings. As well, there is an on/off switch and a pressure gauge. All moving parts of the control module are manufactured to require no lubrication or maintenance.

On connecting the ventilator to the gas supply and switching on, the spool in valve B is initially biased to allow the gas to flow from port 1 to port 2 from where it will flow to the inspiratory timer valve C and to valve F and thence to the patient valve.

The inspiratory time knob restricts the gas flow into the inspiratory timer cartridge C which then fixes the rate of pressure rise within C. At a predetermined pressure, a piston within C moves to allow gas to flow to port 4 of valve B which in turn switches the spool of valve B to the expiratory position. Now the gas flow is directed from port 1 to port 3 and thence to the expiratory timer cartridge D which behaves in a similar way to the inspiratory cartridge. During expiration, a non-return valve within C opens to release the pressure within this cartridge. The expiratory time knob is

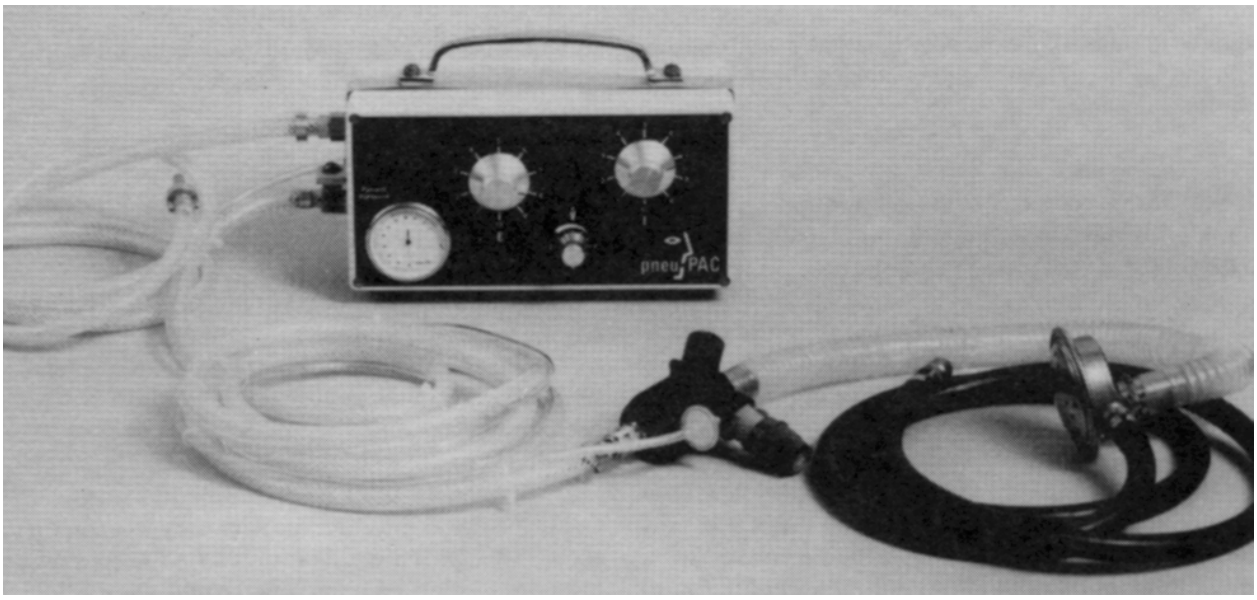

Figure 1. ThePneuPac hyperbaric ventilator control box showing (from left to right) the inflation pressure gauge, expiratory time control (E), flow control(V) and inspiratory time control (I). The patient control valve is in the middle foreground with the overboard dump system via a Scott exhalation regulator on the right.

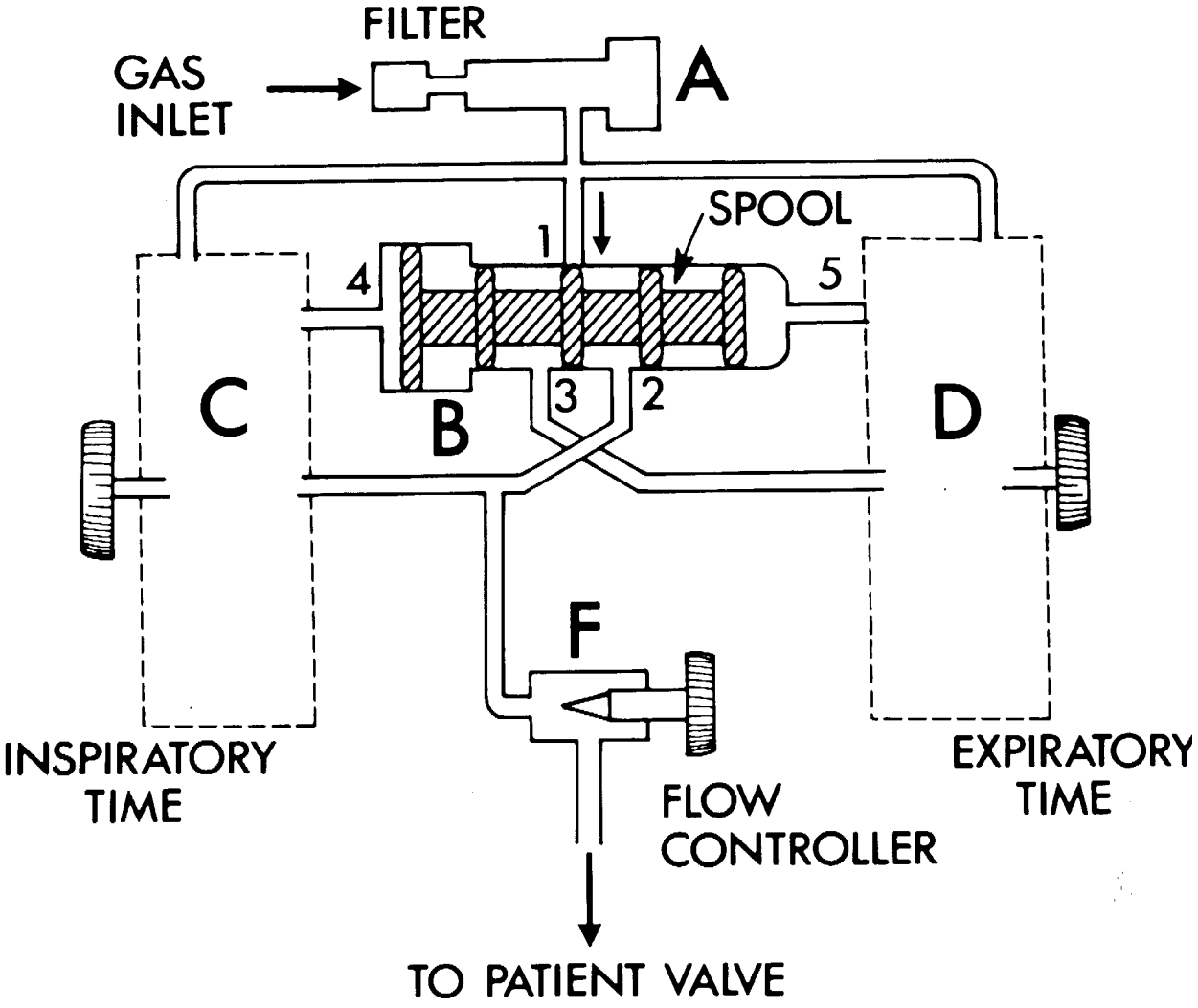

FIGURE 2. Schematic diagram of Pneupac HC ventilator. See text for explanation of terms.

adjusted to control the expiratory time and is used together with the inspiratory time to determine the desired respiratory rate for the patient.

During inspiration, gas is delivered to valve F, one side of which is connected to the flow controller and thence to a reducing valve, the other side to the gas outlet and thence to the patient via the patient valve.

The patient valve operates by using the pressure energy in the gas flowing from the control module. During inspiration, the gas flow to the patient valve passes through an orifice which causes the pressure to drop to patient pressure. The resultant pressure on the end of a piston pushes it against a spring which closes the exhaust port and connects the patient to the gas flow from the orifice. The patient is protected from over-inflation by a pressure relief valve (two are provided, with blow-off pressures of either 40 or 60 cm H_2O). During expiration, the spring forces the piston back to open the exhaust port, the patient exhaling through the valve and out the exhaust port. The patient valve is fail-safe in that should the module be switched off or the gas supply fail, the valve opens to the atmosphere.

Methods

The PneuPac HC ventilator was tested using a simple circuit which included a calibrated Wright's spirometer and a "mock lung" over a range of inspiratory time and flow settings at 1.9 ATA, 2.8 ATA and 6 ATA.

CALIBRATION OF WRIGHT'S SPIROMETER

Using a 2 litre Rudolph gas calibration syringe, the volumes recorded by a Wright's spirometer were checked at each pressure. This was done by taking the average of three readings in 200 ml steps up to 2000 ml, each tidal volume being delivered in approximately one second.

INSPIRATORY TIMES

With a digital stopwatch, the inspiratory time for the ventilator on each of its graduations on the inspiratory time control was measured at each pressure. For each time setting, the inspiratory time was taken as the average of five readings.

DELIVERED TIDAL VOLUME

For each inspiratory time graduation, the flow control was then adjusted from its minimum to its maximum setting (eight-plus revolutions) with the tidal volume being measured using the spirometer. Five readings were taken at each complete revolution between the minimum and maximum settings and averaged. Throughout the measurement of tidal volume, the expiratory time was left on its maximum setting for convenience, since changes in its setting did not influence the inspiratory time. The temperature and humidity within the chamber were also recorded. These measurements were repeated at each pressure.

Results

Calibration of the Wright's spirometer at 1.9 and 2.8 ATA showed that it consistently over-read the tidal volume by approximately 8% at low tidal volumes (< 500 mls), reducing to 5% at higher tidal volumes. This error was slightly greater (11% down to 6%) at 6 ATA. Subsequent recordings of the tidal volumes measured from the ventilator were corrected by these factors and standardised for temperature and humidity.

Inspiratory times progressively shortened with increasing ambient pressure, such that they were on average about 35% shorter at 6 ATA than at 1.8 ATA. At 6 ATA the longest inspiratory time that could be achieved was 1.25 sec. Expiratory times at any given setting shortened with increasing pressure in a similar manner. Ventilation rates down to about 12 breaths per minute were still achievable at 6 ATA. We found that the clinically useful inspiratory times (0.75 - 1.5 sec) were within the control setting range from 6 to 9.

The tidal volume at each setting was measured five times and averaged. There was seldom more than 10 ml variation within each group of readings, demonstrating consistent performance of both the ventilator and the spirometer. As an example, Table 1 gives the results for the tidal volumes generated at 50 m depth for each of the inspiratory time settings as the flow rate is increased stepwise by one revolution of the flow control from its minimum to its maximum setting. At lower chamber pressures, readings were not taken at the longer inspiratory times at high flow rates as the tidal volumes generated were well beyond the clinically useful range. Similar tables were constructed for 1, 1.9 and 2.8 ATA pressures.

Figures 3, 4 and 5 represent graphically the data at each of the 3 depths for a minimum, intermediate and maximum flow setting, over a clinically useful range of inspiratory times.

Discussion

As would be expected for a time-cycled pneumatically-controlled ventilator, the performance changes as the chamber pressure is increased or decreased. Blanch et. al.[1] have described the performance characteristics of a range of ventilators under hyperbaric conditions. The changes observed by us with the PneuPac HC are closely consistent with their data for other time-cycled devices. However, the extended range of the PneuPac HC, with a maximum flow capability of 180 litre min^{-1}, allows compensation for the decrement in performance over a range of chamber pressures at least up to 6 ATA. Beyond 6 ATA it is doubtful that this ventilator would meet the needs of most adult male patients.

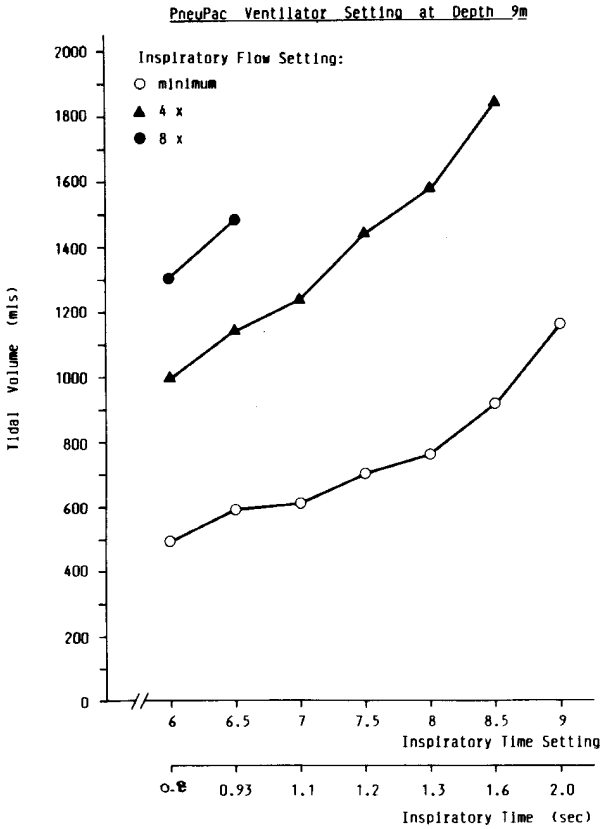

FIGURE 3. Tidal volumes generated at 1.9 Ata pressure at three different flow settings.

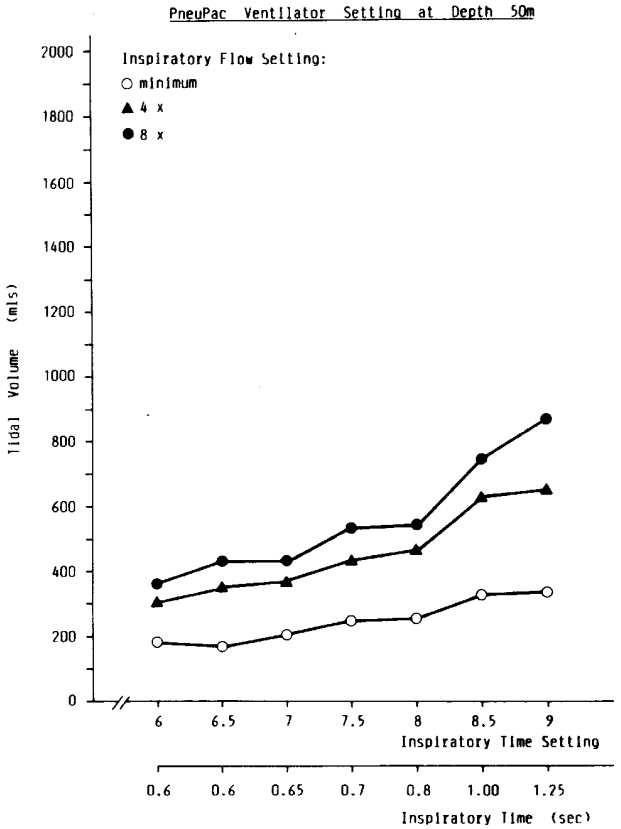

FIGURE 5. Tidal volumes generated at 6 Ata pressure at three different flow settings.

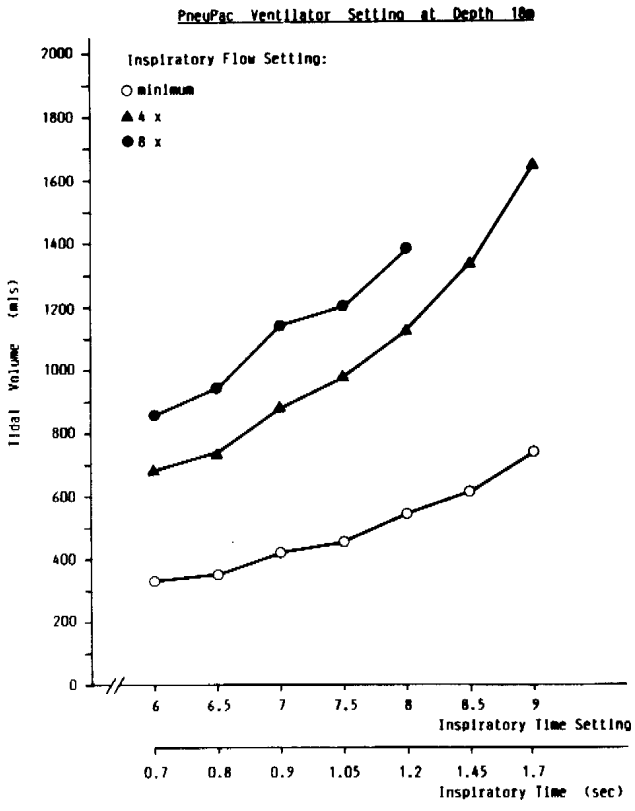

FIGURE 4. Tidal volumes generated at 2.8 Ata pressure at three different flow settings.

By making these recordings over the depth range customarily used for hyperbaric therapy, we have been able to construct a series of tables that allow us to choose appropriate settings on the ventilator to give the desired inspiratory time and tidal volume for any particular patient. The respiratory rate can then be adjusted by varying the expiratory time control, allowing setting of an appropriate breathing rate and minute volume. In addition tidal and minute ventilation should always be checked using some suitable device such as the Wright's Spirometer. The calibration errors for this device are small and likely to be of little importance in clinical use.

A limitation of the testing circuit used was that "lung" compliance which was roughly 30-40 ml/cm H_20^{-1} could not be altered. Whilst the design of the PneuPac HC ventilator as a flow generator is such that tidal volume should remain contant despite changes in patient compliance, we did not confirm this experimentally. In clinical use our impression has been that delivered tidal voume has not altered with changing compliance.

Two further factors influencing performance of ventilators are the driving gas pressure to the unit and the different densities of the delivered gases. In our chamber the delivered gas may be changed by turning a single valve.

TABLE 1
Calibration chart for clinical use at 6 Ata, showing tidal volumes for varying flow settings and inspiratory times. PneuPac Ventilator settings at 50 m depth (6 ATA)

Inspiratory Control Setting	**6**	**6.5**	**7**	**7.5**	**8**	**8.5**	**9**
Inspiratory Time in seconds	**0.6**	**0.6**	**0.65**	**0.70**	**0.80**	**1.00**	**1.25**
Flow control settings	**Delivered volume in millilitres**						
min	180	170	205	245	250	320	330
x1	195	210	235	265	280	345	385
x2	205	230	245	275	295	375	410
x3	250	280	300	340	375	480	515
x4	305	350	370	430	465	625	650
x5	325	390	400	480	490	670	700
x6	335	405	405	500	520	690	820
x7	345	420	420	510	530	720	845
x8	360	430	430	530	540	740	865
Max	425	-	-	-	-	-	-

Although our driving pressures for air and oxygen are slightly different, neither this nor the different densitites of the two gases altered the delivered tidal volumes at given settings to a degree that would be important clinically. Nevertheless it is important to consider these factors in setting up a mechanical ventilator in a recompression chamber. It is also important to ensure that the gas delivery systems can meet the maximal flow rates that any particular ventilator is capable of. Initial tests of the PneuPac HC in our facility quickly revealed that our oxygen delivery system was not capable of this and required modification.

The inspiratory and expiratory times of pneumatic time-cycled ventilators are well known to change with changes in ambient pressure, shortening as pressure increases. This is a serious limitation in the use of some ventilators. If gas density were the factor influencing this, then the converse would be expected to occur. Since cycling between inspiration and expiration is dependent in most such machines on the filling of a pressure cartridge as described above, Desautels and Blance[2] have proposed recently that this shortening of cycling time is due to the change in the compression factor of that compartment with changes in ambient pressure. Compressional gas losses are directly related to the volume of the container (in this case, the time cartridges) and inversely proportional to ambient pressure. This would result in less volume of gas being needed to flow into the cartridge to cycle it as ambient pressure increases. Our preliminary observations have been consistent with this hypothesis.

We have found the PneuPac HC ventilator to be a robust and consistent performer that can generate adequate tidal volumes within appropriate inspiratory times for the vast majority of adult patients, up to 6 ATA pressure. We would recommend manual ventilation of patients during all changes of chamber pressure, but we have found the PneuPac HC ventilator very suitable for clinical use during the prolonged treatments that critically ill patients may require.

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PAPERS FROM THE JOINT SPUMS AND ROYAL HOBART HOSPITAL MEETING NOVEMBER 1988

OSTEONECROSIS

John Shevland

Introduction

Osteonecrosis is defined as the ischaemic death of the cellular constituents of bone and marrow. The osseous components of the marrow are the cells that reside in the cancellous bone and the cellular components are the erthrocytic and the leukocytic cells in their various stages of development, fat cells and a few reticulum cells.

The bone marrow is the fourth largest organ of the body by weight after bone, muscle and fat, weighing up to 3 kg in adults.[1] By convention, the term osteonecrosis has been divided into two broad categories: the terms aseptic necrosis, ischaemic necrosis and avascular necrosis are equivalent, and refer to epiphyseal lesions, particularly those in a sub-articular location. The term bone infarction is applied to lesions in the metaphysis and diaphysis of the bone.

Aetiology

There are many causes for osteonecrosis. Among them are trauma, steroids, alcoholism, pancreatitis and sickle cell disease. Systemic lupus erythematosus (SLE) is now known to be associated with a very high incidence of hip osteonecrosis, occurring in up to 80% of patients. In many cases multiple aetiologies are involved, for example, alcoholism and steroid ingestion. In day to day radiological practice dysbaric osteonecrosis is uncommon.

Radiology

Table 1 shows the several modalities that are available.

TABLE 1

IMAGING MODALITIES FOR OSTEONECROSIS

Radiographs
Scintigraphy
Computed Tomography (C.T.)
Magnetic Resonance (M.R.)
(Venography)

PLAIN FILMS

Plain film findings are listed in Table 2. These are of limited use in the early diagnosis of osteonecrosis. One must therefore question their routine use in screening protocols, particularly in divers. The first sign one might expect to see is osteoporosis. Unfortunately both dead and living bone have the same density on X-rays. It is only during the healing phase, when hyperaemia induces an osteoporosis in normal

TABLE 2
PLAIN FILM FINDINGS IN OSTEONECROSIS OF THE HIP

Osteoporosis
Sclerosis and cysts
"Crescent sign"
Flattening of femoral head
Acetabular involvement
Advanced degenerative charges

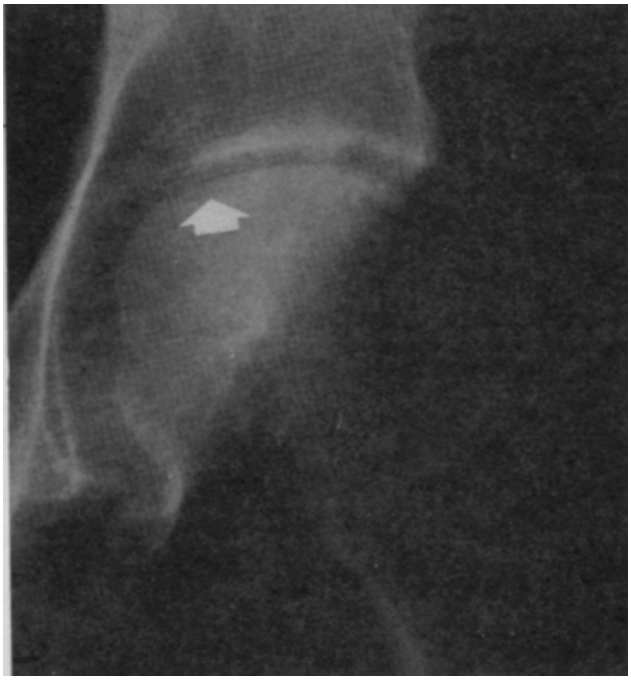

Figure 1. 37 year old male with previous renal transplant. The subtle subchondral lucency in the femoral head (arrow) represents a fracture ("crescent sign").

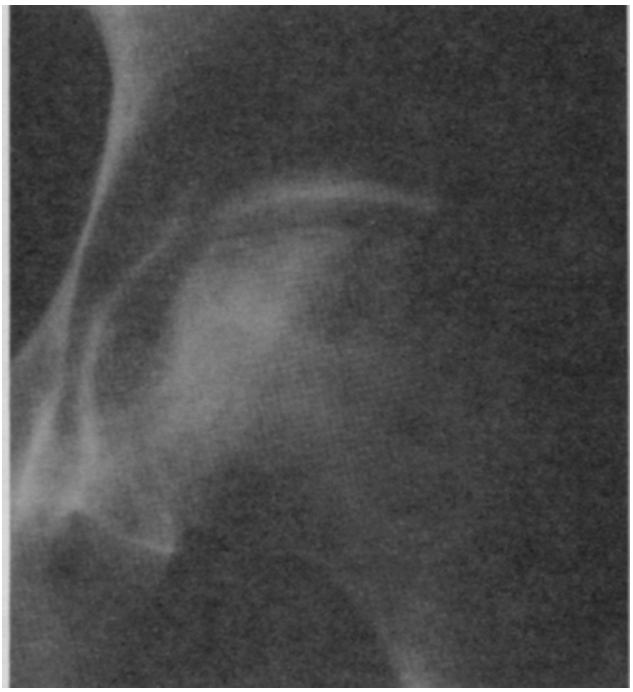

Figure 2. Middle aged male who developed osteonecrosis of left hip while on steroid therapy. Note early flattening of femoral head, and prominent "crescent sign".

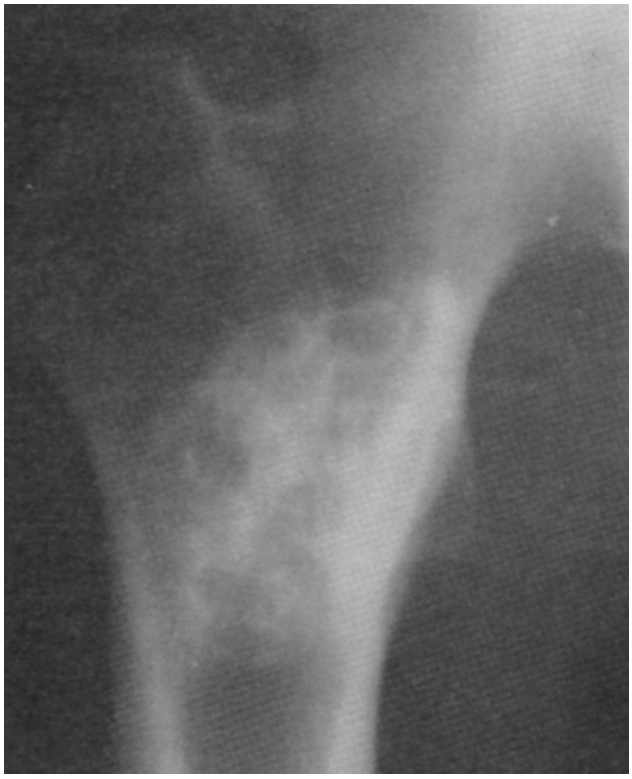

Figure 3. Typical "bubbly" calcified bone infarct in proximal femoral shaft.

bone, that the infarcted bone becomes obvious. The significance of small areas of sclerosis and cysts is controversal as these can be seen in normal people. The crescent sign is a specific sign of osteonecrosis representing a subchondral fracture (Figure 1). Unless treatment is effective at this stage, the late complications of osteonecrosis will become apparent. In the hip joint, these are flattening and fragmentation of the femoral head (Figure 2), eventual acetabular involvement and a destroyed joint.[2]

Bone infarcts have a very characteristic radiological appearance (Figure 3). The typical "bubbly" calcified lesion usually occurs in the diaphysis. These lesions are frequently seen by radiologists in daily practice, and are often asymptomatic.

C.T. SCANNING

C.T. scans are considerably more sensitive than plain films. Where early types of surgery, e.g. rotational osteotomy and bone grafting are contemplated, C.T. scanning is an important part of the work-up, as it demonstrates the architecture of the femoral head and neck well (Figure 4).

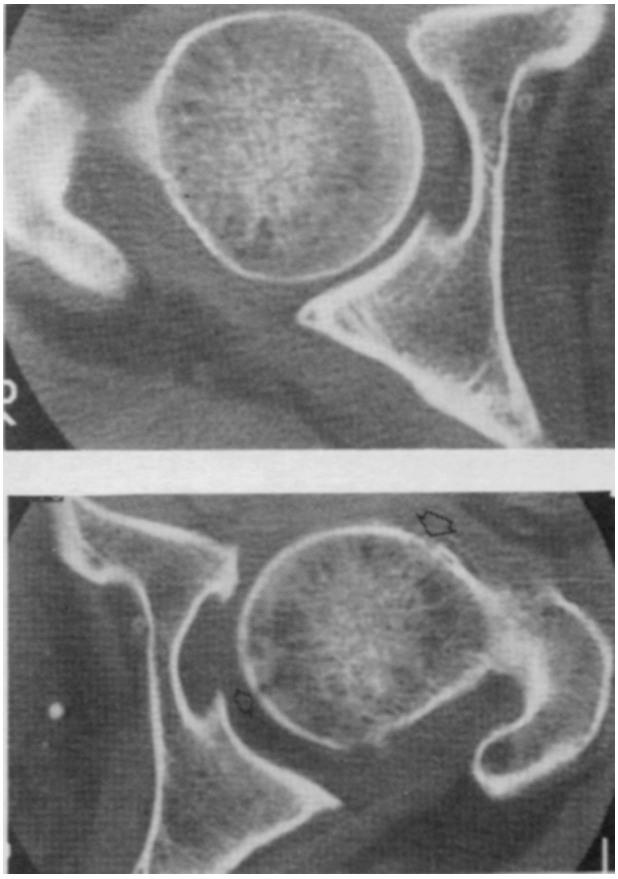

Figure 4. CT scan. 62 year old man with post-traumatic osteonecrosis left hip. Top shows a normal right hip. Bottom shows patchy areas of decreased bone density in the left femoral head. The cortex has been breached in several areas (arrows).

MAGNETIC RESONANCE IMAGING

This technique has many advantages, particularly in its ability to image anatomy in many planes and to detect early bone marrow pathology.[3] Disruptions in the bright signal of normal fat are easy to identify (Figure 5).

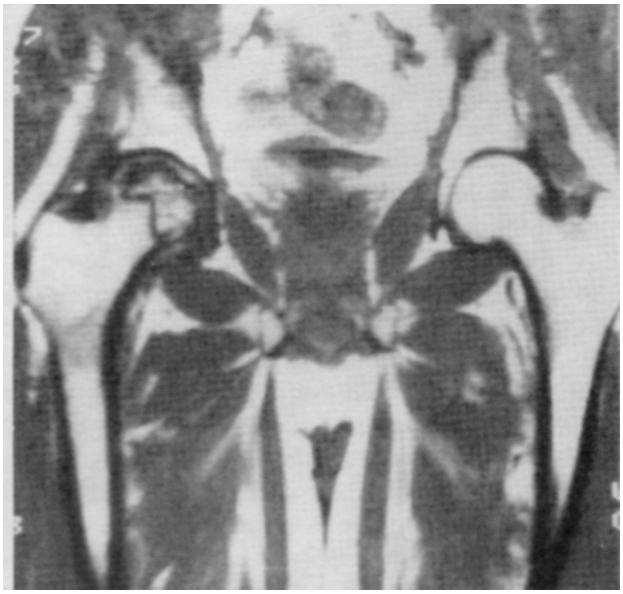

Figure 5. Coronal M.R. scan of pelvis in a man with osteonecrosis of the right hip. Note the absence of a bright signal in the right femoral head.

Diving surveys

Any bone survey in divers should concentrate on the areas affected which are the shoulders, hips, and the areas around the knee joints. Our protocol is listed in Table 3.

MRC classification

In the United Kingdom, the Medical Research Council has classified the radiographic appearance of dysbaric osteonecrosis into two categories, A and B (Table 4). The A category refers to the juxta-articular or epiphyseal lesions and the B. category relates to the lesions in the head and shafts of bones.

This reflects the two forms of osteonecrosis noted earlier, namely bone infarction and ischaemic necrosis. The A lesions are the most important because they occur in the weight bearing areas and are the ones prone to collapse and eventual deformity. However, the significance of A2 and A3 lesions is debatable. B lesions occur away from the weight bearing areas and are commonly asymptomatic.

TABLE 3

DIVER'S LONG BONE SURVEY

A.P. shoulder joints
A.P. hip joints
A.P. and lateral knee joints

TABLE 5

MRC CLASSIFICATION

A JUXTA-ARTICULAR LESIONS
- 1 Dense areas with intact cortex
- 2 Spherical opacities
- 3 Linear opacities
- 4 Structural failures
  - a. Translucent, subcortical band
  - b. Collapse of articular cartilage
  - c. Sequestration of cortex
- 5 Osteoarthritis

B MEDULLARY LESIONS OF THE HEAD, NECK AND SHAFT
- 1 Dense areas
- 2 Irregular calcified areas
- 3 Translucent areas and cysts

Summary

Plain films are of limited use in the early diagnosis of osteonecrosis. Although of high specificity, they are insensitive. Their usefulness in screening programmes are therefore limited.

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SCINTIGRAPHIC DIAGNOSIS OF OSTEONECROSIS

Robert Ware

This will be a discussion about bone scanning and suggestions for screening. A bone scan involves the external imaging of radioactive tracers which are localised in the skeleton. The main tracer we use now is technetium diphosphonate which is readily available. The bone uptake depends on bone blood flow but primarily on bone metabolism. The tracer is taken up in areas of new bone formation and reflects regional osteoblastic activity. After intravenous injection the tracer is distributed to the plasma, to the non-bone extracellular fluid and also to the bone extracellular fluid and a small component of the tracer is deposited in stable lamellar bone. But the largest portion is deposited in active new bone, if the person has significant quantities of that. The remaining tracer, about 30%, is excreted in the urine. The examination involves a fairly low radiation exposure and one of the advantages is that, as one introduces all the radiation at the start of the procedure, taking additional views involves no additional radiation burden. If one is talking about screening young people yearly one has to think about radiation exposure because it may become significant. Because of low radiation dose scintigraphy is very well suited to whole body scanning and screening procedures.

The central feature in the pathogenesis of osteonecrosis is ischaemic and cell death involving the marrow, osteocytes and fat. This can be caused by vascular disruption as in trauma and it can be caused by other things, such as fat cells, which presumably cause vascular occlusion. Growing gas bubbles is the postulated mechanism of interference with the blood supply in the dysbaric disorders. Sickle cells, vasculitis, corticosteroids and some other mechanisms we do not fully understand also cause osteonecrosis.

Not all osteonecrosis is visible on an X-ray. I have an X-ray of a resected femoral head where there were two large necrotic areas but one cannot see them and that is on an X-ray of the specimen not of a patient. So obviously on an X-ray of a patient it is much more difficult to pick up any changes.

Any time one injures bone there is a reparative process starts fairly rapidly. The reparative process involves bone resorption and simultaneous new bone formation. At this stage the scan shows increased activity. We cannot pick cold areas in the femoral head, which represents the necrotic bone, because of the layer of new bone formation surrounding it. Figure 4 in Dr Shevland's paper (page) is the CT scan of a patient who had completely normal plain X-rays. Following a bone scan he had the CT which demonstrated his subcortical fracture.

If the area of bone necrosis and bone resorption is large enough, and particularly if the patient continues to weight bear, mechanical deficiencies appear as manifested by subchondral fractures. This leads very rapidly to articular surface collapse and osteoarthritis which is obviously a disaster in a young person.

That is the theory, how well does it stand up in practice? A study, which is fairly representative of a large number of clinical studies looking at X-rays versus bone scans, was done in 36 patients with lupus who were commencing steroids. The authors looked at bone scans, X-rays and bone marrow pressures, which they took as their gold standard. 27 joints developed avascular necrosis in this group. In the symptomatic group scanning was much more sensitive than X-ray as it was in the asymptomatic group. An interesting experimental model, which may have particular reference to dysbaric disorders, looked at rabbits who had microsphere emboli into their external iliac arteries. X-rays and scans were compared over a period of 12 weeks and these were compared to the histology. Very early on the X-ray is insensitive compared to the scan and the situation does not change very much by 12 weeks. So one is well down the pathogenic path by the time one starts picking up changes on X-ray. All the abnormal areas on the scan were shown at necropsy to be osteonecrosis. However there were 14 areas with osteonecrosis which were not detected on the scans. These were microscopic foci only and therefore probably of minor importance.

Because a large alteration in bone density is needed to demonstrate changes, plain x-rays are insensitive. The features of mechanical deficiency are specific. But one wants to be making a diagnosis before the patient is in a situation where joint replacement may be needed.

Bone scan by contrast is very sensitive. But, because almost anything one does to bone immediately sets up a reparative reaction, the specificity is poor. Recently developed tomographic capabilities (Single Positron Emission Computed Tomography or SPECT) have the advantage that one can section in three planes transaxial, coronal and sagittal. One is able to remove the influence of overlying and underlying activity in a particular plane of reference. Because of this one is able to find the areas of avascular necrosis, or cold areas which do not take up the tracer, when they are smaller in the earlier stage of the disease. So one has increased sensitivity using this technology and as one can find areas of avascularity in the midst of new bone formation, so one increases ones specificity. SPECT is able to be applied straight after the plain bone scan and therefore one is not increasing the radiation dose and one can be directed by the abnormalities on the plain scan.

SPECT studies do not have the resolution of magnetic resonance imaging (MRI) but one gets functional information. In one case where the femoral neck and head had areas of increased activity SPECT showed a large cold

area which was an area of avascular necrosis. From the ordinary scan one could not say what the etiology of the condition was but from the SPECT one could be very certain that it was avascular necrosis.

Coming to aseptic necrosis in commercial divers I must say I could find very little in the literature about the application of bone scans to commercial divers. A paper, in the Lancet, from the Decompression Sickness Central Registry looked at a large number of divers, almost 5,000 over a period of 5 years from 1976 to 1980, with many divers having multiple films. The overall prevalence of osteonecrosis from an X-ray diagnosis was 4.8%. The majority of these were head, neck and shaft lesions which are felt not to be of major significance in terms of producing disability. In fact they are often asymptomatic. Juxta-articular lesions were present in 1.2% and these are the ones that are potentially going to give the divers problems in the long run. Hip avascular necrosis, which is probably the most important site, has a very low prevalence in this group. Shoulders were slightly more commonly affected and these can also produce disabling features if there is articular collapse.

Should one screen divers routinely? I am going to be making suggestions based perhaps on inadequate information. The points against screening are firstly that the prevalence really is very low. Juxta-articular lesions are comparatively rare. It is, as far as I understand, totally unknown whether the presence of head, neck and shaft lesions predicts the future occurrence of juxta-articular lesions. Secondly, it is obviously going to be a fairly costly exercise to screen people. A standard bone scan costs around $300. That would put it in the same ball park as the CT and skeletal survey. The main argument for screening divers is the terrible morbidity of a young person having to have a joint replacement. People who have had decompression sickness are more likely to develop osteonecrosis. The incidence of dysbaric osteonecrosis was 10.7% with definite evidence of decompression sickness and this included the large majority of people with joint damage. Without decompression sickness the prevalence was only 1.7%. It was also apparent that multiple episodes of decompression sickness puts one at greater risk of developing osteonecrosis. They also found the depth of diving was an important predictive factor in developing osteonecrosis. For those diving less than 30 metres there was no osteonecrosis, going up to 15.8% in those diving to more than 200 metres.

I think a reasonable screening plan, if one accepts that it is a worthwhile thing to do and was effective in divers, is to take baseline X-rays of the humeri, the femora and the tibias. I think any screening plan has to include bone scan because of its sensitivity in the early phase of the disease. So a base line bone scan is also indicated. One should repeat the scan if the person develops skeletal pain after an episode of decompression sickness. Perhaps repeat the scan yearly if they are diving to more than 50 metres. As the specificity of bone scanning is not high the correlation is warranted. I think the primary method for following people, if one is going to do it, should be the bone scan. I should emphasise that I am not saying this from any published studies on the subject that I know of, it is just my general feeling about the sensitivity of bone scanning in diagnosing skeletal disorders.

The above has been adapted, by the SPUMS Journal Editorial Staff, from the transcript of a lecture presented at a meeting on Hyperbaric and Diving Medicine, sponsored by SPUMS and the Royal Hobart Hospital, 4th - 6th November, 1988, at the Royal Hobart Hospital, Tasmania.

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MEDICOLEGAL ASPECTS OF AVASCULAR NECROSIS IN DIVERS

Audrey Mills

Introduction

I propose to talk briefly about the diving industry in Tasmania particularly abalone divers. I will use this as a background to the question of what legal remedies are available to divers who suffer avascular necrosis. I will then examine the legal problems involved in diagnosing the condition as that affects medical practitioners.

I would like to acknowledge the assistance of Mr D. Wolfe of The Department of Sea Fisheries for the information on the Abalone Industry in Tasmania.

Professional Divers in Tasmania

Abalone divers would represent the largest group of divers in Tasmania. The abalone industry in Tasmania is one of the biggest abalone industries in the world and is responsible for 22% of the world market. At present, there are 125 abalone licenced divers.

Commercial divers are employed by the Marine Board, CSIRO and Police Department and a few private companies working in salvage and construction areas. The total working in these areas would be approximately 15.

The abalone industry has very few regulations concerning work practices. Whilst diving tables are available and are recommended, it is doubtful that they are strictly

adhered to by divers in the abalone industry, where most are self-employed. I base this purely on reports of people in the industry. There are no governmental regulations incorporating the Australian Standard No. 2299 of 1979, Underwater Air Breathing Operations, so as to make this applicable to the abalone industry.

History

Abalone have been commercially harvested around Tasmania since late 1963. Special licences were required from 1965. During the first years the fishery rapidly expanded until during 1967 more than 250 divers were participating. The Government began to regulate the industry during 1968 and restricted the number of licences in 1969 to those already holding licences in the industry. The number of licences has remained at 125 since 1973.

In 1973 annual medical examinations were introduced for abalone divers and licence holders were required to be full-time abalone divers rather than just full-time fishermen. In 1969 to 1973 production was relatively stable but prices increased by 400%. During this time, diving related illnesses began to cause some concern and it was becoming evident that a number of licence holders were not fully utilising their licences fully with the result that fishing effort and production fell. Recommendations were made to provide a turnover of divers and to allow veteran and/or unfit divers to leave the fishery by allowing those holding licences for more than three years to transfer that is sell that entitlement, to another diver.

In August 1974 the Government agreed to allow divers to transfer their licences. Production then increased until quotas were set in the mid 1980s. The current quota is 24 tonnes. By the end of 1981 the annual catch was almost double the production in the years immediately before 1975; and the value of the fishery had increased by a factor of three in real terms. Fishing returns are submitted annually to the Department of Sea Fisheries by divers and Mr A.J. Harrison has collated figures showing that divers' hours for 1972 to 1973 total 26,773. These hours gradually increased so that in the year 1980/81 the figure was 53,048. The total catch had increased from 2074 tonnes in 1972 to 1973 to 3747 tonnes in 1980 to 1981.

In 1973 when licences were allowed to be transferred, the transfer price was approximately $5,000-$10,000. This steadily increased but has now steadied at $800,000-$850,000. This has caused a lot of speculation with licences. There is a requirement that one diver may hold only one licence at any one time, although it is well known that some divers do control more than one licence. This is done by employing another diver and under a written agreement, transferring the licence to that diver who works for a percentage of the catch. The capital involved in getting into the abalone industry is now so great that there are more financial pressures on divers to meet commitments. In recent years this has been relieved to some extent by the imposition of quotas and the current quota of 24 tonnes is likely to be reduced by 30% next year.

The abalone industry has very few regulations. It is not covered by the Department of Labour and Industry and there is no government body responsible for work practises within the industry. Most divers are self-employed and therefore responsible for their own work practices. The Australian Standard including diving tables is just a guide.

The Department of Labour and Industry does cover divers employed by companies which operate in the construction and salvage areas in inland and estuary waters (includes 3 miles off shore). Australian Standard 2299 of 1979 is applicable to these divers. This sets out tables for diving and recompression and also makes provision for medical examinations and x-rays.

At present, to obtain an abalone licence once you have raised the capital to buy a licence, all you need to obtain the transfer approval from the Sea Fisheries Department is a medical examination by your own general practitioner. There is no compulsory training or experience necessary nor is there any regulation on the work practices. The only regulation which applies is that pressure equipment must be certified and divers must have annual medical examination. There are no requirements for x-rays.

Legal Remedies

When a diver contracts avascular necrosis he has remedies of compensation open to him depending on his work situation. If a diver is an employee, the Workers' Compensation Act will apply. The current Workers' Compensation Act, which is shortly to be amended, specifically recognises avascular necrosis as a "disease". This was an amendment to the Act introduced in 1980. Employees suffering avascular necrosis prior to 1980 would have had difficulty making a claim for workers' compensation because it was not recognised as a Scheduled Disease covered by the Act.

The new Workers' Compensation Act which is to commence operation before the end of the month (November 1988), adopts a wider definition and states (Section 26) that where a worker suffers a disease listed in Schedule 4, it shall be presumed in absence of evidence to the contrary, that the disease arose out of and in the course of his employment and that his employment contributed to a substantial degree to that disease. The disease is described in Schedule 4 as "Compressed air illness including avascular necrosis caused by any work involving exposure to increased or reduced atmospheric pressure from working underground or underwater or from working at high altitudes". By virtue of S26 of the Act an employee suffering from avascular necrosis is

able to claim workers' compensation from his employer and the link between his diving work and the disease will be presumed unless evidence to the contrary is proved.

Another avenue of compensation is to claim damages for negligence or breach of contract against the employer for unsafe work practices, insufficient training and unsafe equipment. Because there is an Australian Standard governing work practices of divers, a successful claim could be made against any employer who did not enforce that standard.

Because avascular necrosis is a long-term problem and may not become evident for many months or even years after it is first contracted, problems can arise with time limits with respect to making such claims. There is a three year time limit in which to bring a claim for negligence and if six years elapse from the date when the condition first became symptomatic no claim for damages can be made at all. Because a condition may take a long time to develop it will be well advanced by the time it is obvious on x-ray and there may be difficulties for some people to come within this time limit.

As most abalone divers are self-employed, the remedies under the Workers' Compensation Act and for damages for negligence will usually be unavailable to them. However most would have income insurance for disability and sickness. The definitions of sickness under these policies vary and some of the more common ones are:

> "Sickness or disease contracted and commencing while this policy is in force".

> "Illness of the insured which declares itself during the period of this insurance and occasions the total disablement of the insured within twelve calendar months after declaring itself".

The nature of the progress of avascular necrosis would mean in some cases that even if diagnosed early, if it did not cause total disablement within twelve months of a person first feeling the symptoms, they would not have a claim, even though at a later stage they may be permanently disabled through having to have a hip replacement operation. In addition because of the difficulty in diagnosis it may not be diagnosed until a late stage which while producing disability may cause problems in working out which insurance company the claim is to be made against if the person is covered at varying times by different companies. Proof of insurance may also be a problem if the claim say arises seven years after the policy was terminated (changed to another company) as sometimes insurance records are not kept beyond this period.

The claim for compensation has to be proved on the balance of probabilities, that is, that it is more probably than not that diving has caused the condition of avascular necrosis. Insurance companies, by their nature will look to seek whether there are any other causes which could be responsible and whether the diagnosis is certain. This has in the past lead to claims being questioned and it is therefore important for full history of a diver to be taken so that other causes can be excluded. If the diagnosis is made perhaps supported only with a suggestion of damage on the bone scan, then other factors such as history of diving and symptoms are very important.

Difficulty in Diagnosis

At present medical examinations are required annually in the abalone industry. X-rays are optional. Given that the condition is well advanced by the time it is obvious on x-rays, there is some argument that bone scans should be performed for divers in risk categories.

At present medical examinations can be performed by any doctor. I would suggest that a panel of doctors experienced in this area would be more qualified to do those examinations and would be better able to judge those divers at risk who should have further diagnostic procedures and also to recognise early symptoms. It may be that there are many divers within the industry who go undiagnosed until the condition is too far gone and major surgery is the only answer.

The problems of not being able to make an early diagnosis may produce major health costs for the community when this problem becomes more apparent as divers grow older.

There are at present 125 divers holding licences and there have been since 1965 another 247 divers involved in the industry. Of the current divers there is one diver who has been in the industry for 20 years, one diver for 19 years, one diver for 18 years, 5 for 17 years, 1 for 15 years, 1 for 13 years. The vast majority of divers are still within the first ten years of diving. The abalone industry in Tasmania is still relatively young and long-term problems in divers' health is likely to become more frequent from now on. No specific records as to divers medical history whilst in the industry are kept. There are no provisions for reporting of major accidents.

This could be analogous to the asbestos industry where major claims are now being made by workers employed with companies over 20 years ago. If avascular necrosis is a major health risk to divers, then because of the resulting cost in health care terms to the community, which in Tasmania would be significant, it is important that more information is known and kept about divers' health. The abalone industry is worth so much in export income to this State, it is surprising that there is so little regulation and relatively little information regarding divers' health.

An interesting argument which I put forward, and it

is no more than that at this stage, would be that the State may be liable to pay compensation to abalone divers suffering avascular necrosis because:-

(a) The Government is aware of the risk to professional divers because of its regulation of the salvage and construction divers and making those divers comply with the Australian standards.

(b) The State Government assumes some responsibility for regulating the industry and providing licences. So it could be argued it is therefore under a duty of care to advise divers of the risks and to insist on proper instruction and training and safe working methods. It could be argued that the Government's failure to do so is negligence, assuming that avascular necrosis is caused by unsafe working methods.

This is similar to the arguments used against the Tobacco Industry by smokers who have contracted cancer.

Problems for Medical Advisers

Lastly, I wish to make some comment on the difficulties for the medical adviser in making the diagnosis of avascular necrosis from the legal point of view.

At the stage of obvious x-ray damage, the option for treatment and prognosis are fairly straight forward.

However, if the diagnosis is uncertain and the evidence can only be seen on, say, bone scans, what are the duties of the medical adviser in advising her or his patient?

If he says "You may have avascular necrosis so do not dive again" then the consequences for the patient may be disastrous. The diver may have to give up a very lucrative profession and suffer financial hardship when in fact his condition may not proceed to serious avascular necrosis.

However, on the other hand, if the medical adviser says "There is a risk that this shadow we can see on the bone scan could be bone necrosis but we can not really say and given there are no other signs you can continue diving" (and there could be a lot of pressure from the patient to continue to dive because of the financial rewards). Then if the patient goes on to develop serious bone necrosis, what is the medical adviser's position?

The test at law is, (though it is under some pressure over the last few years to be changed), "a doctor is not guilty of negligence if he has acted in accordance with a practice accepted as proper by a responsible body of medical men".

So if the medical adviser discloses the uncertainties of the diagnosis and the risks of future damage then he should not be at risk to any claim of negligence.

As medical science advances the problem of diagnosis should become easier for medical advisers.

This paper was presented at the Hyperbaric and Diving Medicine Meeting held at the Royal Hobart Hospital on *November 4th, 5th and 6th 1988. The conference was co-sponsored by SPUMS and the Royal Hobart Hospital.*

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DIVER RETRIEVAL IN TASMANIA

Mike Martyn

Introduction

Tasmania, being an island state, has a large per capita diving population. Diving occurs both commercially and for sport. A hyperbaric facility exists in Hobart and over the years has developed a system for early notification and retrieval of diving medical emergencies. This paper discusses the diving population, the initial contact and notification guide, retrieval options and finally some of the problems.

There are two main groups, commercial divers and sports divers. (See Table 1) Commercial divers can be subdivided into three sub groups. There are some 20 standard work divers. The actual number varies depending on what jobs are around at the time. They have certainly caused some problems with three cases of decompression sickness from the Bowen Bridge building site and more recently the death of a diver from air embolism whilst working on a pipeline at Burnie. There are 125 abalone divers licensed by the Department of Sea Fisheries in Tasmania. One can presume that the majority of these are active although some do sell a part of their licence. The Fisheries also has given out some 300 other commercial diving fish licences, which are specifically for sea urchins and periwinkles. A lot of these are being bought on speculation. There are probably only about 30 to 50 that are actually actively worked. There are also people diving for the aquaculture industry, farming salmon, but most of these are in fact sports divers earning some additional money.

Estimating the number of sports divers is a little more difficult. In 1987 Fisheries sold 3,188 non commercial licences for divers to collect abalone and crayfish. A small percentage will be solely snorkellers. There is also a large number of divers who actually do not possess a licence for a variety of reasons. There are two groups of sport divers. Scuba tank divers of whom most are certified and hookah divers who have generally not undergone certified instruction. Currently there are about 400 people a year certified in scuba diving in Tasmania. It is estimated that there is probably about a 70% to 80% drop out rate. On average there are about 50 tank fills a day in Tasmania. The number of hookah divers is estimated at anywhere between 1,000 and 4,000. The number of hookah units sold or built in Tasmania is around 2,000 to 3,000 in number. So overall, there would be about 2,000 active scuba tank divers and about 1,500 active hookah divers. Active means diving more than the standard three days a year. This equates to nearly 1% of the total Tasmanian population being active divers. A figure of 2.5% of the Australian population having diving certificates is quoted but only a fraction of these would be active divers.

Table 1
TASMANIAN DIVING POPULATION

Commercial Divers	
Work divers	20
Abalone (125)	125
Other Fish (300)	50
Sport Divers (3188)	
Scuba Tank	2,000
Hookah	1,500
Dive Days per Year	
Commercial	10,000
Sports Divers	20,000

Decompression Sickness Rate

These population figures allow for estimates of diving activity and determination of bend rate and mortality. The abalone divers probably dive about 100 days a year accounting for about 10,000 dive days per year. Fisheries estimates for 1983 are 58,000 hours under the water for abalone divers. Assuming an average of five to six hours underwater per day per diver equates well with the estimate of a total of 10,000 days of diving. Other commercial divers would add little to this figure. Assuming five days a year per average sports diver gives some 20,000 dive days total for this group. In Tasmania there has been on average five cases of decompression sickness (DCS) treated each year. There has been only one case of air embolism in the last six years. So about five cases of DCS per 30,000 dives, or five per 3,500 divers per year.

There is obviously an unknown number of DCS cases that are self treated with aspirin, alcohol and other drugs. To date the majority of the DCS has been from the Abalone diver group although over the last two years there has been an increasing number of sports divers visiting the hyperbaric chamber.

Mortality

There has been a fairly constant one death of a diver in the water every two years over the last eight years. That is half per 30,000 days diving or half per 3,500 divers per year. Monaghan's article in the SPUMS Journal[1] estimated the United States diving deaths as one in 95,000 dives or 16 per 100,000 divers. Although small the above Tasmanian figures equate to about one in 60,000 days diving or about 14 per 100,000 divers per year. The mortality in relation to motor vehicles in Australia is 18 deaths per 100,000 motor vehicles per year.

Initial contact and notification

Before 1986 divers often suddenly appeared at the hospital door asking for treatment. This was not an ideal situation. In February 1986 an initial contact and notification guide was worked out with the various retrieval services. This came out as a one page flow chart that was distributed to all medical practitioners, dive operators, hospitals and the abalone divers. Initial contact is through the Tasmanian Ambulance Service via their regional controllers. There are three ambulance regions in Tasmania. They are easily accessible through the '000' emergency toll free telephone number or via radio networks. After recognising the call as a "medical diving emergency" the ambulance controller follows a written protocol. He collects relevant essential information from the caller including location, method of return contact, how many people are involved, their current condition, history of the problem, diving profile, medical history, medical equipment and personnel availability. He gives first aid advice, initiates the retrieval process and contacts the diving medical officer (DMO) who is on 24 hour call via the Royal Hobart Hospital switchboard. The DMO gives further advice on first aid and may contact the dive site. He also advises regarding the appropriate method of retrieval and organises early notification of chamber personnel. This system has streamlined the operation and has generally minimised delays preceding recompression.

Retrieval

Retrieval has improved. The standard retrieval methods of boat, road, helicopter, pressurised plane or portable chamber are available. Boat is usually only used as the initial back to shore transport which occasionally is in

fact direct to Hobart.

The road transport is run by the Tasmanian Ambulance Service in the main regions and by St. John Ambulance volunteers in outlying areas. They are fairly well equipped including the ability to give 100% oxygen using a closed circuit system with carbon dioxide absorption. The main limitation regarding road transport into Hobart is the height of the surrounding hills. To the east Black Charlie's Opening is 336 metres. Coming down the Midlands Highway, Prince's Hill is about 440 metres and from the south, Vince's Saddle is just under 400 metres. Patients who have been brought down by road from Bicheno, where there is an increasing sports diving accident rate, have all said the pain increased in the hills despite being on oxygen.

Helicopter usage is also fairly restricted. There is a single engine Squirrel that is contracted by the Tasmanian Police. It is based in Hobart but may be working anywhere in the state. It is restricted by a single engine to good visibility and to non-night flying. It has certainly been useful in picking up patients from more remote areas in the south where there are no roads or airfields. It has its drawbacks. It is small and cramped and it is difficult to fit a stretcher in and maintain a head down position. However it can provide a speedy retrieval. In one instance an abalone diver with a severe spinal bend was brought from Port Arthur to Hobart fairly quickly.

The fixed wing transport is by small twin engined planes run by Airlines of Tasmania. They are contracted to provide a pressurised and a non-pressurised plane as air ambulance. The pressurised one is a Piper Mojave which is set up to have one stretcher and two seats but can have a second stretcher fitted. Again it is cramped and there are problems with the maintenance of a head down position. The non-pressurised plane is a Chieftain of about the same size. The limitations with fixed wing transport are the condition and location of the various airfields (Table 2). For night use some airfields require local people placing flare pots out to mark the runway. Other airfields such as Swansea and Bruny Island are only suitable at ideal times; i.e. if the pilot can see well, the nettles are not too high, the cows have been herded off and it is not too wet. The distribution of the airfields is quite a limitation. Between Hobart and St. Helens there is an area of popular diving coastline remote from an airfield. In particular Bicheno has been causing some problems with deep sports diving but luckily a coastal road has now been opened up to St. Helens. On the north coast there are Launceston, Wynyard and Smithton but then a large gap on the west coast until Strahan. Abalone diving is popular on the remote west coast and retrieval is difficult. From the north of the state and the Bass Strait Islands retrieval to Melbourne may be more appropriate.

There is no portable chamber in Tasmania and there does not seem to be any great need for one locally as there are only five to six DCS cases a year. Also there is no mating flange on the current Hobart chamber, which means that patients in portable chambers cannot be decanted into the Hobart Chamber. There was an excellent service provided by the National Safety Council of Australia (Victorian Division)(NSCA) from the mainland. This had been used

DIVING MEDICAL EMERGENCIES
IN
TASMANIA

INITIAL CONTACT AND NOTIFICATION GUIDE

IF ACCIDENT OCCURS

GIVE IMMEDIATE FIRST AID AS REQUIRED
(e.g., C.P.R., 100% Oxygen, etc.)

PREVENT FURTHER INJURIES

COLLECT ESSENTIAL INFORMATION

1 Exact location
2 Means of return contact
3 Number of Patients
4 Conscious state
5 Obvious major injury or problem
6 Progressive state of patient(s)
(e.g., stable, getting worse, better)
7 Brief diving history of incident
8 Medical equipment on site (e.g. oxygen)
9 Medical training of people on site

ALSO record details of:

* Full diving history for last 48 hours
* Patient(s) previous medical history
* Time course of events
(problem, treatment and response)

NOTIFY TASMANIAN AMBULANCE SERVICE
(via phone; dial '000' or via radio network)

state: "THIS IS A DIVING MEDICAL EMERGENCY CALL"

FOLLOW INSTRUCTIONS

The Ambulance Service will ask the above essential information, contact the Diving Medical Officer on call at the Royal Hobart Hospital, and organise appropriate transport. The Diving Medical Officer may need to establish direct contact.

Figure 1. The above is a reprint of the leaflet issued to divers on how to handle diving emergencies

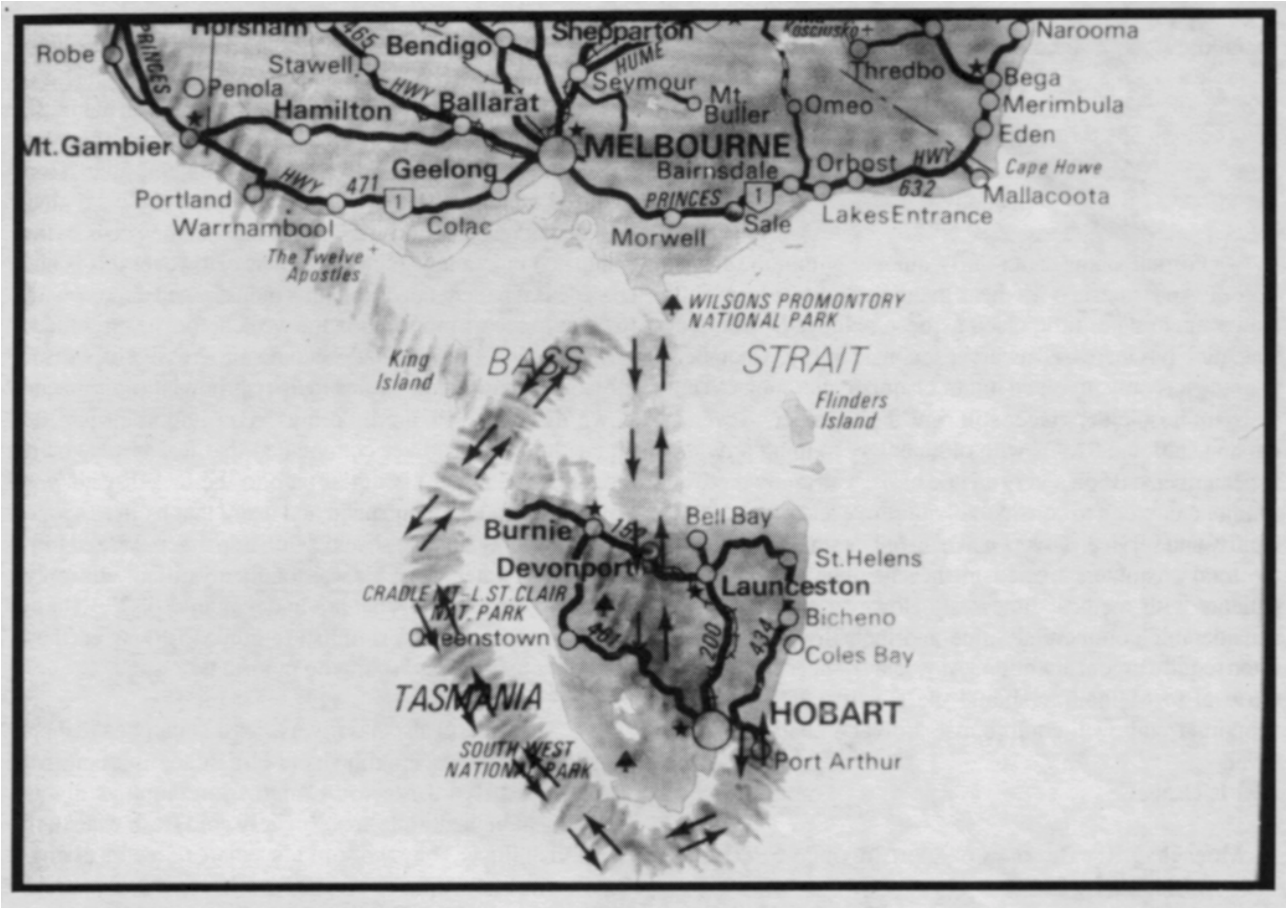

Figure 2. Map of Tasmania showing airfields

on several occasions. They transported the chamber in a King Air and had all the equipment and personnel to maintain and retrieve divers to the mainland. Unfortunately the NSCA has gone into liquidation and will be sorely missed as it provided a very speedy and appropriate service for the severely ill driver.

Table 2

AIRFIELDS AND FLIGHT TIMES IN MINUTES

	Hobart	Melbourne
Hobart		138
St. Helens *	40	114
Launceston	36	104
Wynyard	55	94
Smithton *	65	83
Strahan	43	116
Flinders Island *	74	84
King Island	97	72

* These airports have no fixed landing lights and flares must be used after dark.

Problems

Delays in notification and retrieval are still a problem. Direct notification to the DMO, by-passing the Ambulance Service, can cause delay as then the system is activated in the wrong direction. Considerable delays occur if the patient first presents to a regional hospital, as the diagnosis, paperwork and organisation all take time against a background of feeling safe in hospital. Similarly initial contact with the Diving Emergency Service (DES) in Adelaide has also led to delay. The basic philosophy of DES as a central information body and provider of advice is admirable. However in organised regional areas such as Tasmania there is an inherent delay involved if DES is used as an initial contact in an emergency situation. Knowledge of the local situation and limitations is also important.

The major problems relate to the limitations of the hyperbaric facility. The hyperbaric chamber in Hobart is now 22 years old. It is an old deck decompression unit housed in a garage in the Royal Hobart Hospital grounds. It was run under an ad hoc arrangement until 1985 when a submission was put to the hospital administration. At that stage the following deficient areas were highlighted;

1 Formal status
2 Medical manpower
3 Chamber upgrade
4 Facility manager
5 Chamber staff
6 Education

Formal status came fairly quickly within the Division of Anaesthesia. Medical manpower was organised. However, to date, little else has been achieved. Limited education has increased awareness considerably. Chamber upgrade has only involved minor chamber alterations. The assistant hospital engineer still runs the chamber. Several nursing staff and RMOs with rudimentary training provide the chamber staff on a very ad hoc basis. Currently there is a major campaign to considerably improve and upgrade the facility and service. Divers make up only a small number of the total customers treated in the chamber compared to patients with medical illnesses. However they do have considerable commercial value and their diving illnesses often require urgent attention. Any delay or need for further retrieval to Melbourne should the Hobart facility not be supported may well compromise a diver's chance of survival.

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RECOMPRESSION CHAMBER REQUIREMENTS FOR TASMANIA

Peter McCartney

Tasmania has a population of under half a million people. The distance from Hobart to Launceston is a little bit more than 320 km.

With regard to our hyperbaric facility needs let us look at the two groups of people who provide our patients.

Firstly the divers. We have a lot of divers in Tasmania. Besides the 125 licensed Abalone divers, there are two groups of divers we know little about. There are 300 licensed Sea Urchin divers. We do not know what level of activity these people are operating at, but I certainly get quite a few for diver medicals.

The other group of divers that has crept up on us are the aquaculture divers. I have not been able to ascertain how many of these people there are. But they have been our most generous suppliers of patients over the last 18 months. We have had 5 aquaculture divers with problems that have required treatment in the chamber. Some people say that they do not really count as divers because all they do is swim round at 3 m in a few giant fish bowls. However this is not so. The last patient I had from this industry had done two 24 m dives to secure moorings in the week before he presented, during which time he had become progressively worse. Obviously they do not want their fish bowls to disappear down the river. Fixing moorings is an important part of aquaculture. This diver commented that it was very hard work. He ascribed his initial pain to the fact that he was holding a shackle in one hand and using that hand as a vice while he worked on the shackle with a spanner. I asked him about underwater tools. He was totally mystified. His reply was approximately, "What are underwater tools?". These people are taking a very simplistic attitude to diving yet they are going to 24 m and fixing up moorings.

The CSIRO also has divers. Tasmanian Sea Fisheries has divers. The Department of Main Roads uses contract divers. The Hydro Electric Commission employs divers who dive at high altitude areas, in very cold fresh water and in nil visibility. They work in a specialist area of diving. Disaster awaits people who are used to diving in warmish sea water with good visibility, when they hop into a helicopter, get up to the dive site and are faced with an absolute barrage of new parameters. They may never have been in a nil visibility situation. They have never been in fresh water. They have never done a high altitude dive. And they have never been in such cold water before. Death has been the result for at least one of these people.

And then there are the sports divers, not only the locals but people from the mainland going to Bicheno.

The others who need hyperbaric treatment are patients already in hospital. There are three important areas which will increase patient loads.

The first is burns. We have a very good relationship with our burns unit, but I have made it clear to them that the only patients we can possibly treat at the moment are patients who have problems of wound healing. They are not acute burns at all. It is the case that has been in for weeks which is not healing that is referred. I have to knock back cases of acute burns because we do not have the staff or facilities for proper care in the chamber. The burns unit are very willing to look at our situation and I have adopted a policy with their cases which was suggested to me by Dr Janet Vial. The problem was, how were we going to measure the efficacy of our treatment. The suggestion was to get the referring physician to state the time he would expect the burns to heal without hyperbaric treatment. I believe that the clinical judgement and acumen of those clinicians is some indica-

tion. Then we take them and treat them and see how they get on.

The second group is diabetic ulcers. In my ignorance, I knocked back the first one that was referred to me. Subsequently I have treated quite a number of diabetic ulcers and now have very strong support from both the consultants who look after our diabetics and who are treating diabetic ulcers. In one recent case we quite definitely saved a foot. This patient had had an amputation of one foot previously and had a diabetic ulcer.

I do not have to elaborate on carbon monoxide poisoning. There is no doubt that hyperbaric oxygen treatment is very useful.

Furthermore we are hoping to establish a coronary bypass unit in this hospital in the next six months or so. As neurological changes, probably due to gas embolism, often accompany bypass surgery it is necessary to have a good hyperbaric facility with good transport to and from the facility if one is going to provide the optimal service post-bypass.

Present facilities

When our unit first started up Dr Penny McCartney and I ran it with two nurses. It is quite extraordinary that everything was on a volunteer basis. Our facility is still being run like that. If we could get proper staffing structures officially appointed we would be in a position to offer a much better service. We have a small chamber, acquired when it was no longer useful for a diving company, which has room for one patient lying down and the attendant. A very good indication of what sort of priority hyperbaric medicine has here is that the hyperbaric unit is housed in a garage. We have a splendid theatre complex. When the Quantum team came down to feature our work a very apt remark was made by one their team said, "If you want your leg off go up to the theatres, if you are keen to keep it go down to the garage". To get a stretcher patient into the chamber one has to open the garage doors to get enough room for the stretcher.

Hyperbaric physicians are often fairly sensitive about people criticising their units. Well I am not, I can not afford to be. The funniest comment that I have had from a well meaning colleague, is that it is the lovingly restored FJ Holden of hyperbaric units in Australia. That about sums it up.

I have what I call the "knock back" file. It contains referrals that I can not cope with or which I deem to be inappropriate for hyperbaric medicine. I always make that big distinction. It is very tempting for planners to say if one has knocked back 20 patients in the last 3 months therefore one would have had 20 patients. That is not a true reflection of the situation at all. For the simple reason that if one knocks back 3 cases to one consultant, within a couple of weeks, with the words "I agree this patient needs hyperbaric oxygen therapy, but I am sorry we can not treat as our chamber is too small (or we are too busy)" the consultant is quite justifiably going to stop referring for a while. In other words the number of knock backs is only an indication of the minimum possible patient load. Medical records at the Royal Hobart Hospital keep a data base of admissions according to diagnosis. Mike Martyn ran through that for 1983 using the Undersea and Hyperbaric Medical Society's Category One definite indications for hyperbaric oxygen treatment and found about 98 patients. Then allowing for how many of these we would probably treat and how many treatments they would need we came up with an average of about 5 or 6 treatments a week just on that group. Our figures are purely looking at medical patients. There was a study regarding the of incidence of various diseases according to population and the need for hyperbaric units according to population which came up with figures that were in fact a little bit higher ours. We have had a lot of patients who we would have liked to have treated but were unable to treat with hyperbaric medicine.

As an indication of how little spurts of patient load come in, the last serious case from the intensive care unit (ICU) we were asked to treat was only about a fortnight ago. On that same day there were two other ICU cases that we were asked to treat that were quite out of the question for us to treat because we cannot handle that degree of critical care in our chamber. I believe that when my colleagues view our chamber they will be quite sympathetic towards this attitude and agree that the correct thing is to say no. We believe one has got to have the maxim "first do no harm" firmly fixed in ones mind before we agree to treat. So we are knocking back a lot of cases.

Future needs

I believe we need a hyperbaric facility. I prefer to call it that rather than a chamber, because I believe we require a multiplace chamber, with transfer under pressure (TUP) facilities, which allows the patient to be wheeled in and which has facilities which allow intensive care. I believe we also need a monoplace chamber. There is great polarisation of views about monoplace chambers. In the United States there is a very prestigious unit which is entirely composed of a row of monoplace chambers. The physicians working in this unit say that they are completely satisfied with their equipment and that a row of monoplace chambers is quite adequate. Having seen these units functioning one realises that the people who work in them become very expert at handling difficult situations with the patient remote from the physician and where "hands on" treatment is not possible.

Our basic premise is to have a chamber with a door where we could roll our patient in on a trolley and where we

would be able to fit at least four trolleys. One of the reasons we have to knock back people because of the difficulty of having to lift them in and out of that small chamber.

A four bed multiplace chamber would be quite adequate for us. I mean four people lying down. I am fairly paranoid about patients being able to recline or lie flat in a chamber. I think posture in the chamber is extremely important and a lot of damage can be done if people are not properly postured during their treatment and the attendant can end up having trouble, as happened in one situation in America.

In addition to a multiplace chamber with TUP facilities and a monoplace chamber, we need a transportable chamber with TUP facilities. The entire state of Tasmania in respect of diving is now aware that transfer under pressure facilities are needed. If by some good fortune we acquired a Duocom or Paracel chamber tomorrow, it would be quite useless to us because we cannot lock it on to our chamber. So if we did receive a patient in a portable chamber we would be in the incredible situation of having to return that patient to one atmosphere pressure, that is run the risk of seriously damaging that patient, before being able to place the patient in our chamber.

Australian Standard 2299, up until perhaps a year ago, could be regarded as guidelines and recommendations. The duty of care on the part of the employer is hardening up quite considerably. The Police Department has been very quick to recognize this and have taken very energetic steps to alert our government to this. Other departments also are aware of it. Tasmanian Sea Fisheries, who employ divers, are aware of this and I am quite sure are keen to see a hyperbaric facility becoming available.

A facility which has both monoplace and multiplace facilities has a potential for very large cost saving. The important aspect of having a monoplace chamber is that as the unit gets up and running it is a facility that can easily be added to. One is not looking at doubling the cost to buy another unit because a sizable proportion of the cost of the first unit is the oxygen control unit which can serve more than one chamber. Two chambers, multiplace and monoplace, running in parallel would be a great saving of people having to go into the chamber on a daily basis and the cost effectiveness of that is quite considerable.

If we install a hyperbaric facility with intensive care capabilities, I believe that in the end it will be very cost effective. I can point to some studies from America which have looked at cost effectiveness. There are some absolutely untouched areas such as the compartment syndrome where we should be looking at treating acute cases far more seriously. This is again a backing up argument for our claim for further finance.

We have situations here where patients occupy hospital beds for ages, when they could be out of hospital much quicker after hyperbaric therapy. Even our primitive "guess the time to healing" index shows this quite clearly. Nearly all of our clinicians who deal with these patients have become aware of the benefits of hyperbaric therapy over the last few years.

I emphasise again we will need to have proper staffing. If we have proper hyperbaric facilities we will then just have to have a more generous staffing arrangement so that we can supply a better service.

The above has been adapted, by the SPUMS Journal Editorial Staff, from the transcript of a lecture presented at a meeting on Hyperbaric and Diving Medicine, sponsored by SPUMS and the Royal Hobart Hospital, 4th - 6th November, 1988, at the Royal Hobart Hospital, Tasmania.

Dr Peter McCartney's address is P.O.Box 1317N, Hobart, Tasmania 7001, Australia.

CRITICAL CARE IN THE HYPERBARIC CHAMBER

Ian P. Unsworth

Introduction

Some of the indications for hyperbaric management include slightly over 50% of cases that require intensive care nursing, either before or after hyperbaric oxygen (HBO) treatment. It seems illogical to have a patient in intensive care or critical care very well monitored, well ventilated, well looked after and then, for a period of two hours, take the patient off all those monitors, off the ventilator and pushed through a fairly small orifice into a small chamber for hyperbaric oxygen. What then do we need? The management of critically ill patients in a recompression chamber

Table 1

CRITICALLY ILL PATIENTS SUITABLE FOR HYPERBARIC OXYGEN.

Gas Embolism
Severe Infections
Trauma
Poisonings

must be exactly the same as in an Intensive Care or Critical Care Unit, except that it is under pressure. The chamber should be an extension of the critical care ward, both in equipment and, equally important, in staff. I believe it is absolutely vital to have adequate and appropriate staff for a chamber. No matter how good the equipment, if one has not got people who are accustomed to using it, then it is useless.

Chamber requirements

What are the chamber requirements? Obviously, apart from pressure and it is always nice to have a very high head of pressure available, one needs facilities for resuscitation and continuing resuscitation, for ventilation and the intravenous administration of agents. Medicinal drugs must be available. Some carbon monoxide patients can be very agitated, in fact manic. If one shuts these sorts of patients up in a chamber, they can do severe damage to property and personnel, so one needs restraints and restraints often can be in the form of pharmacologic material. Thirdly, one needs adequate and sufficient monitoring facilities in the chamber.

I like the idea of using a monoplace chamber for elective, non-serious, patients who can look after themselves in the monoplace situation. I do not espouse the concept of putting critically ill patients into a chamber of this nature, even though there are units around the world that do. One is separated from the patient, who may be unconscious and who may be on a ventilator, by 9 mm of plexiglass. There is nothing worse than standing by, unable to interfere, to watch one's patient fit, vomit and inhale. One knows that the only way is up and that indeed could be down for the patient. The monoplace is fine but for fit patients only.

The chamber that one needs for critical care patients is a chamber with plenty of space, certainly enough for the patients to be lying down on trolleys. A family of carbon monoxide poisoned patients was brought to Prince Henry Hospital. There were six patients and we were rather fortunate because we could put all six into the chamber simultaneously. So I suggest to anyone that one have a big chamber and then one can deal with big families.

Patients

The tableabove shows the types of critically ill patients who might benefit from HBO. There is a very wide range. I have just selected a few of these in this table. All divers are familiar with gas embolism from diving. In many people's minds, this is the only cause of gas embolism. It is not and it is by far and away the rarest of all forms of gas embolism. Severe infections, both anaerobic and mixed anaerobic-aerobic infections certainly can produce critically ill patients who, if they are treated with hyperbaric oxgyen, need exactly the same monitoring and management in the chamber that they have in the intensive care ward. Terrible trauma occurs from motor vehicle accidents, from climbing accidents or from falling accidents and crushing injuries. If the patient has something of the nature of an acute compartmental syndrome, then the patient must be monitored and looked after accurately in the chamber. To turn to the poisoning. In this symposium there have been two very eloquent presentations on carbon monoxide poisoning. These indeed are the most common of the poisonings, but one must not forget the other two needing HBO therapy, hydrogen sulphide poisoning and cyanide poisoning. In both cases they certainly need intensive care monitoring and ventilation during their chamber sojourn.

Gas embolism

I have divided gas embolism into two, normobaric and hyperbaric. Gas embolism can occur from investigations in hospitals, operative procedures in hospitals and not an insignificant number occur from cardiopulmonary bypass surgery. If the Royal Hobart Hospital is intent on going into cardiopulmonary bypass surgery, then it is mandatory to provide a well recognised means of managing the gas embolism occurring from the pump. Trauma certainly can produce normobaric gas embolism, and in my experience a number of cases of chest trauma from penetrating instruments have resulted in gas embolism.

The other type of gas embolism with which one is more familiar, is the diving or decompression accident which can occur out in open water, in other chambers, or perhaps (one hopes not) even in ones own chamber. If one causes an injury to a patient or staff member, then of course one immediately turns around and treats the injured person in that chamber.

The non-diving aetiology can be quite immense. In fact there are about 43 ways of getting arterial gas into the circulation. The problem is that hospitals around the country are very reluctant to recognise that gas embolism has occurred in their patients. Doctors, both medical and surgical, know very little about air embolism, and certainly in Sydney the only hospital that ever seems to produce air embolism is Prince Henry! I believe it is because we recognise the cases when they occur whereas the other hospitals in Sydney do not. Gas embolism is certainly an important case load.

Severe infections

What about severe infections? . Myonecrosis, both clostridial and non-clostridial, is life threatening and certainly can be helped markedly with hyperbaric oxygen. In the mixed infections, necrotising fasciitis and the progressive postoperative gangrene, if one has hyperbaric facilities then they should be used. If the facility is not immediately available then it is, as it were, optional. One does not transfer patients long distances to get the benefit of hyperbaric oxygen in necrotising fasciitis, for example. The manage-

ment is radical surgery and antibiotics. This is the type of patient which I imagine one would be using the chamber for here.

Other conditions

Crush injury, compartmental syndrome, and acute spinal trauma are experimental at the present time). However, we have been using hyperbaric oxygen for these for five or six years at Prince Henry Hospital. I am told by spinal colleagues that there is certainly evidence hyperbaric oxygen used adjunctively has long term benefit. So I am continuing to treat their patients as requested but there are certain criteria we apply.

What is the rationale, very briefly, for HBO in spinal injury? In the centre of the cord one can conceive that there is a segment of dead cord, that is irretrievably damaged. On either side of this proposed dead section there is a zone of hypoxia that is reversible. There may be small vessel thrombosis, oedema and vasospasm that renders this area nonfunctional. If one can reoxygenate this area rapidly after injury, one may well push through oedema and vessels that are still patent sufficient oxygen to restore function in these areas. The patient is left with much smaller zones of residual neurological damage. I think that this does have value and we are continuing to use hyperbaric oxygen for these injuries. If in the future this comes to be accepted as an adjunct for acute spinal trauma, then one may well be asked at the Royal Hobart Hospital to manage cases of cervical or thoracic spinal trauma in the chamber. There will have to be plenty of room with some of these patients because they are, or may well be, on neck traction or skull traction, and one will need that little extra space to hang the weights off the end of the bed. Another reason for plenty of room in any proposed chamber.

In crush injuries and ischaemic limbs hyperbaric oxygen is of adjunctive value. Once restoration of blood flow has occurred surgically or by whatever means these cases can and should be treated with hyperbaric oxgyen. These sick patients may have other injuries besides just crushed limbs.

The other speakers have said sufficient on carbon monoxide this morning. Remember that it is the combination with haemoglobin and myoglobin that reduces oxygen carriage and with the cytochrome oxidase A3 in the brain and other tissues that reduces energy production. It is the cytochrome involvement that gives the clinical manifestations of nerve damage, not the haemoglobin involvement.

Management in the chamber

Very briefly about the management of some cases in the chamber, getting down to the nuts and bolts of chamber management with acute critical care patients. One has several systems to take care of. First the respiratory system. One has to make a decision, does the patient need intermittent positive pressure ventilation, or can the patient breathe spontaneously. Along with that of course, the patient should be intubated. One can have intubated patients breathing spontaneously in the chamber, and know that they are getting very high concentrations of oxygen.

Many years ago when I had more time, money and sense I was doing animal experiments in the chamber. We were looking at full scan EEGs in the pigs breathing hyperbaric oxygen, intubated but breathing spontaneously with the CO_2 absorber on, to see what happened when they were at high oxygen pressures. In fact they did exactly the same as humans do, they fitted.

The tightest masks are certainly known to leak. One may only be in fact giving the patient about 97% oxygen because of leaks around the mask. Intubate the patient and one knows there will be no leak and that they are getting 100% oxygen. Carbon dioxide absorption is very important because a rise in arterial carbon dioxide leaves the patient open to the central nervous system problems of oxygen toxicity by causing cerebral vasodilatation and increased cerebral blood flow. It is very important to keep the PCO2 down. The use of oxygen in chambers requires a good scavenging system. If one allows the oxygen percentage in the chamber to rise, one is creating a totally unnecessary fire hazard. So the patient's oxygen must be vented overboard. When being ventilated, some critical care patients may be on positive end expired pressure (PEEP). At a constant pressure, or constant depth, there is no problem, but while during ascent, the PEEP must be reduced. In fact it must be removed because cases of burst lungs from the maintenance of PEEP during ascent have been recorded. PEEP during ascent is dangerous and should never be used.

Cardiac Arrest

Another system that certainly needs care is the cardiovascular system, the monitoring of which we will look at shortly. I have often been asked what happens if the patient has a cardiac arrest under pressure while breathing hyperbaric oxygen. One manages the arrest just as one would in intensive care or critical care ward. If the patient requires it one uses the defibrillator. People have asked whether there is an unacceptable risk of ignition in the chamber. It is not particularly high, certainly there are no problems. The humidity is usually very high in chambers. If the patient has gone into ventricular fibrillation then certainly one must defibrillate.

Oxygen toxicity

Central nervous system (CNS) toxicity manifested

by fits is a possibility, so obviously the patient will have to be closely watched to observe the onset of oxygen toxicity signs such as lip twitching, small muscle twitching and so on. Anaesthetics can be given in chambers using a Boyle's machine with a carbon dioxide absorber.

Equipment

A number of ventilators at the Prince Henry Hospital can be used under pressure. The Bird, Mark VII works well, but there are other ventilators which work satisfactorily under pressure. I have used a Campbell to ventilate a goat at 200 feet with the goat surviving. Some of these ventilators are satisfactory for work at quite high pressures such as the Monaghan and the Manly.

If one does not have a ventilator in the chamber, one can ventilate a patient with by hand. In my experience of the three self inflating bags, the Air Viva, the old Ambu bag and the Laerdahl, only the Laerdahl is any good in a chamber. It is always wise, with a ventilator in the chamber to have a bag as well as a backup system because ventilators have a habit of suddenly malfunctioning at the worst possible time.

One can use a closed circuit absorber system. The absorber is to extract carbon dioxide from the circuit. However this circuit should never be used on patients poisoned with carbon monoxide because carbon monoxide comes out the same way that it goes in, which is through the lungs. If the patient who is poisoned with carbon monoxide is on a recirculating circuit he is excreting carbon monoxide which is then rather carefully put back into the inspired gas.

Monitoring patients is done in exactly the same way in the chamber as in the intensive care ward. One must not use mercury manometers as mercury vapour is toxic and mercury if spilt is almost impossible to remove from the chamber. One can use aneroid manometers. One can insert an intra-arterial cannulae and read the blood pressure in exactly the same way, through a transducer, as in intensive care. There is no problem whatever. Monitor the patient's electrocardiogram by whatever means one likes. This is important because arrhythmias can sometimes occur in hyperbaric oxygen. One can monitor the patient from outside, or have a monitor inside with the attendant. In this case the monitor must be battery powered as no 240 volts mains electricity is permitted in a chamber.

Although I was doing EEGs for a large number of carbon monoxide patients in the early days, I now do not routinely monitor EEGs. The trace is always very good because the patient is in a shielded environment. If there is the appropriate plug through the chamber wall, then EEGs on patients are technically good quality.

The Datascope works very well under pressure for a simple ECG and pulse rate. It can go in with the patient or one can have bigger monitors outside. We have tried other monitors in the chamber. The Datex Cardiocap is not very good under pressure, whereas the Kontron works much better. The Minimon works quite well and one can use that for pressures as well if one so desires. The Life-Pak 6 defibrillator can be used in a chamber if the hot wire ECG component on top can be removed. The hot wire component is a potential for fire under increased oxygen tensions. However it can be removed and the defibrillator component alone taken in. Then it is not a synchronised defibrillator because there is no ECG, but that does not matter if from the monitor one sees that the patient is in asystole. One has the paddles and one charges up the defibrillator and just hits the patient as one would anywhere else. The thought of fire is least in one's mind, one wishes to save a patient's life.

In the chamber there is really no problem or reason why ordinary intravenous infusion sets should not work well. There are closed air spaces, but one can adjust these with changing pressures. The ordinary drip set works perfectly well provided it is looked after. Power driven intravenous lines can be used. Even little syringe pumps can work well down to 12 m (45 feet) or so. The IMED is perfectly acceptable for giving inotropes to a very sick patient.

There are some very, very good bubble filters available and these should be put on all intravenous lines because the introduction of bubbles into the circulation at pressure will cause problems on ascent. Closed systems of fluid administration are much better than open. Plastic packs of blood and other fluids are readily available which do not have to be vented, they just collapse.

Urine measurement is usually done in intensive care and there is no reason why it should not be done in the chamber as well.

Chest drains occasionally crop up with prior pneumothoraces or if the patient has had thoracotomies or pneumonectomies. The management of these is no particular problem. Always make sure however that the chest drains are open to atmosphere during changes in pressure, either ascent or descent.

Staff

Very occasionally one might have problems with staff. Sometimes the medical staff are very reluctant to get into the chamber, but I think that once the chamber is organised within ones hospital, one will find a lot of doctors creeping around and having a look at this thing in a very quiet way. They have heard about it. That is the opportunity to really stimulate a desire in these young people to go into the chamber and be an attendant, with a member of the trained nursing staff, and look after patients. The nursing staff are the vital component of any hyperbaric unit. They are

literally worth their weight in gold. They must be intensive care trained. I have no doubts about that whatsoever. There are no worries about putting any critical care patient in the chamber when the staff are able to manage all the equipment without any problems whatsoever. Trained staff, critical care trained and perhaps scuba divers as well are what makes a unit.

Recommendations

I must reiterate several items. If a hospital accepts that hyperbaric medicine has a use in the hospital, then it is vital, if not mandatory, that satisfactory equipment is provided, that the chamber or chambers are big enough and are adequately equipped and are adequately staffed.

The above is an edited transcript of a lecture on Critical Care in the Hyperbaric Chamber given by Dr. Unsworth at a meeting on Hyperbaric and Diving Medicine, sponsored by SPUMS and the Royal Hobart Hospital, 4th - 6th November, 1988, at the Royal Hobart Hospital, Tasmania.

Dr Ian P. Unsworth's address is the Hyperbaric Unit, The Prince Henry Hospital, Anzac Parade, Little Bay, New South Wales 2036, Australia.

MINUTES OF THE EXECUTIVE COMMITTEE MEETING
Held on 5th November 1988 at Hobart

Present

Drs A Slark (President), D Davies (Secretary), G Barry (Treasure), D Walker (Editor), C Lourey, P McCartney, C Acott (Past President) D Gorman, P Chapman-Smith (Chairman NZ chapter) and J Knight.

Meeting commenced at 1415.

1 **Minutes of the last meeting**

The minutes were read by the Secretary.Acceptance moved Dr J.Knight. Seconded Dr C.Acott. Carried.

2 **Matters arising**

2.1 The President was to write to various registration bodies informing them of the SPUMS Diploma of Diving and Hyperbaric Medicine. This has yet to be done.

2.2 Travel insurance for SPUMS conferences was discussed. It was noted that the GRE Company will not cover retrieval for diving accidents but QBE will. The Committee recommends that insurance be purchased by all attending the conferences but it must remain the responsibility of the individual member.

2.3 The article in "Chest" of December 1987 was further discussed. There has been a big response in the Letters to the Editor and a letter has been accepted from the Hyperbaric Unit at Royal Adelaide Hospital. Members are recommended to seek out the article and follow its progress through the letters column.

2.4 The results of the Incorporation plebiscite were accepted by the Committee. Motion was put by the President, seconded by Dr Knight that "the Committee undertake appropriate measures to proceed with Incorporation of the Society". Passed unanimously. Dr Lourey will contact the Solicitors on our behalf.

3 **Correspondence**

3.1 Journal

3.1.1 Dr J Williamson proposed formation of Editorial sub-committee and made other meritorious suggestions about the Journal. Dr Williamson will be asked to join the enlarged sub-committee with the possibility that it be enlarged further as required.

Moved by the President. Seconded Dr P.McCartney. Carried.

3.1.2 Advertising policy for the Journal has not yet been discussed. This has been postponed sine die.

3.2 Dr J McKee transmitted a request from the Mana Island Resort Rugby Team for sponsorship in the way of Rugby Jumpers. These would cost $50.00 each and they require at least 17. The committee felt that it was not our policy to support a single tourist organisation.

3.3 Mediclinic International Conventions are holding conferences at several venues along the Great Barrier Reef. They request videos on medical aspects of diving and a list of doctors who would give talks about diving medicine. The Secretary wrote requesting further details but has received no reply.

3.4 Dr Yehuda Melamed has requested publicity for the XVth Annual Scientific Meeting of the EUBS which is being held in Eilat from the 17th to 23rd September 1989. The Editorial sub-committee agreed to advertise the meeting in the Journal.

3.5 The Executive Director of UHMS has invited members of SPUMS to the UHMS Annual Scientific Meeting to be held in Honolulu from the 7th to 11th June, 1989. This follows on the week after the SPUMS Conference in Vila. Connecting flights can be arranged either via Sydney or

Nadi. Secretary will write to Dr Greenbaum.

3.6 A copy of the minutes of the last CMAS Meeting was received as well as notification of the next International Congress of Diving Medicine which will be held in Martinique (West Indies) from April 29 to May 7. Topics to be discussed include fitness to dive, cave diving, dangerous marine animals, initial treatment for diving accidents and decompression computers.

4 **Other business**

4.1 SPUMS Diploma of Diving and Hyperbaric Medicine.

A group of doctors active in this field in Australia and New Zealand has been invited by the Committee to apply for this Diploma under a "Grandfather Clause". This group was proposed by the Censors and ratified by the Executive. It was stressed that all future applicants will be required to fulfill all the normal requirements for the Diploma. The total list of holders of the Diploma will be published in the Journal at a later date.

4.2 1989 Annual Scientific Meeting.

Dr Lourey reported that the facilities at Le Lagon, at Port Vila were excellent and the conference will be held from May 27 - June 3rd, 1989. Dr J How of Singapore and Dr M Davis of New Zealand have agreed to be guest speakers. Members are reminded that malaria is endemic and appropriate prophylaxis is strongly recommended.

4.3 1990 Annual Scientific Meeting.

This was discussed. The Secretary will enquire further. Dr Lourey will contact Dr G Atkinson.

4.4 Membership.

It was decided that the Secretary report to each Executive Meeting on the state of the Membership

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4.5 Mana Island Incident Reports.

4.5.1 Dr Acott reported the results of the survey conducted at Mana Island. These reflect badly on the standard of diving at the Conference.

4.5.2 Dr Walker proposes to expand the operations of Project Stickybeak to include diving incidents rather than just diving deaths. A data bank is being designed which can be used to glean statistical information on diving incidents and accidents but which will maintain anonymity of the participants. An appropriate form is being designed at the moment.

4.6 New Zealand Subscriptions.

There has been a loose arrangement between SPUMS (NZ) and SPUMS with regard to the transfer of subscriptions. In future SPUMS (NZ) will transfer the balance in July each year and tender a balance sheet for ratification by the Executive.

4.7 Diving Medical Forms.

At a combined meeting between PADI, FAUI and Dr Gorman, modifications to the AS2299 form for diving medical examinations were agreed. The Secretary will distribute copies to all members of the Executive for their comments.

Meeting closed at 1720.

MINUTES OF THE EXECUTIVE COMMITTEE MEETING
Held on 1st April 1989 in the Sando Room, Royal Adelaide Hospital

Present

Drs A Slark (President), D Davies (Secretary), D Walker (Editor), J Knight, P McCartney, D Gorman and C Lourey.

Apologies

Drs C Acott (Past President), G Barry (Treasurer) and P Chapman-Smith (Chairman NZ Chapter).

Meeting commenced 1335.

1 **Minutes of the previous meeting**

The Minutes of the previous meeting were discussed. Alteration was made to Section 5.8.

Moved Dr J Knight. Seconded Dr P McCartney. Carried.

2 **Business arising**

2.1 Incorporation.

2.1.1 Letter received from the lawyers requesting details of the Society's formation and history and the appointment of a Public Officer. The responsibilities of the Public Officer were discussed and Dr J Knight was appointed to this position, as he had served de facto in this capacity for some years now.

2.1.2 The Statement of Purposes and proposed Rules of the Society were then discussed in detail and amendments were made accordingly. These will be transmitted to the lawyers for incorporation into the rules prior to submission to the Corporate Affairs Commission.

2.1.3 Alterations were made to Section 8, Section 10, Section 12, Section 13(c), Section 22(a) and Section 24(i).

2.1.4 The amended rules and Statement of Purposes will be printed and, if possible, presented for approval at the AGM in May.

2.2 Editorial Committee.

2.2.1 The Deputy Editor has formulated a list of

costs for advertising in the SPUMS Journal. Diving organisations and drug companies will be approached with a view to their buying advertising space.

3 **Correspondence**

3.1 The Secretary has written to all Medical Boards in Australia notifying them of the SPUMS Diploma of Diving and Hyperbaric Medicine (Dip. DHM). Further correspondence will be undertaken by the Secretary to clarify the status of the Diploma.

3.2 Dr L Greenbaum.
3.3 Dr M Davis.
3.4 Dr I Unsworth.
3.5 Dr M Vladica.
3.6 Mediclinic International Conventions.
3.7 Standards Association of Australia.
3.8 Dr Y Melamed.

4 **Membership**

4.1 The Secretary presented the names of 14 people who had applied for membership and 5 who applied for Associate Membership. These were accepted.

A list of 51 members and associates who remain unfinancial at the 31st of March, 1989 was presented to the Meeting. These people will be removed from the mailing list.

The total membership of the Society now stands at 880 of whom 635 reside in Australia and 158 in New Zealand.

5 **Other business**

5.1 Hobart Seminar Expenses.

5.1.1 The President requested information on any correspondence requesting financial support for the Seminar held in Hobart and any previous Minute by the Executive on financial support for such a meeting.

5.1.2 The Secretary stated that he had expressed his support for the meeting and had informed Dr McCartney that previous seminars had received some support.

5.1.3 Dr McCartney stated that he had included his personal expenses as he understood the Treasurer to have requested it.

5.1.4 The budget was discussed and Dr McCartney was requested to provide further clarification to the Treasurer before any payment could be made available.

5.1.5 The Committee agreed that in future, no financial commitment can be met by the Society unless it receives prior approval by the Executive.

5.2 Standards Association of Australia.

5.2.1 The Secretary had received correspondence from the SAA requesting representation on a committee to formulate revision to the DR 88026 standard. It was explained that meetings would be held approximately 6 monthly, usually over 2 days. Dr J Knight had offered his services and was appointed as the SPUMS representative. Any suggestions and alterations from members should be directed to Dr Knight.

5.3 Annual Scientific Meeting.

5.3.1 Port Vila 1989. Organisation of papers is progressing but there is still room for more papers.

5.3.2 Collapse of the NSCA (Vic Div) has resulted in several speakers being unable to attend.

5.3.3 Venue for 1990. Several destinations were discussed as was the need, in light to the current economic climate, to hold a meeting at a cheaper venue.

5.3.4 The Executive expressed its disquiet about the costs of tours organised by Allways Travel. Quotes from other travel agents for the same or similar packages revealed significant financial savings. In future, quotes will be obtained from several travel agents.

5.4 Failure of the NSCA (Victorian Division).

5.4.1 The Committee discussed the potential catastrophic consequences of the collapse of the NSCA. Currently the DES network is receiving about 1000 calls per year for which Royal Adelaide Hospital (in straitened circumstances) is now paying.

5.4.2 The Committee will write to the Premiers and Ministers of Health in those states affected by the NSCA collapse, expressing its disquiet about the potential interruption to diving retrievals and hyperbaric therapy.

5.4.3 The Committee decided that a donation be made to the RAH directed specifically to the telephone bill for maintenance of the DES service. The donation amount was fixed at $500.00.

5.5 Annual Subscription.

5.5.1 The Treasurer has expressed his unease at the current financial situation. In his absence the Committee discussed the subscription and compared it with similar organisations. It was agreed that an increase was required and, it would be recommeded to the AGM that this should be $15.00. The Committee noted that the last increase had been in 1986 and, by making a reasonable adjustment now would probably obviate further change for a significant period.

The Meeting ended at 18.40.

DIVER EMERGENCY SERVICE
Calls received by the Royal Adelaide Hospital
Figures provided on June 1st 1989

The total number of calls to the Diver Emergency Service received by the Intensive Care Unit, Department of Anaesthesia and Intensive Care per annum on aspects of

diving is approximately 1000. They may be categorised thus:

"008" true emergency calls 400 per annum.

Other less urgent calls
MO's with queries on diving medical examinations; persons with queries on types of tables to use, etc., etc., etc..
(These are referred to Dr Des Gorman's office if the 008 line is used. No details are taken by ICU.) 600 per annum.

Total - 1000 per annum.

The breakdown in terms of location of the true "008" calls is as follows:

Queensland	35%
New South Wales	25
Victoria	15
Tasmania	3
South Australia	8
Western Australia	2
Northern Territory	6
Overseas (eg. Fiji)	6

There is a considerable seasonal variation. The winter sees a marked reduction in calls from the southern states.

The following letter has been received by Dr John Knight in reply to one he wrote on behalf of the Secretary while the Secretary was attending the AGM.

Commonwealth of Australia
Department of Community Services and Health
GPO Box 9848
Canberra ACT 2601

4 July 1989

Dr John Knight
South Pacific Underwater Medicine Society
80 Wellington Parade
EAST MELBOURNE VIC 3002

Dear Dr Knight

The Minister for Health, Dr Neal Blewett, has asked me to thank you for your letter of 26 May 1989 concerning funding for the Diver Emergency Service (DES), and to reply on his behalf.

I am pleased to be able to inform you that under the National Community Health Program the Minister has recently approved a grant of $12,500 for the DES operated from the Hyperbaric Medical Unit, Royal Adelaide Hospital.

The grant is to meet half the costs of a Clerical Officer (as originally requested by Dr Gorman, Coordinator of the DES) to enhance the education program of the Divers' Emergency Service.

The grant will be made to the Australian Underwater Federation, and will be administered by that organisation.

Mike Doherty
Director
National Community Programs Section

OBITUARY

Andonis Neofitou, better known as Anthony Newly.

Anthony came to Australia twenty years ago having grown up in, among other places, Malaysia and the U.K. At first he worked for British Airways selling tickets and learning about the travel business.

I first met Anthony in 1973 when the Melbourne Port Division RANR Diving Team was ordered to teach the newly formed Underwater Instructors Association of Victoria (UIAV) about diving medicine, diving safety and how to teach diving. The UIAV had been formed after four trainees had died under instruction in one summer. It was mainly composed of diving club instructors, and was, as far as I know, the first Australian diving instructor organisation to seek help from outside expertise. The RANR Diving Team taught two courses for the UIAV and on both Anthony was an outstanding student. Within two years FAUI, in Victoria, had upgraded its training programmes and the UIAV, having acted as a catalyst, was disbanded.

By 1976 Anthony had left British Airways and set up Allways Travel. About the same time I became Secretary of SPUMS and had to organise the AGM somewhere where there was good diving. I was struggling with this task when Anthony rang and offered to do all the arranging and suggested SPUMS go to Truk. I jumped at the suggestion and thereafter he and I learnt a lot about the travel industry, hotel managements, airline competence (or lack of it), dive shops and their supreme and unrealistic confidence, while we organised the trip to Truk and back.

From 1977 to 1987 every SPUMS Annual Scientific Meeting trip was organised by Anthony and usually to places where the dive shop needed to get in extra boats or

tanks or compressors. All who attended these meetings will remember Anthony, burnt almost black by the sun, solving problems, coercing the dive shop into providing what they had promised, and working hard to see that everyone enjoyed the trip.

Not only was he a superb diving trip organiser, who devoted many hours a year to SPUMS, but he was a very successful travel agent. Allways Travel was accepted by IATA two years earlier than expected so rapidly did its business increase.

In 1986 Anthony, who had many contacts in Fiji from years of sending divers and holiday makers there, decided to leave Allways Travel and set up a diving resort in the Wasawas. He and Linda sold up and emigrated with Stefan, Melanie and Simon. The result was the resort (Dive Expeditions) on Waya Island, where at first they lived in a house in the Chief's compound, a few yards across the stream from the palm fringed resort with its palm leaf bures. In a working partnership with the local villagers they were building a restful, casual, delightful diving resort when the Fijian government was removed by a military coup and tourism became a fading memory.

It is ironic that in November 1989 when tourism has become a reality again that Anthony should have been injured by a large stingray, and tragic that the injury opened a femoral artery. He died, from blood loss, a few hours after his injury.

Anthony had many sides to his character, a friendly interesting companion, an excellent diver who used almost no air, an exciting musician, and a loving husband and father.

I am privileged to have been Anthony's friend for the past 13 years and have learnt much from knowing him. I am sure that the SPUMS members who knew Anthony will join me in wishing Linda and the children a speedy recovery from the devastation of his death, and the ability to always have their happy memories of him.

I understand that Linda will continue to run the resort while Trevor and John, the Fijian divers who worked with Anthony, will run the diving. A practical way of helping Linda would be to take your next diving holiday at Waya.

John Knight

We reproduce some of a recent newsletter from the Confederation Mondiale Des Activities Subaquatiques (CMAS) know in English as the World Underwater Federation. It is good to see that Project Stickybeak isspreading its message around the world even if the emphasis is changed in the process. It is sad to see the report of yet another spearfishing competition death.

EXTRACTS FROM THE AUGUST 1989 NEWS LETTER OF THE CONFEDERATION MONDIALE DES ACTIVITES SUBAQUATIQUES (CMAS)
Commission Medicale et de Prevention

(World Underwater Federation Medical and Prevention Commission)

Editorial

Dear Friends,

We are pleased to present you the first MPC News Letter prepared by our friend, Michel Leloup.

This semestrial News Letter, bilingual French/English, will be a link and a mean of exchange between us and our colleagues of the world.

We intend to publish information about the various aspects of diving medicine, its progress, its discoveries, we shall also announce the great medical meetings of the underwater world and some interesting medical books.

We hope through this publication to be able to create dynamics within our medical commission and we invite you to write us, to submit texts and to send us your suggestions in order that this news letter will be an efficacious information tool.

Dr Marcel Bibas

C.M.A.S.

The true United Nations of diving, the World Underwater Federation is composed of 75 countries.

Affiliated to UNESCO, officially recognised by the IOC, the CMAS intends to develop, teach and co-ordinate the underwater activities whether of a sporting, technical, scientific and medical nature.

Provisional Report on Australian Diving-Related Fatalities 1986

Dr Douglas Walker. Provisional report on Australian diving-related fatalities 1986. SPUMS J 1988; 18: 42-53

The South Pacific Underwater Medicine Society reports fourteen (14) diving-related fatalities that were identified as having occurred in Australian waters during 1986. Two (2) were breath-hold divers, nine (9) were using scuba, and three (3) were using surface supply (hookah) systems.

While there is no clear common reason for the scuba diver incidents the one clear fact is the frequency with which the critical action of the incident occurred at the surface. Many of the victims had only slight diving experience

although some were both trained and reportedly experienced. The hookah incidents indicate some of the dangers associated with the use of this equipment.

After summaries of cases they are classified with seventeen different items, though the information is not always available: age of victim, dive skill level of victim and buddy, dive group, dive base, dive purpose, water depth, incidence depth, weight of belt, on or not contents gauge, buoyancy vest, remaining air, equipment check, equipment owner, wet suit and significant factors.

Examination of cases reveals many matters which are of importance in the context of diving safety but major attention is warranted to two matters in particular, the fact that a medical factor was so frequently present and the number of cases where a buoyancy vest failed to save its wearer.

Certainly a medical examination would not have predicted all disasters. The situation may be that the number of people diving is now so great that a statistical expectation arises that illness-related deaths will occur in sufficient numbers to invite comment. There must be many unfit persons undertaking all types of activities and only a small number will become fatally ill. It would be helpful in this context if an increased attention were given to investigating the medical history of victims, but there is a high probability that a person undertaking diving or other potentially strenuous activity will be unlikely to tell others of any symptoms he suspects might lead them to advise him to refrain.

Of greater importance is the finding that an unconscious person may drown despite wearing an inflated buoyancy vest, a fact which is disquieting and deserves urgent attention. There is also a fact noted previously, that an empty tank makes scuba-feed vests virtually useless in time of need. Obvious, but nonetheless having real significance and likely to be overlooked by wearers until an emergency situation occurs.

One B.H. case illustrates the effect of using unfamiliar equipment, in this case the victim would have survived if he had thrown away his snorkel and thought of himself again as a swimmer rather than someone breathing through a hole. The other breath-hold fatality underlines the sad fact that one is alone in a crowd, that supervision of an unregulated group is not possible. It is for such reason that the instructor and pupil ratio has to be kept low, particularly in the open water phase of teaching scuba.

Fatalities among hookah users usually result from failure of the compressor portion of the apparatus or from a hose problem of some sort. In these cases there was hose entanglement and hose separation as well as a cut hose and compressor engine failure. A sad collection of reminders of the critical factors deserving the keenest attention by hookah users.

Readers are invited to consider what further lessons they can discover from a careful consideration of these case histories so that such events never confront them or their buddies.

At the end of the newsletter the following appeared. Emphasis has been added by the Journal

We have just been informed of the death of Didier Delonca, the son of our Colleague Georges Delonca, who deceased during a spearfishing dive.

We address to Georges and his family our heartfelt sympathy.

ARTICLES OF INTEREST REPRINTED FROM OTHER JOURNALS

WHAT TO DO WHEN YOUR AUTOMATIC INFLATOR STICKS OPEN

Dennis Graver

If the valve of a low pressure inflator on a buoyancy compensator or a dry suit sticks open, a dangerous, out-of-control ascent can result with an accompanying great risk of injury. What can be done to minimise the problem with stuck inflator valves?

The best thing to do, of course, is to prevent the problem. Proper maintenance and frequent inspection of the valve can go a long way toward preventing the valve from sticking. The second way to minimise the potential problem is to have a high level of awareness while diving: the sooner you recognise a problem, the sooner you can deal with it.

If, despite attempts to prevent an inflator valve from sticking, the valve does stick during a dive, here is the sequence of actions that should be taken.

1 Disconnect the low pressure hose.

Do not fiddle with the inflator button first. Valuable time will be lost while buoyancy is being increased. Simply unhook the hose, then inspect the button. Even if the inflator button can be returned to its normal position, it is probably not wise to reconnect the inflator hose until the entire mechanism can be inspected out of the water after the dive. Continue the dive using oral inflation techniques. (Do you know how to orally inflate your BC under water? Low pressure inflators are used so commonly now that some divers have not learned oral inflation procedures.)

2 Maintain control of buoyancy.

If you become positively buoyant from air admitted to your suit or BC by a stuck low pressure inflator, swim down immediately as hard as you can while disconnecting the hose. Use pressure to reduce the volume of any extra air. The shallower you are, the more important this is because the rate of change of volume becomes greater the closer you are to the surface. Swim down hard, grab hold of something on the bottom, turn to an upright position and vent any excess air. Avoid rising passively. Fight to remain at depth.

3 If control is lost and you rise toward the surface in an ungoverned manner, manoeuvre yourself into a face-up position and flare your arms, legs and fins to create the maximum cross-sectional body area.

This will slow your ascent rate dramatically. *After* achieving this position, attempt to vent excess air. Maintain a normal breathing pattern and ride out the ascent. Always disconnect the low pressure hose, even if you have to do it during an out-of-control ascent. Implement this as soon as you realise that you will be unable to swim down hard enough to overcome excess buoyancy. If you simply try to swim down all the way to the surface, a point will be reached where you will lose directional control due to the buoyancy problems, you will then be unable to achieve the flare position, and your overall ascent rate will be much greater than if you had flared earlier in the ascent.

To summarise: the keys then to minimising low pressure inflator problems are: prevention; quick recognition and reaction; the ability to overcome excess buoyancy; and the ability to flare during an uncontrolled ascent.

Dennis Graver, is the Director of Education for NAUI (National Association of Underwater Instructors) U.S.A.

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The address of UNDERCURRENT is P.O. Box 1658, Sausalito, California 94965, USA.

ADVERSE REACTIONS TO SCOP

Scop is a recently released transdermal delivery system which contains hyoscine designed to prevent symptoms of motion sickness such as nausea, dizziness and vomiting. It is in the form of a patch which is applied to the skin behind the ear to result in a controlled release system for the drug.

Each Scop patch comprises an adhesive layer containing a priming dose of the drug to saturate local binding sites, and a main reservoir of drug enclosed in a semipermeable membrane. This allows for a constant release of the drug.

The active ingredient, hyoscine, also known as scopolamine, is a naturally occurring belladonna alkaloid, closely related to atropine. It has central and peripheral anticholinergic activity and produces depression of the cerebral cortex. It also acts as a hypnotic.

Adverse effects[1] of hyoscine are predominantly those anticipated from the anticholinergic nature of the drug. The most frequently observed adverse effect of the transdermally administered hyoscine is dry mouth; drowsiness is also commonly reported. Transient impairment of ocular accommodation has been noted and, less frequently, other adverse CNS effects such as dizziness, confusion, disorientation, hallucinations and memory disturbances.

There have been 10 reports of adverse reactions to Scop received by the Adverse Drug Reactions Advisory Committee in the period from March 26, 1987 to August 31, 1988. Of these reports, seven have included disorders of vision. In all cases, Scop was the sole suspected drug. Reactions included blurred vision (six out of seven reports), mydriasis, abnormal accommodation, scotoma and retinal disorder. There have also been two literature reports[2,3] of the more serious ocular disorder, acute angle-closure-glaucoma associated with the use of Scop.

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Reprinted from the Australian Adverse Drugs Reactions Bulletin, March 1989.

GETTING BENT BY COMPUTER
A diver's story

Bill Lovin

To me, "bottom time is money". I produce underwater films and the ability to spend more time on the bottom translates into more film shots.

Many of my dives fall into the same profiles as many sport divers, that is, Caribbean wall dives with deep penetrations, and a significant amount of time spent in shallow water. To maximise my bottom time, I settled on the Edge and tested it during 60 dives in the Caribbean and the Red Sea against a calibrated precision depth gauge and the US Navy Dive Tables. In all of those sixty dives my computer gave extremely accurate depth readings while allowing incredibly more bottom time. I felt comfortable with the computer and trusted it completely.

Soon I began shooting for a wreck diving movie. During one of my dives I suffered decompression sickness while well within the limits prescribed by my computer.

It is impossible to describe the physical and emotional trauma to someone who has never stared for long hours at the ceiling of a recompression chamber. My bends began as a curious sensation in my elbow which I can only describe as the same feeling I get when I swim over to another diver and feel thousands of exhaled bubbles over my body. It migrated quickly up my arm and into my shoulder, where it just plain *hurt.* The feeling ranged from, at its worst, six needles stuck in my arm to, at its best, feeling like I severely overdid it playing tennis. Additionally, I felt a strong tingling sensation in my fingers and an almost complete loss of strength. A can of soda fell out of my hand while I was watching it.

At first, I completely denied the possibility that I might be bent. I lay down with my feet up and did not move, but also did not tell anyone I felt bad. The boat trip home was three hours, and I just kept telling myself that I had wrenched my arm and the pain would go away. Looking back, it seems ridiculous, but this was serious denial.

Once on shore, I felt better, but the act of getting up and unloading the gear rekindled the pain. By this time I had begun to accept the fact that I might have "minor" DCS. Only after I was in the car heading home did I tell my diving companion that I was sick. Always prepared, she had carried oxygen on the dive and I agreed without much hesitation that I ought to breathe oxygen on the way home (about three hours).

During the trip home, the pain would get better and then worse. Still, I did not even consider the possibility that I might require treatment.

Six hours after surfacing and the onset of symptoms, I was home in bed. I made a most uncomfortable decision to call for medical help, something that I viewed as an ultimate failure.

I live only 12 miles from Duke University, site of the F.G. Hall Hyperbaric Laboratory and the Divers Alert Network (DAN). I was completely familiar with DAN and had friends who worked there, but it was still amazing to me that when I dialed that telephone number (the emergency number, which made it even harder), within minutes I was talking with a physician expert in diving medicine.

In less than an hour I was at the Duke University Emergency Room, met by three physicians all arranged with incredible smoothness by DAN. Thirty minutes later I was in one of the huge chambers at F.G. Hall Lab with a nurse, while two doctors, a chamber engineer and another hyperbaric nurse ran things outside. My treatment was accomplished in three and one-half hours. Still, it gave me a good deal of time to think.

I found myself totally embarrassed by the situation. It was a Saturday night and I could not help thinking about what each of these people were doing when they were rudely summoned to work in the middle of the night. When I walked out of that chamber at 2:00 a.m., I still felt some pain. It would take several more days to go away, but I knew I done the right thing in seeking treatment.

My treatment cost was about $1,500, and I got off *light*. That 14 minutes of decompression I did not take cost me roughly $100 per minute. It also cost me a good deal of anxiety and depression, and a lot of time. For the months following I was required to have many neurological examinations.

Three months later I still felt the effects of being bent. Deep dives and dives in very cold water rekindle the shoulder pain (not as bad, but *very* noticeable). Three years later my shoulder only hurts when there is a change in the weather or a sudden change in temperature, much like arthritis.

When I was in the chamber I thought a great deal about the dives that led to my being there. Although my computer had suggested that no decompression was required after my second dive, the US Navy dive tables suggested that 14 minutes at ten feet might have been a good idea.

Perhaps our desire for those extra minutes on the bottom and fewer minutes on the line has become too great. Today when I dive I spend as much time hanging on the line as on the bottom. I still use my Edge, but I do not blindly trust it. I keep close tabs on it and carry a backup Skinny Dipper, a backup depth gauge, a backup timer, and a US Navy Dive table permanently cemented to each of my camera housings. I always make sure that the pixel display on the decompres-

sion computer has moved well away from the "mythical" danger line before I surface. I will not again become complacent and overly trusting of any computer.

But I do believe that the use of decompression computers is valid. There is every reason to believe that figuring our decompression time using so-called multi-level tables should work. But I suspect that we are in an "experimental" situation where we are the guinea pigs. Nonetheless, I believe that a person's health and welfare is that person's own responsibility. Unless we are deliberately deceived then I feel we essentially pay our money and take our chances by making our own choices based on our own knowledge and inclinations.

You must never feel embarrassed or reluctant to seek help following any diving accident. This is no time for "macho". I was told that breathing oxygen probably saved me from greater problems, there was oxygen on board the boat, but I was too embarrassed to ask for it. I could have essentially begun treatment three hours earlier and, perhaps, saved myself from even more long-term effects.

Join the Divers Alert Network. DAN is currently doing exhaustive research on decompression for sport divers, and provides a compassionate and knowledgeable voice on the other end of the telephone when that emergency happens.

Undercurrent Editor's note

Bill Lovin ,who is an underwater cinematographer from Chapel Hill, North Carolina, got bent while on assignment. The incident occurred nearly three years ago, but the story continues. We include this as a supplement to our series on computers and computer diving.

Lovin continues with his cinematography off the North Carolina Coast and in the Great Lakes, but limits his depth to a maximum of 150 feet and never misses a stop at 20 feet or ten feet. He speaks of his bends incidents to groups around the country, stressing his experience and the need for rapid treatment. He also has another insight into his Edge.

"Interestingly, the depth transducer on my computer failed on the first dive I made after being bent. It produced increasingly erroneous depth indications until it was as far as fifty feet off. Is it possible that I was bent because the computer was using incorrect depths supplied by a faulty transducer? I would assume that depth would not have to be far off to push a diver into the 'danger zone'. It seems to me that the weak link in the computer can be the depth transducer (which converts depth information from an analog form to a form that can be processed by the computer)."

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BEYOND EMBARRASSMENT
The musings of an occasional diver

A sailing friend of mine once said that 90 per cent of what sailors do is to look good for other sailors.

Probably the same comment could be made about diving. Embarrassment is commonly defined as "experiencing a state of self-conscious distress", but I prefer the vernacular syllables right there within the word itself: "bar(e) ass".

As an occasional diver, "bare ass" is something I too often experience. I dive six to twelve times a year usually on one or two tropical vacations. And like anything else, after a six to twelve month layoff, I am a "rusty diver". I am not unsafe. I have 13 years' experience and am very conservative. But some things that happen can often make me the butt of diver jokes.

Preparation and gear

I like to use dive gear for as long as it is serviceable, which can be forever with proper maintenance. (My equipment works well and I do not dive enough to warrant an expensive changeover. This carries into other areas as well. It caused my preppy wife no end of discomfort when I was still wearing bell-bottoms in the 80s.) All of my dive gear is at least 10 years old, and some of it closer to 15. I did splurge on a new knife and mask for my last trip. The knife was inconsequential and the 13-year-old mask worked better than the new one. I am about to inherit a "second generation" wet suit top from the same buddy who gave me his original (I cut the arms off that one because there were unpatchable elbow holes in the dried-out neoprene). I still use a horse collar BC. My gauge console is jury-rigged and I do not have an octopus. I pack my gear in an ugly, 20-year-old suitcase. (No one will steal that at the Miami airport.) Once on site, I transfer it to a net bag. However, despite the utility, I get a lot of comments from people. "Nobody uses a horse collar anymore." "Does that top still keep you warm?" "What's the suitcase for?"

Though I check the gear before I leave, occasionally age does show. The last time out, the zipper on my wet top stuck. I forgot how to climb through all those little straps and into my horse collar. And it took some thought to remember over which shoulder the regulator should fall (it helped to watch someone else hook up his regulator first), then it took a couple of false starts before I got mine attached to the tank correctly. Everyone is helpful, but heads shake. I even learned of a magic substance to unstick the zipper, spit.

The diving frame of mind

I accept that I am rusty. To counter this, I review the basics before a trip and even listen to DAN's diving medicine review tapes. I ask the guides for a little extra watchfulness.

Unfortunately, this often garners as unhelpful response: "Do you have a log?" (No.) "Listen to the lecture for the people being certified (do not hold your breath, here is how to clear your mask)." "Can you handle the first dive?" Granted, there are safety concerns being expressed. Yet, I feel like it is the third grade again.

The pace and the race

My life is swift with a serious job, a family (two children) and numerous other responsibilities. When I vacation, I want to *slow down*, to leave the rush of life behind. Patrick Finley, a San Francisco Bay area writer, states that the word "vacation ... comes from the Latin "to vacate, to empty" ... nothing to account for, support accomplish ... or justify".

Yet many of us turn vacations into quasi-duties, filled with schedules and intentions. Everything a diver does can be subsumed under the phrase, "do it slowly". Yet, I find the pace frequently set by other divers more like my work pace.

Everyone rushes to the boat. They are anxious to leave the dock. The pace does slow somewhat during the ride, but once there, it is like Marines landing at Iwo Jima, "everyone in, move it, move it". What is the rush? And once in, it is full bore around the site. The philosophy seems to be "cover the most territory". And the trip home is often a race across the waves. Pile out, unload the boat, pay the bill. Go. Go. Go.

I like to follow photographers and let the reef come to me. On a trip to the Grenadines, it was myself, my dive buddy and our own sailboat (the two of us!). No herd, slow down, take your time. After a few dives, I heard, "Are you usually the last off the dive boats?" And even when I rushed, it never was quick enough. I started sounding like my four-year-old son "I am trying, I am trying".

Going down, coming up (where is the anchor chain?) and in between

A big problem is remembering how much weight I need, and it never seems to be the right amount ("You look about 9 pounds"). I let the air out of my BC and watch everyone else descend while I keep bobbing, the water barely over my head, getting sick from the surge. I fin down like a wounded fish. On Bequia, the guide was concerned and guided me down. Thanks. The price: "How long have you been diving?" "13 years." More head-shaking.

I suppose part of the thrill is large fish, especially sharks. I tend to stay clear, dorsal fins make me anxious. On one dive my rapid retreat became a positive signal for my buddy. When he saw me race out of a cave, he knew it was something he wanted to see (a 12-foot nurse shark with a head that appeared two feet across).

Surfacing, I have a terrible fear of the open water swim to the boat. I am an average swimmer and fatigue somewhat easily. And on occasion, a current has made this a reality. On one dive, after slugging against a current and being asked twice, from the boat, whether I was okay, I decided to pack it in. The guide swam the distance in five seconds and hauled me on board. Everyone had a chance for a few wise cracks. Fortunately for my ego, someone else was bitten on the ear by a turtle.

On another occasion, while drifting toward Africa, I had five minutes of near panic. The current was swift, the boat was lost in the swells and the guide was searching in the wrong area. I clamped onto my buddy and, at his encouragement, listened to stories about how we could survive for two days! We were picked up 20 minutes later and, as expected, my "near panic" received a full (and reasonably gentle) airing.

Years ago, after a dive in Hawaii, I had a pain in my toe. A couple of days later, still worried, I confessed the pain to my buddy, and wondered out loud whether I had been bent. When the pain returned after the next dive, I learned that my fin was too tight. We both laughed, he more than I.

Moral

On my next vacation I will probably repeat some of the above lapses and gaffes or create some new ones. I have learned that I can avoid some if I figure out my gear before my first dive. And before each individual dive, I need to anticipate the time we are going to hit the water so I can start getting my stuff together early to respect my own pace.

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CONFERENCES AND COURSES

COURSES IN DIVING AND HYPERBARIC MEDICINE

ROYAL ADELAIDE HOSPITAL HYPERBARIC MEDICINE UNIT FEBRUARY 1990

Basic Course in Diving Medicine

Content: Concentrates on the assessment of fitness of candidates for diving. This is a Health and Safety Executive (U.K.) approved course.

Venue: Royal Adelaide Hospital, Adelaide.
19 - 23 February 1990.

Cost: $AUS 500.00.

Advanced Course in Diving and Hyperbaric Medicine

Content: Discusses the diving-related, and other emergency indications for hyperbaric therapy.

Venue: Royal Adelaide Hospital, Adelaide.
26 February - 2 March 1990.

Cost: $AUS 500.00
($800.00 for both courses)

For further information, or to enrol, contact:
Dr. D.F. Gorman, Director, HMU
Royal Adelaide Hospital,
North Terrace,
ADELAIDE,
SOUTH AUSTRALIA 5000.
Telephone: (ISD 61) 08 224 5116.

FIT TO DIVE 1990

28 and 29th April, 1990
Hotel Nelson, Norwich,England

A further weekend revision course for Health and Safety Executive (HSE) Approved doctors in the series 'Fit to Dive', is to be held next April.

The theme will be 'Diving Neurology', a topic which is increasingly in the spotlight.

How valid, for instance, is the view that the absence of clinical signs is all that is needed neurologically to re-issue a fitness certificate?

Dr. Ian Calder, HSE, whose autopsy material provides the basic present knowledge and concerns, has agreed to speak as has Dr. Zoltan Torok, Admiralty Physiological Research Laboratory, who, in addition to being an occupational physician, is well experienced in neurophysiology. Besides other consultants speaking on the investigation of neurology dysfunction, Dr. Maurice Cross has also agreed to speak and, at his suggestion, we will include the orthopaedic aspects of fitness to dive since, in practice, this usually means lumbar problems in relation to potential neurological deficits.

Registration fees

Before 31st January 1990	£105
After 31st January 1990	£135

For further details contact
Biomedical Seminars,
7 Lyncroft Gardens,
Ewell, Epsom,
Surrey KT17 1UR,
England
Telephone (ISD) 44-1-393 3318

PULMONARY FITNESS FOR DIVERS

Thursday, 26th April 1990
Birmingham, Alabama, U.S.A.

A one day revision course for HSE Approved doctors. Fitness criteria will be reviewed for professional divers in the commercial diving industry and those in the sports diving industry. This year the focus will be on pulmonary assessment and laboratory investigations.

The course will be run by Biomedical Seminars in association with the
Annual Meeting of the
GULF COAST CHAPTER OF THE UNDERSEA AND HYPERBARIC MEDICAL SOCIETY
28/29th April, 1990

Details can be obtained from any of the following:

Miss Jane Dunne
Undersea & Hyperbaric Medical Society
9650 Rockville Pike

Bethesda,
Maryland 20814,
U.S.A.

Mrs Karen Reeves,
Biomedical Seminars,
7 Lyncroft Gardens,
Ewell, Epsom.
Surrey KT17 1UR,
ENGLAND.

Miss Lynne Scoggins, R.N.
Hyperbaric Medicine Department,
Carraway Methodist Medical Center,
1600 26th Street,
N. Birmingham,
Alabama 35234,
U.S.A.

JOINT MEETING ON
DIVING AND HYPERBARIC MEDICINE
AMSTERDAM
August 11 - 18, 1990

Xth International Congress on Hyperbaric Medicine
Xth International Symposium of the Undersea and Hyperbaric Medical Society
XVIth Annual Meeting of the European Undersea Biomedical Society

THEMES

Oxygen Toxicity
Deep Sea Diving
Shallow water Diving
Decompression Sickness
HPNS
HBO Therapy
Technical Aspects of HBO Chambers
Nursing Problems in HBO Environment
Free Papers

The Meeting will be held at the

HOTEL OKURA AMSTERDAM
Ferdinand Bolstraat 333
1072 LH Amsterdam
THE NETHERLANDS
Tel: (020) 78 7111

Hotel Okura Amsterdam is within close proximity to Schiphol International Airport (15 minutes) and major highways A4 and A10. The hotel is only 10 minutes from Amsterdam's bustling city centre.

Detailed information regarding hotel accommodation for participants and guests will be available in November 1989.

For further details of the meeting contact the Secretariat of the Joint Meeting

Mrs Lidy Schipmölder-de Bruyn
Academic Medical Centre, G4-105
Meibergdreef 9
1105 AZ Amsterdam
The Netherlands
Tel: (020) 566 5781
Telex: 11944
Fax: 31-30-566 4440

Official Carrier

KLM Royal Dutch Airlines has been appointed Official Carrier for the Joint Meeting. KLM offices all over the world will meet your travel requirements.

ASSOCIATED COURSES

Dr David H. Elliott is preparing a modular pre- and post-course, meeting the training recommendations of DMAC and NATA on the Medical Aspects of Diving Accidents and Illnesses. The plans are as follows:

Compressed Air Diving.
A practical course in diving medicine for doctors, at Den Helder with the Royal Netherlands Navy, 6-9 August 1990.

The Medical Management of Decompression Illnesses.
An advanced seminar and review, at the Hotel Okura Amsterdam, 10-11 August 1990.

Mixed Gas Diving.
An advanced course for doctors at the Cattedra di Medicina Subaquea ed Iperbarica and with the co-operation of the Italian Navy, 20-24 August 1990.

For additional information about thesecourses contact

Dr. David H. Elliott, Courses Co-ordinator,
Biomedical Seminars,
7 Lyncroft Gardens,
Ewell, Epsom,
Surrey KT17 1UR,
ENGLAND.

GLEANINGS FROM MEDICAL JOURNALS

The following articles have come to the notice of the editorial staff and these notes are printed to bring them to the attention of members of SPUMS. They are listed under various headings of interest to divers. Any reader who comes across an interesting article is requested to forward the reference to the Journal for inclusion in this column.

DECOMPRESSION PROBLEMS

Cerebral perfusion deficits in dysbaric illness

G H Adkisson[1], M A Macleod[2], M Hodgson[1], J J W Sykes[1], F Smith[3], C Strack[1], Z Torok[4], R R Pearson[1]. Cerebral perfusion deficits in dysbaric illness. *Lancet* 1989; ii: 119-121.

From Institute of Naval Medicine, Alverstoke, Hants PO12 2DL[1], Royal Naval Hospital,Haslar, Gosport[2], Aberdeen Royal Infirmary, Aberdeen[3], and Admiralty Research Establishment, Alverstoke[4].

Summary

Decompression sickness (DCS) is usually categorised as type I (mild; peripheral pain, non-neurological) or type II (serious; neurological). Type II is regarded as predominantly a spinal cord disease with infrequent cerebral involvement. Cerebral perfusion was studied by injection of $^{99}Tc^{m}$-hexamethylpropyleneamine oxime and single photon emission tomography in 28 divers with confirmed incidents of DCS and cerebral arterial gas embolism (CAGE). Cerebral perfusion deficits were present in all 23 cases of type II DCS and in all 4 cases of CAGE. No deficits were present in the single case of type I DCS. Type II DCS should be recognised as a diffuse, multifocal, central nervous system disease.

Cerebral perfusion deficits in dysbaric illness.

Lancet 1989 ; ii: 674-5.

(Letter) Wilmshurst P and Nunan T.O. From the Department of Cardiology, St Thomas' Hospital, London SE1 7EH, England.

(Reply) Adkisson G.H. From the Institute of Naval Medicine, Alverstoke, Hants PO12 2DL.

CARBON MONOXIDE POISONING

Hyperbaric and Normobaric Oxygen in Acute Carbon Monoxide Poisoning

Lancet . 1989; ii: 799-800.

(Letter) James P.B. From Department of Community Medicine, Wolfson Institute of Occupational Health, Ninewells, Dundee DD1 92Y, Scotland.

OXYGEN TOXICITY

Biochemistry of reoxygenation injury.

L. Ernster. Biochemistry of reoxygenation injury.*Crit Care Med* 1988 Oct; 16(10): 947-953.

This paper summarizes current knowledge on the biochemistry of oxygen toxicity in general and ischemia-reoxygenation tissue injury in particular. The superoxide radial, hydrogen peroxide, and the hydroxyl radical in cells can be formed enzymically or non-enzymically. Primary effects of oxygen radicals result in lipid peroxidation, which is believed to be initiated by a perferryl radical. Secondary effects are believed to be due to a disturbance in cellular calcium homeostasis. Reactions and treatment potentials are highly complex and their effects on cells, tissues, and organism are difficult to predict. Treatment potentials include superoxide dismutase, catalase, calcium entry blockers, iron chelators, xanthine oxidase inhibitors, and agents to prevent leukocyte adhesion. Reoxygenation injury mechanisms during resuscitation from clinical death can be studied in animals by evaluating the effects of antireoxygenation injury therapies and by monitoring free radical reactions.

Keywords
reoxygenation/ oxygen toxicity/ hydrogen peroxide.

IMMERSION

The resuscitation of immersion victims.

A.D. Simcock. The resuscitation of immersion victims. *Appl Cardiopulmonary Pathophysio* 1989; 2: 293-298.

This paper reviews the outcome of 150 victims of drowning and near-drowning brought to a district general hospital close to the sea. The pathophysiology of the drowning process is reviewed. Patients were treated immediately on arrival by a resuscitation team. Respiratory difficulties were relieved as quickly as possible. Common problems were hypoxia, hypothermia, acidosis and low blood pressure. The apparently dead were assessed very carefully. The results show an excellent prognosis for those patients who had not suffered cardiac arrest before arrival. There were, however, two survivors from the cardiac arrest group. Survival rates in this group will only improve if the hypoxia is relieved before cerebral damage occurs.

Keywords
near-drowning/ hypoxia/ cardiac arrest/ death.

SELECTED ABSTRACTS

Reprinted from the Program and Abstractsof the UNDERSEA AND HYPERBARIC MEDICAL SOCIETY ANNUAL SCIENTIFIC MEETING 6-11 June 1989

PORTABLE CHAMBERS

Protocols for the use of portable hyperbaric chamber for the high altitude disorders.

R.L. Taber.
Department of Emergency Medicine, St. Luke's Hospital, Denver, Colorado, U.S.A.

Despite a recent explosion in research on high altitude illness, descent remains the definitive treatment. However, in alpine settings of over 10,000 feet where these problems arise, immediate evacuation is often impossible. Recently, a portable, lightweight (7kg) hyperbaric bag has been developed capable of withstanding a pressure of 2psi. The bag's use has been reported anecdotally in the past few years as a means of simulating descent and thereby acting as a treatment modality. However, no work has been conducted to determine the optimal length of time of treatment in the bag to achieve resolution of symptoms and to prevent recurrence once the patient is out of the pressurized bag and "returns" to altitude. This was evaluated at the Himalayan Rescue Association's altitude research clinic at 14,150 feet in Pheriche, Nepal. At this altitude, inflation of the bag to 2psi effects a descent to 8,200 feet. By providing such descent, dramatic improvements could be seen with the symptoms of acute montain sickness (AMS), high altitude pulmonary edema (HAPE) and cerebral edema (HACE). At the clinic, patients were evaluated and diagnosed accordingly and those with potentially fatal HAPE and HACE were arranged for immediate evacuation. At times this was not feasible and these patients were put in the bag. Repeat serial observation of symptoms and recovery revealed that with 15 AMS, nine HAPE and seven HACE patients, time frames of two, four and six hours respectively were required to provide resolution of the symptoms with no subsequent complications or deterioration. The author advocates the role of the bag as an effective adjunctive and temporizing measure for the treatment of HAPE and HACE.

PATIENT TRANSPORT

Transportation of the critically ill hyperbaric patient.

Brian A. Youn M.D.* and Roy A.M. Myers M.D.+
* Geisinger Medical Center Danville, P.A. 17822, U.S.A.
+ Maryland Institute for Emergency Medical Service Systems, Baltimore, M.D., U.S.A.

In the Department of Hyperbaric Medicine at the University of Maryland, treatment of the critically ill ventilator dependent patient with hemodynamic monitoring makes up a significant proportion of the patients treated each year. Because of the growing concern about transportation and unique needs of the hyperbaric patient, we prospectively evaluated transportation in the ICU/hyperbaric patient. This study was a prospective pilot study of 20 transportations of the critically ill ventilator dependent patients with hemodynamic monitoring to and from the ICU. The following parameters were monitored: heart rate, respiratory rate, tidal volume, blood pressure, continuous pulse saturation and pulmonary artery, central or intracranial pressures if available. The pilot study revealed many problems in utilizing transport monitoring equipment for hyperbaric patients principally due to size constraints. In three of the 20 patients monitoring detected ventilator malfunction (two patients) and hypoxemia secondary to pneumothorax (one patient). In all three cases monitoring led to corrective action. Monitoring during transportation of the critically ill hyperbaric chamber patient is important to provide state-of-the-art care of these patients. Although problems are infrequent during transportation, special monitoring may detect equipment failure or patient distress that requires immediate attention. Further data and special equipment recommendations will be presented.

ANAESTHETIC VAPORISERS

Anaesthetic vaporizer performance under hyperbaric conditions.

J.M. Satterfield, G.B. Russell, J.M. Graybeal and R.B. Richard.
Department of Anesthesia, The Pennsylvania State University College of Medicine, Hershey, P.A. 17033, U.S.A.

Underseas projects with prolonged hyperbaric exposure present special anesthetic challenges when emergency surgery becomes necessary. Volatile anesthetic agents such as isoflurane may offer particular advantages in such situations. The output of volatile anesthetic vaporizers at hyperbaric pressures have not been rigorously studied. Henry's Law dictates that atmospheric pressure does not affect volatile anesthetic partial pressure (the factor determining anesthetic affect). Previous studies conclude that vaporizer output increased under hyperbaric conditions when compared to 1 atmospheric absolute (ATA). Isoflurane partial pressures from a Vapor 19.1 vaporizer (North American Drager) were measured by a Perkin Elmer 110 MGA mass spectrometer at 1, 2, 3 and 4 ATA during varied mixtures and flow rates of O2, N_2O and air. Carrier gas composition and/or flow rates did not affect vaporizer output of the volatile agent. Vaporizer output increased linearly in response to varied vaporizer dial settings from 0.4 to 2.0%. At >1 ATA vaporizer output was significantly less than ($p<0.5$) that at 1 ATA. However, the variation in output fell within the normal clinical range of accepted vaporizer output (±20%). Isoflurane can be delivered in a clinically acceptable range at hyperbaric pressures with presently available vaporizers.

PAIN RELIEF FROM INCREASED PRESSURE

Analgesia produced by scuba diving.

Robert P. Iacono M.D. and Laura K. Pomerenke M.D.
Dept. of Surgery, University of Arizona Health Sciences Center, Tuscon, Arizona 85724, U.S.A.

Strenuous exercise, noxious stimuli and other forms of stress are known to cause release of central endorphins. Two patients with chronic pain due to spinal injuries experienced dramatic and consistent relief of symptoms with scuba diving. The patients, aged 35 and 28, sustained spinal injuries during military service with transient paraparesis 14 and 10 years respectively prior to presentation. Each had undergone operative procedures for spinal decompression and in one for Harrington-rod stabilisation, but developed chronic back and leg pain which were resistant to both conservative and surgical management. These patients, however, experienced incidental pain relief with recreational scuba diving. In each patient pain relief occurred immediately after ocean dives below 50 feet (2.5 ATA). Analgesia persisted for an average of five hours with a maximum duration of up to nine hours after surfacing. This affect was reproduced on each occasion where diving pressures reached 2.5 ATA or greater, and no habituation or tolerance occurred following multiple successive days of repetitive diving. Scuba diving was thus pursued by the patients specifically for pain control. Scuba diving with its pressure-induced stresses may be associated with activiation of central endorphins and provide yet another example of human stress-induced analgesia. In addition, endorphinergic mechanisms may be implicated in some of the therapeutic effects of hyperbaric oxygen treatment, which will be discussed in relationship to proposed models for further investigation.

DECOMPRESSION SICKNESS

Responses of decompression computers, tables and models to"yo-yo" diving

M.H. Hahn,
Institut fuer Biophysik & E.M., Med. Einr. d. Uni, Moorenstr. 5, D-4000 Duesseldorf 1, Federal Republic of Germany.

Edmonds drew attention to a habit of multiple repetitive diving, called “yo-yo” diving: emptying a 72 cuft tank at about 120 fsw, taking a surface interval of 1-2 hours, going for a similar dive again a.s.o. for a whole day (eg. on Truk Lagoon). Also instrutors busy all day in teaching and taking exams may pile up similar profiles.

Most table reading rules do not forbid this, although repetitive systems were obviously not designed with “yo-yo” profiles in mind, let alone chamber testing of such profiles. Edmonds already lashed the fact, that the “low-pass-filter” properties of monoexponential, parallel compartment models, used in some decompression computers yield no specific “punishment” for this kind of diving.

The responses of various brands of ’89-generation decompression computers as well as table repetitive systems and published decompression models to some idealized “yo-yo” profiles are displayed.

Conservative aspects of dive computer decompression algorithms.

P.A.Heinmiller.
ORCA Industries, Inc., 10 Airport Way, Toughkenamon, Pennsylvania 19374, U.S.A.

Recent attention has been given to the fact that most dive computers allow more repetitive diving and longer multi-level dives than the U.S. Navy tables. Little notice has been taken of the conservative simplicications made to existing models when implemented as computer algorithsms, and to the effects of these modifications upon dive profiles. The ORCA decompression algorithm utilizes the Workman equations, in the form $M = M_o + {}^{\Delta}M * D$, where M_o is the maximum surfacing compartment pressure and ${}^{\Delta}M$ is the increase in allowed compartment pressure with depth. The conservative aspects come from the selection of constants M_o and ${}^{\Delta}M$, and the computer’s use of the results of the calculation. First, M_o values are selected that produce no-decompression limits similar to the doppler no bubble limits, rather than the symptom based limits of the U.S. Navy. Second, the inclusion of slower compartments, in the 120 to 480 minute half-time range, produces less available dive time than tables, beyond the third or fouth day of an intensive schedule. Third, the no-decompression limit is defined as the time to reach the controlling compartment M_o at depth, not including outgassing during ascent to the surface. Fourth, the use of a ${}^{\Delta}M$ of 1.0 in Workman’s equations to simplify computer processing produces decompression ceilings that are 1.5 to 2.2 times deeper than the Navy model for similar compartment pressures. Fifth, ascent rates as slow as 20 feet per minute, enforced by visual feedback, produce more controlled ascents in the field. Finally, in the latest addition to the algorithm, altitude modification of M_o produces acceptably conservative time limits at altitude without reduction of sea level limits. As a result of these conservative elements, over 1.5 million dives have been made on the ORCA algorithm, with an overall field bends rate less than that of the U.S. Navy tables.

Further selections of abstracts from this meeting will appear in later issues of the SPUMS Journal. The next Annual Scientific Meeting of the Undersea and Hyperbaric Medical Society will be the Joint Meeting with the European Undersea Biomedical Society and theInternational Congress on Hyperbaric Medicine to be held in August 1990 in Amsterdam. *See page 205 for further details.*