The Editor's Offering

This year's Annual Scientific Meeting (ASM) is from May 8th to 17th. The recent devaluation of the Australian dollar has sent the cost of the air fare, accommodation and meals package up by just over $Aust 400. Such are the costs of visiting countries where all charges are in $US. It is to be hoped that this rise in cost will not keep members from making the trip to what promises to be an interesting and informative meeting. The workshop on the Aging Diver should be of interest to many members who have been regular attendees at ASMs. Like everyone else they are getting older. They will have their opportunity to put their views forward from an over-40s' perspective. Yes, there are people around whose medical opinion is that divers over 40 are "older" and should be put through a battery of tests before being passed fit to dive. What are they going to recommend to divers who hold a Senior Citizens card, available to the over 60s who work less than 20 hours a week? Ring up Allways Dive Expeditions, the official travel agents, and book your trip to find out.

For all who live by, or visit, the sea or own a swimming pool drowning is a topic they should know about. This summer at least 19 people in Victoria died in the surf because they did not swim between the flags. Carl Edmonds' paper about the drowning mechanisms clearly explains the history of investigations of the mechanisms and describes the pathophysiology which leads to death. The salt water aspiration syndrome, first described from the Royal Australian Navy (RAN) School of Underwater Medicine over 25 years ago, can well be described as minimal drowning. The Editor has suffered from it on two occasions. The first was after a dive doing buddy breathing in the sea water filled swimming pool at HMAS PENGUIN and the second was during the 1984 ASM at Phuket in Thailand. On both occasions he was distinctly blue around the edges, clearly demonstrating the effects a small amount of sea water in the lungs on oxygenation. Incidentally fresh water does the same nasty trick.

Graham Simpson has had the luck to find three "resort divers" over a period of three years who developed shortness of breath after their escorted "dive experience" sufficiently disabling to make them seek medical assistance. They all suffered from a giant bulla in their lungs, the expansion of which, to the size of a large pneumothorax, had caused their symptoms. Treatment with normobaric oxygen resulted in improvement in all cases, although one later required surgery. Is it time to reinstate chest X-ray in the diving medical? Or is the chance of detection too low to be worth the cost and exposure to radiation?

We publish a letter from Germany, e-mailed to the Editor, about diving for the disabled. The writer's institution has been investigating scuba diving as an adjunct to physiotherapy for those who have suffered severe neurological damage affecting the legs. We also publish two letters proposing a Diver Support Network for those who have diving accidents and consequent hyperbaric treatment. The idea is to provide names and addresses of people who are willing to help these victims rehabilitate themselves and know what they are talking about either by diving medicine training or by having suffered the same way themselves.

The papers from the 1997 ASM are all concerned with decompression illness (DCI). James Francis' paper on the pathophysiology of spinal DCI is of great interest. The Editor has always had difficulty accepting that spinal DCI was caused by arterial gas emboli (AGE) as spinal DCI is seldom accompanied by the signs of cerebral AGE. The theory of back pressure of bubbles blocking the valveless spinal venous plexus has a certain elegance but the simplest idea, that bubbles formed the fatty parts of the spinal cord, as they had been demonstrated to do in the omentum of experimental animals, always seemed the most likely. Pressure on nerves causes neuropraxia, easily demonstrated by sitting with one knee over the other until the leg "goes to sleep" and then trying to stand up without support. Spontaneous recovery from DCI induced paraplegia has been recorded from the early days of diving and compressed air work. It would appear that designing experiments which regularly produced paralysis in animals required the equivalent to exposures well outside those giving rise to occasional case of DCI paralysis in humans, so resulting in greater damage, which may explain why autochthonous bubbles were disregarded as a cause. Richard Moon writes on assessing the diver with DCI. There are four papers on the emergency advice and treatment networks in the UK, USA [DAN (Divers Alert Network)], Australia and New Zealand [DES (Diver Emergency Service)].

The final paper is again about a long past world. Taken from *Historical Diver*, it covers a world record dive, in Lake Michigan in the (northern) winter of 1937, on heliox to the depth of 420 ft (127 m). The paper was not written by a medico but by a historian, however it is quite probable that the oxygen percentage was around 20% as the diver had one cylinder of heliox and one of oxygen, the flow of both controlled by the diver and oxygen was only used when he felt it was needed, connected to his self-contained rebreathing suit. He had to be able to tolerate surface pressure inside his suit. However at 127 m 20% oxygen would have given a partial pressure of 2.7 bar, quite enough to cause an oxygen convulsion. As the paper sellers on the street used to yell.

"Read all about it"

ORIGINAL PAPERS

DROWNING SYNDROMES: THE MECHANISM

Carl Edmonds

Key Words

Accidents, deaths, drowning, incidents, near drowning, salt water aspiration

Abstract

The drowning syndromes should be viewed as a continuum between the aspiration of a relatively small amount of water, causing symptoms and respiratory-based signs, through near-drowning, in which there is loss of consciousness but with survival, to the fatal cases of drowning. The latter rarely involve the gross haemodynamic and biochemical changes seen in some animal experiments.

The behaviour of the victims, animal and human, during the incident is reviewed, as are the experiments conducted on animals, with various types and quantities of aspirate, to model the physiology. These experiments are compared with adult human clinical case series. "Quiet" drownings are described and classified. The clinical features of near drowning are reviewed. Factors which influence survival are noted.

The pathological findings are discussed, with a critical approach to the concept of "dry" drowning, and some postulates on the findings of cranial haemorrhages. Lungs are the primary and dominant organ involved and hypoxia is the major physiological abnormality.

The salt water aspiration syndrome, including its development, clinical and laboratory findings as seen in scuba divers, is also reviewed.

Finally a brief review of the literature specific to scuba drownings is given.

Introduction

Excellent general reviews on this subject have been presented by Donald, Modell, Tabeling, Neuman and the UHMS Workshop on Drowning at Cancun, Mexico in June 1997.[1-6] This paper is the basis of one of the presentations at the UHMS Workshop.

Terminology

Drowning refers to the death of an air-breathing animal due to immersion in fluid.

Delayed drowning or *secondary drowning* occurs when the victim appears to recover from the incident, but then proceeds to die.

Near-drowning refers to the loss of consciousness from the incident, which did not lead to death.

The *aspiration syndrome* refers to the effects of aspiration of fluid into the lungs, but without loss of consciousness.

There is a continuum in the severity of symptoms and signs between aspiration, near-drowning and drowning. They can be incorporated together as *the drowning syndromes*, for a greater understanding of each. The continuum needs to be appreciated if a rational approach to the management of near-drowning is to be made.

Post-immersion syndromes refer to the disorders that develop after immersion and subsequent rescue.

Other classifications have been proposed, based on the type and amount of fluid inhaled. These will be referred to later.

Behaviour during drowning

Over the range of animals tested and observed, consciousness is usually lost within 3 minutes of total submersion.

Observation on experimental drownings showed the typical behaviour of *animals* was an immediate struggle for freedom, sometimes with an inhalation.[7] This was followed by suspension of movement, possible exhalation of a little air and frequent swallowing. Later there was a violent struggle for freedom, followed by convulsive movements and the exhalation of air with spasmodic inspiratory efforts, then death.

Observations of *human* drownings parallel the animal experimentation, involving a panic reaction with violent struggling followed by automatic swimming movements.[8,9] There may be a period of breath holding and swallowing of large amounts of water. Vomiting may occur, followed by gasping and aspiration of water. Blood stained froth develops in the airways and the patient convulses and then dies.

More recently the observations on children exposed to drown-proof training, as it is euphemistically called, has modified our knowledge of the normal response in human drownings.[10] There is usually a failure of the infant to struggle. The child holds its breath and makes automatic

but ineffectual paddling type movements as the child sinks to the bottom. For many years, drowning was considered a "fight for survival",[2] but this is now changing.[3]

When a fully conscious human accidentally falls in the water, he or she fights to survive. In other circumstances drowning may proceed in a quiet and apparently unemotional manner. Examples of *quiet drownings* include:

1 Hyperventilation with breathhold diving. Craig reported eight cases of hyperventilation before breathhold diving, which resulted in loss of consciousness due to the development of hypoxia.[11] This developed before the blood carbon dioxide levels rose sufficiently to require surfacing to breathe. In these cases loss of consciousness occurred without any obvious warning and the underwater swimmer then aspirated and drowned.

2 Uncontrollable hyperventilation in cold water, hypothermia and/or cardiac arrhythmias, leading to loss of function and drowning have been well described by Keatinge and others.[12-14]

3 Drugs and alcohol increase the incidence of drowning by impairing judgment, increasing risk taking, vomiting, heat loss and reducing the physical and emotional capacity in the struggle to survive.[3,15-17] It is likely that nitrogen narcosis may have a similar effect in divers.

4 Diving equipment problems may produce hypoxia. These include the dilution hypoxic effects with mixed gas breathing, ascent hypoxia and carbon monoxide toxicity by the blockage of oxygen metabolism. They are all likely to cause loss of consciousness without excess carbon dioxide accumulation, associated dyspnoea or distress.[17]

5 Salt water aspiration. In animals 2.2 ml of fresh water per kg body weight drops the PaO_2 to approximately 60 mm Hg within three minutes, or to 40 mm Hg with sea water.[18,19] A similar situation was observed clinically in the salt water aspiration syndrome of divers[20] Exercise may exacerbate this situation.

6 Other causes of unconsciousness leading to drowning have been described e.g. some marine animal envenomations, coincidental medical illnesses, cerebral arterial gas embolism (CAGE) etc. These may suppress the psychological stress component of the drowning.

Animal experiments

In the 1930s many animal experiments were conducted, both in Europe and North America, demonstrating that if an animal was immersed and drowned in water containing chemical traces or dyes, these would spread through the tracheo-bronchial tree to the alveoli surfaces.[7,21,22] In the case of fresh water, this was also absorbed into the blood stream.[21] A consistent fall in arterial oxygen content was observed, followed by a rise in arterial carbon dioxide and sometimes ventricular fibrillation.[23,24]

Swann and his colleagues from Texas, in a series of accurate but misleading experiments,[25,26] flooded animals' lungs with fresh or salt water and demonstrated significant differences between the two, due to osmotic pressures. In both cases, flooding of the lungs produced a reduction in $Pa0_2$ and pH, with a rise in the $PaCO_2$. This was attributed to airway obstruction.

Because fresh water is osmotically much weaker than blood, it moved into the bloodstream and produced haemodilution, reducing the concentration of most of the blood contents including proteins, sodium, chloride, etc. The subsequent reduction in the osmotic pressure of the blood resulted in haemolysis and liberation of both haemoglobin and potassium, with various metabolic and renal complications. Deaths were often cardiac in nature and due to ventricular fibrillation.

However, when the animals' lungs were flooded with sea water, which has a higher osmotic concentration than blood, water was drawn from the bloodstream into the lungs producing pulmonary oedema and haemoconcentration. This caused an increase in the haematocrit, blood proteins and electrolytes.

For many years physicians attempted to correct these presumed electrolyte, metabolic and cardiac abnormalities in human drownings, but their cases did not seem to conform to this animal model. Moreover, earlier workers had shown that in dogs that drowned there was still large volumes of air in the lungs,[7] as there is in humans. The Texan model had water-filled lungs.

Colebatch and Halmagyi, working in Australia in 1961, produced an animal model of drowning, more relevant as regards clinical management of patients, by aspiration of only 1-3 ml/kg body weight.[27-32] By using these smaller volumes they demonstrated the dominance of arterial hypoxia, largely independent of the amount of fluid inhaled. Pulmonary hypertension, vagal inhibition and reduced compliance were also noted. Sea water aspiration was usually associated with significant pulmonary oedema but the fresh water was often absorbed from the lungs within 2 to 3 minutes. Lung surfactant is affected differently by fresh water, which appears to destroy the surfactant, and salt water, which dilutes and washes it out.[33]

Subsequent animal experiments, using intermediate volumes of aspirate, by Modell and others, verified that shunting of blood, the perfusion of blood through non-

ventilated areas of lung, was the predominant factor causing persistent arterial hypoxaemia.[18,19,33]

Human series

Clinical series. described by Fuller in 1963,[34,35] Griffin in 1966[36] and Modell et al. in 1976,[4] illustrated the considerable differences between near-drowning humans and animals with flooded lungs. Human cases did, however, reflect the animal experiments in which smaller amounts of aspirate were administered. Significant electrolyte disturbances and cardiac arrhythmias were not frequent.

The initial symptoms were respiratory, then came the effects of hypoxia causing subsequent pulmonary and cerebral damage. Aspiration of vomitus, following the ingestion of sea water, was frequent in both the early and the resuscitation phases.

Investigations reveal: Hypoxaemia; a variable arterial $C0_2$; acidosis; the effects of reduced pulmonary compliance; patchy and variable consolidation in lung X-rays; and a polymorphonuclear leucocytosis. Others changes depend on other organ involvement.

Excellent reports of series of paediatric drownings have been published[10] and much work on the relevance of hypothermia to drowning has been reported. Neither will be dealt with here.

Survival from near-drowning

In human drownings, deterioration after initial resuscitation is frequently recorded and this influences management. The likely causes for delayed deaths include extensive and progressive lung damage, cerebral hypoxia, secondary infections (usually of the lungs), renal failure and iatrogenic factors.

Factors which negatively influence survival have been well documented by Modell.[3]

1 Prolonged immersion
2 Delay in effective cardio-pulmonary resuscitation
3 Severe metabolic acidosis (pH <7.1)
4 Asystole on admission to hospital
5 Fixed dilated pupils
6 A low Glasgow coma score (<5).

Nevertheless, none of these predictors is infallible and survival with normal cerebral function has been reported with all of the above factors. Even a flat EEG may be reversed.

Cases have been reported that have been submerged for between 15 and 45 minutes,[37-43] and have survived without neurological sequelae. There have been many other cases that have not been reported. Two such cases with which I have been involved were submerged for 15-20 minutes. Such cases have been used to encourage rescuers to persevere with resuscitation efforts.

The explanations offered for survival after such prolonged submersion are :

1 Hypothermia is protective and develops very rapidly with aspiration of water.[10,12,41-43] In swimmers and divers hypothermia may well develop before the incident.

2 The “diving reflex” is a possible, but contentious, explanation.[10,43] Within seconds of submersion the diving reflex (reflex inhibition of the respiratory centre in the medulla) may be triggered by sensory stimulation of the trigeminal nerve. This results in a bradycardia and shunting of the blood to the brain and coronary circulations. It is independent of baroreceptor or chemoreceptor inputs. The diving reflex is more intense in the frightened or startled animal, compared with those which dive or submerge voluntarily, but it is not known whether this is applicable to humans. Typically water temperatures above 20° do not inhibit the diving reflex, but progressively lower temperatures augment it.

3 Respiratory gas exchange in the lungs can continue after submersion. With or without the effects of laryngospasm, often some litres of air remain within the lungs, allowing for exchange of oxygen and carbon dioxide. Whether fluid enters the lungs in an unconscious victim will depend on many factors, including the orientation of the body. The nose and mouth being below the lungs will reduce the chances of fluid replacing air in the lungs. Increased pressure (depth) might increase the availability of oxygen uptake through Henry’s Law. In a comatose state, with low oxygen utilisation and the effects of hypothermia and the diving reflex, a retained respiratory gas volume might add considerably to the survival time, although this is not often mentioned in the drowning literature.

4 Gas exchange between the pulmonary blood and the aspirated fluid might have a marginal effect on prolonging life.

Despite the fact that spectacular and successful rescue can be achieved after prolonged submersion, it is more frequent that this is not so, many victims lose consciousness and die after only a few minutes of submersion.

Pathology[10,44-46]

Drowning is the commonest cause of recreational scuba deaths, but is usually a secondary effect, with the primary cause leading to loss of consciousness.[17] Other accidents (dysbaric, medical illnesses, trauma etc.), occurring whilst immersed or submerged, are likely to result in the secondary complication of drowning, with all its pathological sequelae. Drowning often complicates the interpretation of the diving accident and produces a combined pathology.

Various aspects of drowning pathology need to be addressed.

"Dry drowning"

Many references have been made to the possibility of drowning without any aspiration of fluid. It was stated by Cot in 1931 that 10% of victims of drowning do not aspirate water.[47] They died from acute asphyxia while submerged, this is now attributed to reflex laryngospasm.

Virtually every review of drowning over the rest of this century refers, without question, to this belief and the incidence is often extended to 20%. "Dry drowning" conflicts with the animal work of the 1930s, but was given support by Pearn in a fascinating philosophical review of pathophysiology, in 1985.[10] Pearn, who is an eminent paediatrician, admitted that it is not so frequent in children's drowning.

In 1993 a recent review by Modell reiterated the concept.[3] Three references were given. Two refer to autopsy findings,[16,44] the third to one of Modell's own papers,[4] however perusal of that paper reveals little supporting evidence. He stated that by the time 81 near-drowning/aspiration victims had reached hospital, 10 had a $Pa0_2$ of 80 mm Hg or greater. No information was available as to whether those cases were fresh or salt water victims, but the majority were in fresh water.

As it has been stated earlier, fresh water is absorbed very rapidly from the lungs, and therefore autopsy findings, and indeed the $Pa0_2$, cannot really be used to imply (let alone prove) the absence of an aspirant. Especially is this so when these investigations are performed some time after the event. So, there is good reason to question the literature on "dry drowning".

Reference to the anaesthetic literature is also informative. Miller's textbook of Anaesthesia,[48] with its associated references,[49-51] define and describe laryngospasm as the exaggerated and prolonged response of a protective glottic closure reflex. There is no airflow and the true vocal cords cannot be seen. The development of hypoxia and hypercarbia remove the effect. Thus laryngospasm eventually ceases spontaneously as hypoxia and hypercarbia develop. This implies that laryngospasm will not, by itself, be continued until death. The glottic closure will relax prior to death, allowing the passage of gases and fluids into the lungs.

In the absence of more definite information, it would probably be sensible to presume that all near-drowning or drowning victims have aspirated and base one's first aid and management on this presumption. "Dry drowning" could well be an artefact of fluid absorption from the lungs, or death from other causes. I have never witnessed it in divers who drowned in salt water.

Autopsy observations[10,44-46]

The theory and the practice of "drowning" autopsies are surprisingly contentious for such a common disorder. Frequently there are coincidental signs of immersion, marine animal injury or resuscitation damage. The stomach may contain swallowed fluid.

Autopsy examination of 118 consecutive drowning cases suggested that 85% aspirate 22 ml of fluid, or less, per kg body weight.[45] It was therefore considered unlikely that drowning victims die acutely of electrolyte imbalance and/or ventricular fibrillation. Death is more likely to be secondary to asphyxia and hypoxia.

The respiratory findings are congested, voluminous lungs and, in the airways; frothy, haemorrhagic sputum (especially in salt water cases), vomitus, foreign bodies and particulate matter. Respiratory infections, abscesses etc. are not infrequent if death is delayed. Otherwise there is little typical to describe macroscopically.

The pulmonary changes at autopsy reflect not only the pathology of drowning, but the effects of resuscitation and the changes in the lung fluids between the time of rescue and the time of death. Many such factors may influence the final macroscopic result.

Histological changes may demonstrate toxic effects both of chemicals and the specific aspirate. The surfactant changes, including denaturation, can progress even after apparent clinical improvement. The usual epithelial and endothelial changes, with detachment of the basilar membrane and cellular disruption have been described.[10] Usually death is due to progressive, or irreversible, pulmonary damage associated with the drowning per se and there are obvious reasons for this. They include progressive surfactant damage despite rescue, pneumonitis from the aspirate or vomitus, infections etc. Pulmonary oxygen toxicity, associated with resuscitation attempts, may also be present.

The major effects on the neurological system are those of hypoxic brain damage and subsequent cerebral oedema with raised intracranial pressure. Delayed drowning deaths follow damage to lungs, brain or kidneys.

Neither the Gettler chloride tests nor the specific gravity of serum can be relied upon to establish a diagnosis of death by drowning.

Identification and comparison of environmental and systemic diatoms has been recommended, but is complex and infrequently performed. Also it does not prove drowning, merely aspiration of water while the circulation is still functional.

Conventional pathology teaching claimed that mastoid and middle ear haemorrhages were indicative of drowning. As we explained in 1976 these haemorrhages are the sequel of barotrauma, not drowning.[46] The ready acceptance of this explanation by diving pathologists took us by surprise and I have decided to extend the concept a little further.

Autopsies on drowning cases who have submerged while still alive, although unconscious, may show other cranial haemorrhages which are sometimes interpreted as a cause of the accident. Meningeal haemorrhages, both dural and arachnoid, are frequently observed. These are usually not very extensive and are quite different to the brain haemorrhages of arterial gas embolism or decompression sickness. They are probably derived from the haemorrhages of descent sinus barotrauma, which ruptured into the cranial cavity when the enclosed and compressed gas expanded as the body surfaced.

Salt water aspiration syndrome

A common diving illness in the Royal Australian Navy (RAN) in the 1960s was the salt water aspiration syndrome.[17,20] It is called "salt water fever" by the Australian abalone divers. Its importance lay not only in the light it shed on near drowning cases, but also because it was often confused with other diving or infectious diseases.

This condition, which is due to the aspiration of small amounts of salt water during diving, may occur because of inexperience, during buddy-breathing training or due to a faulty regulator. At that time RAN regulators did not have purge valves. Novices were trained in buddy-breathing during their first dive, in the open ocean. This frequently led to aspiration of sea water. In other cases the aspiration occurred on the surface, after the diver had removed his regulator.

Experienced divers often developed it after a fast towed search, while abalone divers often used inadequate surface supply equipment and "leaky regulators". Their term "salt water fever" indicated that they were well aware of the cause.

Other water users to present with a similar disorder, but possibly not as frequently, include snorkellers, surfers and helicopter rescuees.

A prospective survey was carried out on 30 consecutive cases who presented for treatment, their symptomatology was documented and investigations performed. Subsequently, "volunteers" were encouraged to aspirate sea water through doctored demand valves. The clinical and laboratory manifestations they developed were consistent with those in the clinical series. The following observations were made on the clinical cases.

IMMEDIATE SYMPTOMS

On specific interrogation a history of aspiration was given in 27 (90%). Often this was not causally associated by the novice diver with the subsequent events. Over 90% noted an immediate post-dive cough, with or without sputum. It was usually suppressed during the dive. Only in the more serious cases was the sputum bloodstained, frothy and copious.

SUBSEQUENT SYMPTOMS

The wide range of symptoms included rigors, tremors or shivering; anorexia, nausea or vomiting; hot or cold sensations; dyspnoea; cough; sputum; headaches; malaise; and generalised aches. Table 1 gives the incidence of each symptom

TABLE 1
SYMPTOMS OF SALT WATER ASPIRATION

Symptoms	%
Rigors, tremors or shivering	87
Anorexia, nausea or vomiting	80
Hot or cold sensations	77
Dyspnoea	73
Cough	67
Sputum	67
Headaches	67
Malaise	53
Generalised aches	33

RESPIRATORY SYMPTOMS

There was often a period of 1-2 hours before dyspnoea, cough, sputum and retrosternal discomfort on inspiration developed. In the mild cases, respiratory symptoms persisted for only an hour or so while in the more severe cases they continued for days. The respiratory rate

roughly paralleled the degree of dyspnoea. Respiratory stimulants appeared to aggravate the dyspnoea and tachypnoea.

Physical examination of the chest in about half the cases revealed crepitations or occasional rhonchi, either generalised or local. Rarely, they were high pitched and similar to that of obstructive airways disease. Signs usually disappeared within the first 24 hours.

Administration of 100% oxygen was reliably effective in relieving respiratory symptoms and removing cyanosis when this was present.

X-ray of the chest revealed areas of patchy consolidation or a definite increase in respiratory markings in about half the cases. These usually cleared within 24 hours, but remained longer in severely affected cases. X-rays taken after the incident and repeated within a few hours sometimes showed a variation of the site of the radiological abnormality.

Expiratory spirometry performed repeatedly over the first six hours showed an average drop of 0.7 litres in both FEV_1 and FVC measurements. These usually reverted to baseline levels soon after this time, although the changes could persist in a lesser form for up to 24 hours. Even those patients who had no respiratory symptoms demonstrated a reduction in lung volumes. Arterial blood gases, when performed, revealed oxygen tensions of 40-75 mm Hg with low or normal carbon dioxide tensions.

GENERALISED SYMPTOMS

Patients complained of being feverish, some with rigors, in most cases. Rigors were usually some hours after the aspiration of sea water. Malaise was the next most prominent feature. Headaches and generalised aches through the limbs, abdomen, back and chest were important in some cases, but usually not dominant. Anorexia was common and unexpected in this group of healthy young men.

In some there was an impairment of consciousness, including a transitory mild confusion (three cases), syncope with loss of consciousness on standing (two cases).

The feverish symptoms were interesting and are also seen in near-drowning cases. Shivering, similar in some cases to a rigor, and in some cases to generalised fasciculation, was a characteristic feature in the colder months. It was precipitated or aggravated by exposure to cold, exercise or breathing 10% oxygen (a research procedure, not recommended clinically). It was relieved by administration of 100 % oxygen. It occurred especially in those exposed to cold because of duration and depth of dive, clothing worn, and environmental conditions during the dive and subsequently. The association of shivering with hypoxia and cold has been described by others.[52] The shivering occurs concurrently with the pyrexia, which also takes an hour or two to develop.

Pyrexia was able to be verified in half the cases, up to 40°C (mean 38.1°C, SD 0.6), and the pulse rate was elevated (mean 102 per minute, SD 21), over the first six hours.

Some patients realised that relief from these symptoms could be obtained by either hot water baths or showers, or lying still in a very warm bed.

These systemic signs and symptoms also usually reverted to normal within six hours, and rarely persisted beyond 24 hours, unless the case was of considerable severity.

Haemoglobin, haematocrit, ESR and electrolytes remained normal. The white cell count was usually normal, although a mild leucocytosis (not in excess of 20, 000 per cu mm) was noted in a few cases, with a moderate polymorphonuclear increase and a shift to the left.

Lactic dehydrogenase estimations revealed a mild rise in some cases. X-ray and lung volume changes are described above.

A subsequent investigation into the causes of recreational scuba diving deaths[53,54] revealed that water aspiration was part of the sequence leading to death in 37% of the cases, often a consequence of equipment or technique problems. In these cases “leaking regulators” were often observed and commented on by the victim beforehand or demonstrated during the diving equipment investigation. Although there was often a fault in the actual regulator, with a failure of valve seating, the degree of leaking was frequently demonstrated to increase with the volume of air being required (e.g. with exertion, swimming against currents, panic etc.) and/or with a diminished line pressure to the second stage. Salt water aspiration often formed a vicious circle with panic and exhaustion.

Hypoxia from salt water aspiration, as could be expected, aggravated the problems of fatigue and exhaustion, and was a precursor to loss of consciousness (with or without dyspnoea) in both near drowning and drowning cases.

Discussion

There is no distinct division, in the initial presentation, between aspiration, near drowning and drowning. Aspiration syndromes merge with near drowning, often the intensity of symptoms and the degree of consciousness depend on circumstances, the activity of the victim and whether oxygen is being administered.

Near drowning cases sometimes die hours, or even days, later. They are then re-classified as delayed or secondary drowning. Some of the apparently drowned victims, by virtue of adequate CPR and enthusiastic intensive care management, surprisingly recover without sequelae.

In a prelude to the 1997 UHMS Workshop on Drowning and Diving, the Chairman made the following statement: "As you know, the drowning literature ignores diving whilst the diving literature ignores drowning."[55]

It is paradoxical that drowning, which causes more than 80 times the number of deaths in recreational divers than either decompression sickness or contaminated air, does not rate more than a paragraph or two in some diving medical texts. Nevertheless, of the major seminal reviews presented on this subject, many have been by diving physicians.[1,5,6]

In reviewing the literature on drowning, the only papers that I could find that specifically relate any of the drowning syndromes to scuba diving, was my own one on the salt water aspiration syndrome,[20] and one with an anecdotal review followed by a case report.[56]

One can only assume that it is too common a disorder to excite much academic interest.

In general, apart from the treatment of the near drowned, which is frequently reviewed, very little critical thought or assessment has been directed towards the literature that is available and most reviews are merely a rehash of previous presentations. Even more uncommon is the addressing of the problem of drowning syndromes with scuba, either death or accidents.[57]

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Dr Carl Edmonds, FRANZCP, FRACP, Dip DHM, who was the one of the founders and the first President of SPUMS, is Director of the Diving Medical Centre, 66 Pacific Highway, St Leonards, New South Wales 2065, Australia. Phone +61-(02)-9437-6681. Fax +61-(02)-9906-3559.

This paper served as the basis for a presentation to the UHMS Workshop on Drowning and Near Drowning, June 1997, at Cancun, Mexico.

ROYAL AUSTRALIAN NAVY
MEDICAL OFFICERS UNDERWATER MEDICINE COURSE
November 1998

For further details apply directly to
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PRIMARY LUNG BULLAE AND SCUBA DIVING

Graham Simpson

Key Words

Case reports, fitness to dive, medical conditions and problems, pulmonary barotrauma, resort diving, treatment.

Summary

Three cases are described where unsuspected congenital lung bullae led to diving accidents. In two cases the bullae ruptured causing spontaneous pneumothorax and in the third a tension bulla led to symptoms. All incidents occurred on introductory "resort" dives. The implications with regard to fitness to dive medicals are discussed.

Introduction

Current Australian standards for diving medicals lay considerable emphasis on detection of conditions that may cause air trapping because of the potential risk of barotrauma. Attention is generally concentrated on the presence or absence of airflow limitation and particularly asthma, although the evidence that asthmatics are actually at greater risk of barotrauma is hard to find and the objections are largely based on theoretical considerations. Standards for recreational scuba diving do not mention bullous disease, although this is mentioned in the Australian Standard AS1299 (1992) for underwater workers for whom a chest radiograph is mandatory. I report three patients with asymptomatic primary bullous disease who all developed life threatening complications of their lung bullae on "resort" scuba dives.

Case 1

A 19 year old Canadian tourist was admitted to hospital as an emergency. He gave no past history of asthma or any respiratory symptoms and was normally fit and well. He is a lifelong non-smoker. Earlier on the day of admission he had performed two resort scuba dives. The first dive was to 10 m for 20 minutes and he had no problems. His second dive was to 6 m for 30 minutes. On ascent he felt tightness in his throat and after surfacing was aware of some alteration of his voice quality. On examination he had surgical emphysema at the root of the neck and his chest radiograph confirmed a small right apical pneumothorax with some mediastinal emphysema. There were no signs of cerebral arterial gas embolism or any other decompression illness. The chest radiograph (Figure 1) also shows a large thin walled bulla in the right upper lobe containing a small amount of fluid. He was treated with 100% oxygen overnight and by the following morning his symptoms had improved and he was discharged. Follow up x-ray one week later showed complete resolution of the pneumothorax, but persistence of the right upper lobe bulla. He flew home to Canada one week later without incident.

Case 2

A 19 year old English tourist with no past history of cigarette smoking, asthma or other respiratory problems presented to a general practitioner one day after doing two resort scuba dives. He had never dived before. His first dive was to 10 m with a bottom time of 15 minutes and he experienced no problems. He enjoyed the experience so much he had a second dive again to 10 m, but for this time for around 20 minutes. He did a safety stop at 3 m for five minutes and then ascended to the surface. As he came up he developed pain in the left side of his chest which he described as more of a pressure than a sharp pain, but he did get some discomfort on taking a deep breath. He had no other symptoms. The general practitioner sent him for a chest radiograph which was reported by a consultant radiologist as showing a large left pneumothorax and he was referred for a specialist opinion regarding insertion of an intercostal drain. On examination he did have diminished, but not absent breath sounds over the left hemithorax and his chest x-ray (Figure 2) is more in keeping with a giant bulla than a pneumothorax. He was admitted to hospital and treated with 100% oxygen. A CT scan of the chest confirmed that he had a very large bulla occupying most of the left upper lobe, but which had not ruptured. His symptoms improved overnight. Follow up chest x-ray two weeks later showed no change and he flew back to England without further incident. It seemed likely that his symptoms were related to expansion of the bulla on ascent causing compression of neighbouring structures.

Case 3

A 30 year old English tourist was admitted as an emergency with severe right sided chest pain and breathlessness following his first resort scuba dive. He is a non-smoker with no past history of asthma or other respiratory problems. He had dived to 7 m for 30 minutes. He ascended in a controlled fashion, but as he approached the surface developed pleuritic right sided chest pain and breathlessness. Chest x-ray (Figure 3) showed a right sided pneumothorax and a very large thin walled bulla containing a significant amount of fluid, presumably blood. As he was significantly breathless it was decided to insert an intercostal drain. Because of the danger of puncturing the bulla this was done after a CT scan of the chest. This confirmed almost complete collapse of the lung and the presence of a large bulla in the right upper lobe. The bulla had however, deflated considerably compared with its appearance on the chest x-ray performed some hours earlier. The pneumothorax resolved and follow up x-rays

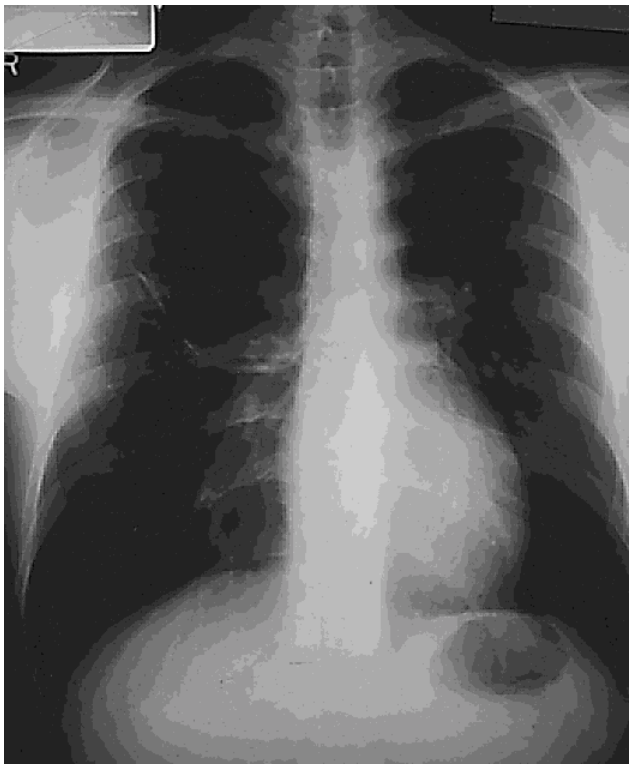

Figure 1. Chest radiograph (Case 1) showing small right apical pneumothorax with mediastinal emphysema and a large bulla in the right upper lobe.

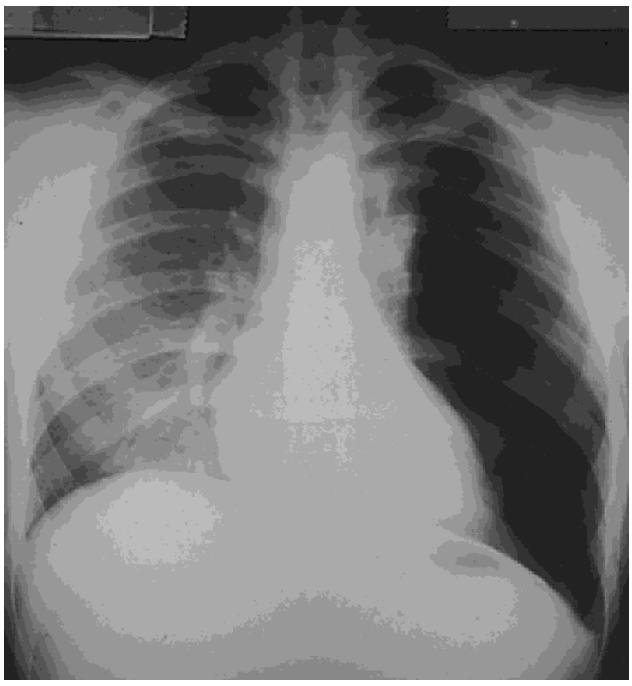

Figure 2. Chest radiograph (Case 2) showing giant bulla in left upper lobe but no pneumothorax.

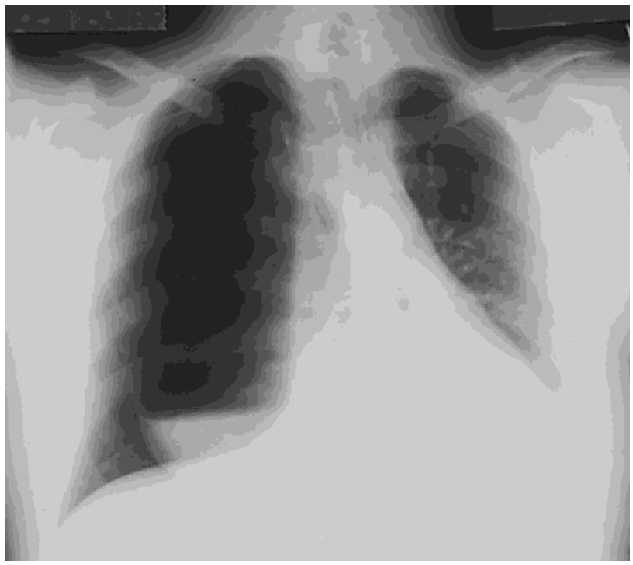

Figure 3. Chest radiograph (Case 3) showing large right pneumothorax with giant right upper lobe bulla containing fluid.

confirmed the persistent bulla in the right upper lobe. Unfortunately this became infected and was later resected at thoracotomy. He was allowed to fly home two weeks later.

Discussion

Large bullae occurring in the lung in the absence of any other lung disease are not infrequent, though there seems to be no information as to the exact prevalence in the normal population.[1] They are more frequent in patients with Marfan's syndrome and Ehlers-Danlos syndrome with a prevalence around 10% in both conditions.[2,3] However, in the absence of other pathology most bullae do not cause any symptoms. The mechanism of formation of primary bullae is not understood. In order to remain air containing they obviously have to communicate with the tracheobronchial tree, but ventilation of these bullae is often poor and, on ascent from a scuba dive, there must be a considerable risk of expansion of the bulla with risk of rupture. This is not inevitable, as I have seen one Japanese diving instructor who was found to have a large apical bulla after successfully completing over 150 dives without any problem.

All three patients in this series presented to one practitioner over a three year period in a single centre and the problem may therefore be a significant one. All developed problems during resort dives which do not involve any pre-dive medical assessment. However, in none of these cases would the standard SPUMS medical for recreational scuba diving have detected the problem.

Although all of these instances took place on introductory resort dives where the diver had not undergone any training before diving there was no suggestion in any case that any of these divers had held their breath on ascent and increased the potential for barotrauma. It may be that there is a case to be made for a plain chest radiograph to be included in all diving medicals. It seems at least possible that a routine chest radiograph would prevent more diving accidents than the bronchial provocation testing which is so widely performed and for which the justification is entirely theoretical. Plain chest radiographs however, do not detect bullae reliably[4] though films taken at full expiration are more sensitive.[1]

One final point concerns Case 2 where symptoms were caused by acute enlargement of the giant bulla on ascent rather than rupture causing pneumothorax. Giant bullae can easily be confused with a pneumothorax, but insertion of a chest drain in these circumstances is exceedingly hazardous and is very likely to lead to broncho-pleural fistula and necessitate thoracic surgery. Unless the patient is *in extremis* intercostal drains should not be inserted into pneumothoraces before specialist advice is obtained.

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UHMS ANNUAL MEETINGS 1998 AND 1999

The UHMS annual meeting for 1998 will be in Seattle from 20th-24th May. This date was chosen so that UHMS could meet with the Aerospace Medical Association.

The 1999 meeting will either be in Boston or Philadelphia in June or July.

For further details contact

Undersea and Hyperbaric Medical Society
10531 Metropolitan Avenue, Kensington
Maryland 20895-2627, USA.
Phone +1-301-942-2980
Fax +1-301-942-7804
URL = http//:www.uhms.org

IN-WATER RECOMPRESSION
A SYMPOSIUM AND WORKSHOP

Sunday, May 24, 1998 Memorial Day Weekend
at the
Seattle Sheraton Hotel
Washington, USA.
Registration: $125.00
(Workshop Proceedings $20.00)

For further information contact
Jane Dunne
UHMS Headquarters
10531 Metropolitan Avenue, Kensington
Maryland 20895-2627, USA.
Telephone +1-301-942-2980, Ext. 102

This interesting subject is now under scientific scrutiny. The Undersea and Hyperbaric Medical Society (UHMS) is hosting a symposium and workshop to be held in conjunction with the 1998 UHMS Annual Meeting in Seattle, Washington, USA. This event is co-chaired by Drs Merrill Spencer and Edmond Kay and is open to the public. Internationally known experts in Diving and Hyperbaric Medicine will address the issues of how and why a diver might accomplish In-Water Recompression (IWR). A crucial question to be addressed is when the benefits of early recompression outweigh the risks of this inherently difficult and sometimes dangerous procedure. The workshop objectives are to provide the Society and the diving community with guidelines for proper use including indications, triage, support. risks, benefits and contraindications to the use of IWR. The workshop is dedicated to Dr Ed Beckman and his pioneering work in IWR and early recompression.

To register for the IWR Workshop visit our web site and print out a "Fax Pre-Registration Form".
http://weber.u.washington.edu/~ekay/IRW.html

THE WORLD AS IT IS

INFORMATION FROM DAN SEAP

Key Words

Accidents, decompression illness, oxygen, training, transport.

Introduction

In their first newsletter for 1998 the Board and staff of Divers Alert Network S.E.Asia-Pacific (DAN SEAP) took the opportunity to provide members with certain important information that could affect all divers.

Evacuation costs

At the recent International Divers Alert Network (IDAN) Directors meeting in Tokyo it was noted that the number of evacuations for diving injuries is increasing rapidly, as is the cost of providing this service to the diving community.

Some of the most expensive evacuations have involved DAN SEAP members who have been evacuated to Australia, and elsewhere, from places such as Vanuatu, Papua New Guinea and the Solomon Islands. Such evacuations have cost up to $Aust 40,000 and there have been an alarming number of them in the past 2 years. Also, DAN SEAP members have constituted a disproportionately high number of evacuations for the actual number of members.

If this situation is not contained it will, without doubt, increase the cost of cover for such circumstances. Several travel insurers have become concerned about the cost of diving evacuations and it is possible that cover for divers could become more restricted sometime in the future unless the situation improves.

The re-emergence of deep diving and the easy access to deep dive sites in remote locations, without appropriate medical facilities, has played a major part in the increase in cases of decompression illness (DCI) requiring evacuation.

Divers must be aware that DCI can be extremely serious and can and does leave a substantial number of divers with permanent injury. The deeper the dive and the greater the number of dives, the higher the risk of DCI. In addition, too many divers have far too much faith in the ability of their dive computers and tables to protect them from decompression illness. Such devices have many limitations and most cases of DCI occur in divers who are diving within the limits of the computers and tables.

The sooner DCI is treated, the lower the likelihood of residual problems. Treatment involves immediate provision of 100% oxygen and recompression in an appropriate chamber with minimal delay. Evacuations from remote locations can often involve delays of up to 12-24 hours which is far from ideal.

Another topic of debate was the emergence of privately owned chambers for the treatment of recreational divers. Certain operators in remote places have purchased small recompression chambers in the hope that this will assist management of an injured diver. Unfortunately, such chambers may not adhere to appropriate maintenance requirements and safety standards. In addition, the staff available to manage the chamber may not always be adequately trained to deal with problems that can arise from time to time.

The need for adequate chamber maintenance and safety procedures became all too evident recently when a well-known and generally well-respected hospital-based chamber in Italy caught fire and claimed the lives of all eleven people inside.

These are issues that divers generally take for granted but are an everyday concern for DAN.

Divers lost at sea

In December, five DAN SEAP members disappeared after diving off Sulawesi in Indonesia. It appears that the divers surfaced some distance from the boat and were unable to attract the attention of the boat operator, despite using visual and audible devices. The dive guide who accompanied the group was picked up by a fishing vessel some time afterwards, but tragically, the five other divers still have not been found.

The message from all of this grim news is to dive sensibly and conservatively, especially in remote areas. Consider where you are diving and take appropriate precautions to maximise your own safety.

DAN SEAP works hard to improve diver safety services throughout the S.E. Asia-Pacific Region, and we need the support of many divers so that we can continue to improve the effectiveness of our efforts. Please encourage your diving friends to join DAN and to support our programs. **Remember, we are not an insurance company but a non-profit, member-supported dive safety association. *Without support from divers DAN SEAP will not continue to exist.***

Oxygen instructor programs

Level 2 Instructor
Townsville, Queensland, Australia 4-5 April
Instructor-Trainer
Melbourne, Victoria, Australia 9-10 May

Application forms are available from DAN SEAP PO Box 134, Carnegie, Victoria 3163, Australia. Phone +61-3-9563-1151, fax +61-3-9563-1139, e-mail danseap@c031.aone.net.au .

Oxygen

DAN SEAP has gained approval for distributing a 635 litre aluminium oxygen cylinder. The cylinder, which is colour-coded black and white, is fitted with a pin-index medical oxygen valve. Its working pressure is 150 bar and it has been tested in accordance with Australian Standards. The price is $Aust 295.

HTNA Seminar in Townsville

The Hyperbaric Technicians and Nurses Association (HTNA) will hold its annual scientific meeting in Townsville, Queensland on 28-29 August 1998. DAN will be supporting a dive safety seminar day to be held before the HTNA meeting on 27 August. The Directors of the various IDAN organisations will present lectures on aspects of dive safety and accident management. Enquiries should be directed to Mr. David King (Phone +61-7-4781-9476, fax +61-7-4781-9582).

HYPERBARIC TECHNICIANS AND NURSES ASSOCIATION INC.
AUSTRALIAN AND NEW ZEALAND HYPERBARIC MEDICINE GROUP

6th Annual Scientific Meeting
On
Diving and Hyperbaric Medicine

August 28 - 29, 1998
Mercure Inn ,Townsville 4810, Queensland, Australia.

Closing Date for abstracts is 1st June 1998. For conference arrangements and abstract guidelines contact:

HTNA '98 Conference Committee
Townsville General Hospital Hyperbaric Medicine Unit
PO Box 670, Townsville 4810, Queensland, Australia
Phone +61-(0)7-4781-9455 or Fax +61-(0)7-4781-9582

SPUMS NOTICES

SOUTH PACIFIC UNDERWATER MEDICINE SOCIETY

DIPLOMA OF DIVING AND HYPERBARIC MEDICINE.

Requirements for candidates

In order for the Diploma of Diving and Hyperbaric Medicine to be awarded by the Society, the candidate must comply with the following conditions:

1 The candidate must be a financial member of the Society.

2 The candidate must supply documentary evidence of satisfactory completion of examined courses in both Basic and Advanced Hyperbaric and Diving Medicine at an institution approved by the Board of Censors of the Society.

3 The candidate must have completed at least six months full time, or equivalent part time, training in an approved Hyperbaric Medicine Unit.

4 All candidates will be required to advise the Board of Censors of their intended candidacy and to discuss the proposed subject matter of their thesis.

5 Having received prior approval of the subject matter by the Board of Censors, the candidate must submit a thesis, treatise or paper, in a form suitable for publication, for consideration by the Board of Censors.

Candidates are advised that preference will be given to papers reporting original basic or clinical research work. All clinical research material must be accompanied by documentary evidence of approval by an appropriate Ethics Committee.

Case reports may be acceptable provided they are thoroughly documented, the subject is extensively researched and is then discussed in depth. Reports of a single case will be deemed insufficient.

Review articles may be acceptable only if the review is of the world literature, it is thoroughly analysed and discussed and the subject matter has not received a similar review in recent times.

6 All successful thesis material becomes the property of the Society to be published as it deems fit.

7 The Board of Censors reserves the right to modify any of these requirements from time to time.

South Pacific Underwater Medicine Society Annual Scientific Meeting Palau May 8th-17th 1998

The Guest Speakers at this years meeting are **Professor David Elliott** (UK) and **Dr John Bevan** (UK). The Convener of the Annual Scientific Meeting is Dr Chris Acott.

The theme of this year's meeting is "**Highlights from the History of Diving and Diving Medicine"** and this year's workshop theme is "**The Ageing Diver".**

Those wishing to present papers are asked to contact:

Dr. Chris Acott
Hyperbaric Medicine Unit,
Royal Adelaide Hospital,
North Terrace, Adelaide, South Australia 5000
Telephone +61-8-8222-5116
Fax +61-8-8232-4207.
E-mail guyw@surf.net.au

Intending speakers are reminded that it is SPUMS policy that speakers at the ASM must provide the printed text of their paper and the paper on disc to the Convener before their presentation .

The Official Travel and Acommodation Booking Agent for the meeting is:

Allways Dive Expeditions
168 High Street
Ashburton,
Victoria 3147
Australia

Telephone 03-9885-8863
Toll Free 1-800-338-239
Fax 03-9885-1164.

MINUTES OF THE ANNUAL GENERAL MEETING OF SPUMS

held at the
Quality Resort, Waitangi, New Zealand
on 19/4/97
at 1245 local time

Apologies

Drs D Davies and T Wong.

Present

All members attending the Annual Scientific Meeting.

1 Minutes of the previous meeting

These had been published (SPUMS J 1996; 26 (3):161-166).

Motion that the minutes be taken as read and are an accurate record. Moved Dr J Knight, seconded Dr C Acott. Carried.

2 Matter arising from the minutes

None

3 President's report

Printed on page**s 11 and 12.**

4 Treasurer's report

Auditor's report and Statement of Receipts and Payments printed on page 13.

Motion that the financial statements be accepted. Proposed Dr V Haller, seconded Dr J Knight. Carried.

5 Annual subscriptions

Motion that the subscriptions remain the same (Full and corporate membership $100 and associate membership $50). Proposed Dr C Acott, seconded Dr V Haller. Carried.

6 Election of Office Bearers

A postal ballot of financial members had been held to elect the three committee members. The results were declared at the meeting. All other positions had only one nomination.

The Committee for 1997-1998 is

President	Dr Guy Williams
Secretary	Dr Cathy Meehan
Treasurer	Dr Timujin Wong
Editor	Dr John Knight
Public Officer	Dr Guy Williams
Education Officer	Dr David Davies
Committee Members	Dr Chris Acott
	Dr Vanessa Haller
	Dr Robyn Walker
New Zealand Representative	Dr Michal Kluger

7 Appointment of Auditor

Motion that Mr Douglas Porter FCA of Newport Beach, New South Wales, be reappointed as auditor.
Proposed Dr G Williams, seconded Dr J Knight. Carried.

8 Business of which notice has been given

8.1 That Dr David Elliott be elected a Life Member
That Rule 3 Life Members, (b) be altered by replacing the word *five* in the last sentence by the word *eight*.
The new sentence would read: *The number of life members shall at no time exceed eight nor shall more than one such member be elected in the one financial year.*
Proposed Dr G Williams, seconded Dr J Knight. Carried

8.2 That Rule 8 Annual General Meeting, (e) be altered by removing the words *of which notice has been given*.
The new rule would read: *The annual general meeting may transact special business in accordance with these rules*.
That Rule 11 Order of business at general meetings, (a) be altered by adding a new sub-section *(x) Any other business*.
That Rule 12 Notice of meetings, (b) be altered by replacing the second *the* by *a special meeting*.
The new rule would read: *No business other than that set out in the notice convening a special meeting shall be transacted at the meeting*.
Proposed Dr J Marwood, seconded Dr J Knight. Not carried

8.3 That Rule 23. (a) (ii) be changed by replacing *28* by *56*.
The new wording would be: *Shall be received by the Secretary of the Association not less than 56 days prior to the date of the annual general meeting*.
Proposed Dr G Williams, seconded Dr J Knight. Carried.

Closed 1315

PRESIDENTS REPORT 1997

I am pleased to report on my initial 12 months as President of the Society. This period has been relatively uneventful, compared with some of our recent years.

SPUMS's first Annual Scientific Meeting (ASM) in New Zealand has been a great success, with an excellent attendance, with SPUMS members for the first time experiencing temperate water diving. Those who said

SPUMS members would not dive with wetsuits have been proved incorrect. The Diving program this year has been very popular. I would like to thank Mike Davis and his assistant, Des Gorman, for their hard work, which has resulted in this year's meeting being such a success. Until one has convened a SPUMS Annual Scientific Meeting, one has no concept of the hard work and time devoted to each meeting. I would also like to thank our guest speakers, James Francis and Richard Moon, for attending this year's meeting and for their excellent presentations. Also, I thank all of the other speakers for their participation.

1997 is the first year of the new system for convening Annual Scientific Meetings (ASMs) where the convener of each meeting will take responsibility for organising the meeting, reporting to the SPUMS Committee and seeking assistance from others as required.

In 1998 Chris Acott will be convening the ASM in Palau, Micronesia, which will run from May 8th to 17th. The theme is "The History of Diving and Diving Medicine" with a workshop on The Ageing Diver. Guest Speakers will be David Elliott and John Bevan. Full details of the 1998 ASM will be circulated shortly in the SPUMS Journal, but early bird bookings may be made immediately with Allways Travel.

I would like to give notice that at next year's Annual General Meeting I will be proposing Dr Tony Slark for Life Membership of the Society in acknowledgment of his long and excellent service to the Society and Diving Medicine.

During the next twelve months I would like to see growth in membership of our Society, particularly in America and Europe. SPUMS has a representative in the USA in Terry Brown and in Europe with Henrik Straunstrup from Denmark. With further expansion of our membership it will be possible to improve our service to members and diving medicine.

The popular format that has been adopted at our ASMs, combining a scientific program and workshops with a diving program will continue in Palau in 1998. I believe our credibility in the recreational diving community is enhanced by the large number of our members who are active recreational divers, even if it is only once a year at the ASM.

I thank the sponsors of this year's ASM, the Accident Rehabilitation and Compensation Insurance Commission, PADI Worldwide, Northpower NZ and Patterson Distributors NZ. Also our conference organisers, Fullers Northland, for their hard work and The Quality Inn for caring for our delegates. I also thank the other members of the SPUMS Committee for their help and assistance over the last twelve months. Finally, I hope to see many of you in Palau in 1998.

Guy Williams

CONSTITUTIONAL AMENDMENTS

There have been no objections received by The Secretary of SPUMS to the motions amending the constitution. It is therefore assumed that the membership has approved the motions and the Rules of the Society have been amended.

Rule 3 Life Members, (b) now is: *The number of life members shall at no time exceed eight nor shall more than one such member be elected in the one financial year.*

Rule 23. (a) (ii) now is: *Shall be received by the Secretary of the Association not less than 56 days prior to the date of the annual general meeting*.

Cathy Meehan
Secretary of SPUMS.

Key Words
Consitutional amendment, notice.

1998 ANNUAL GENERAL MEETING

The 1998 Annual General Meeting will be held at the Palau Pacific Resort on Friday 15/5/98. The agenda will be as laid down in Rule 11 (a). The following motions have been submitted for discussion, as *Any business of which notice has been given*, at the 1998 AGM.

1 That Dr Tony Slark be elected a Life Member of the Society.

2 That Rule 22 (b) be altered to increase the period of election to the Committee to three years. The actual wording of the amendment appears below under Consititutional Amendment.

Cathy Meehan
Secretary of SPUMS.

Key Words
Meeting, notice.

CONSTITUTIONAL AMENDMENT

The Committee will be moving the following motion at the 1998 Annual General Meeting.

That Rule 22 (b), which reads "Each officer of the Association shall hold office until the annual general meeting next after the date of that person's election but is eligible for re-election.', shall be changed by replacing the word *next* by the words *three years*.

The new wording would be: *Each officer of the Association shall hold office until the annual general meeting three years after the date of that person's election but is eligible for re-election*.

Cathy Meehan
Secretary of SPUMS.

Key Words
Consitutional amendment, notice.

AUDIT REPORT
TO THE MEMBERS OF THE SOUTH PACIFIC UNDERWATER MEDICINE SOCIETY

I have conducted various tests and checks as I believe are necessary considering the size and nature of the Society and having so examined the books and records of the South Pacific Underwater Medicine Society for the year ended 31 December 1996 report that the accompanying Statement of Receipts and Payments have been properly drawn up from the records of the Society and gives a true and fair view of the financial activities for the period then ended.

15/7/97 David S Porter
361 Barrenjoey Road, Newport Beach, New South Wales 2106. Chartered Accountant

THE SOUTH PACIFIC UNDERWATER MEDICINE SOCIETY

STATEMENT OF RECEIPTS AND PAYMENTS FOR THE YEAR ENDED 31 DECEMBER 1996

			1996		1995
Opening Balance					
ANZ Bank	1995 ASM Fiji	11,090		-	
	Access Accounts	(3,722)		(364)	
	ANZ V2 Plus	92,320		68,053	
			99,688		67,689
Receipts					
1995 ASM Fiji Accounts		-		11,090	
Subscriptions and registrations		161,430		76,061	
Interest		5,707		4,956	
Advertising and Journal sales		1,201		979	
Sundry income		1,455		168	
			169,793		93,254
			269,481		**160,943**
Payments					
ASM costs		20,393		1,583	
Travel		22,779		-	
Secretarial		10,865		5,573	
Stationery and Printing		5,183		985	
Journal		36,792		22,199	
Postage and facsimile		6,074		3,552	
Conferences and telephone		9,324		6,608	
Computer equipment		36,300		3,405	
Miscellaneous and subscriptions		952		1,559	
Bank Charges	2,604		1,273		
Audit		1,080		540	
Insurance		799		-	
Editor’s honorarium		16,250		13,978	
			169,395		61,255
Closing balances					
ANZ Bank	1995 ASM Fiji	-		11,090	
	Access accounts	32,041		(3,722)	
	ANZ V2 Plus	68,045		92,320	
			100,086		99,688
			269,481		**160,943**

These are the accounts referred to in the report of D S Porter, Chartered Accountant, Newport Beach, NSW 2106, dated 15/7/97

LETTERS TO THE EDITOR

PUBLICATION OF SPUMS POLICIES

Hyperbaric Medicine Unit
Fremantle Hospital
PO Box 480 Fremantle
Western Australia 6160

13 January 1998

Dear Editor

I write to express some concern about an aspect of the current editing of the SPUMS Journal.

Under "SPUMS Notices" there is a segment entitled *The SPUMS Policy on Initial Management of Diving Injuries and Illnesses.* There are five authors and this appears merely to be an article.

In the first segment under "An Introduction to SPUMS Policies" there is what appears to be an attempted explanation of the rationale for society policies. However, it would seem that these "have been the product of individuals or small working groups", also workshops, and in future, the newly formed ExPresidents' Committee will be asked to develop some "Society Policies".

As a long-time member of this Society, I believe that a "Policy" is a document which should:

1 reflect best practice and
2 represent the views of the Society, or of its representatives.

It follows that nothing should be published as a Society Policy unless it has been presented, at the very least, to a formal meeting of the Executive Committee and ratified by them, as representatives of the members, as representing the views of the Society.

The views of individuals, however worthy, knowledgeable, and prominent in the society, should not, in my view, be labelled as policies of SPUMS without going through this process.

I hasten to add that I have little problem with the content on a personal note, but I believe that to push out individuals' views as the Society's Policies without any form of endorsement by the Society or its elected representatives reduces enormously the validity of the so-called Policies.

I make these remarks as constructive. If the Society is seen to allow any member, ex-member or friend of the in-group to write a piece, and then publish it as Society Policy, the Society will inevitably lose credibility.

I hope you will accept these comments as constructive, from a long-time member and occasional critic!

Harry F Oxer
Director

Key Words
Letters, policies.

Editor's Comment

In fact the Committee did consider the document and decided that it should be published at its meeting in Adelaide on November 1st 1997. However as the next meeting of the Committee has not yet occurred the Minutes of that meeting are not being published in this issue of the Journal. Dr Oxer can rest assured that the Committee has acted on behalf of the membership in the way that he desires.

A DIVER SUPPORT NETWORK

13 Rockwall Place
West Pennant Hills
New South Wales 2125
12/1/98

Dear Editor

Some time ago I had a diving accident overseas, which resulted in neurological decompression illness (DCI) which was successfully treated before I returned to Australia.

After my return I found it difficult to access a co-ordinated means of contacting medical professionals and therapists who were divers and who I felt would be more able to understand the nature of the post-traumatic stress that I was experiencing. I would also have liked an opportunity to discuss my accident with a sympathetic diver who had experienced a similar incident.

I wish to bring to your readers' attention the idea of a Diver Support Network which would offer a range of easily accessible services such as:

1 A means of putting an injured diver in touch with a sympathetic diver who has shared a similar experience. This would enable the diver to talk through the incident and help make re-entry to diving that much easier.
2 Provide a list of doctors and therapists who are divers and therefore more able to understand the nature of the diving environment and any on going problems the diver may be having. These would include anxiety disorders or post-traumatic stress.

To provide such a Diver Support Network there is a need for volunteers. The staff at the Hyperbaric Medicine Unit at the Prince of Wales Hospital, Randwick, who have helped me a lot, support the idea of a Diver Support Network. Both John Kershler and Greg Melbourne have agreed to act as contact person for the Network. Please phone them on 02-9382-3881 (0730-1630 Monday to Friday).

We need

1 Divers who have had a diving accident and are willing to share their experiences to help another diver.
2 Divers who have had a diving accident and need someone to share the experience with.
3 Doctors and therapists who are divers and are willing to provide professional assistance to divers after a diving accident.

If you would like to be part of this network please phone John Kershler or Greg Melbourne on 02-9382-3881 (0730-1630 Monday to Friday).

Tricia Johnson

Key Words
Accidents, letters, stress, trauma, treatment.

Department of Diving and Hyperbaric Medicine
The Prince of Wales Hospital
High Street
Randwick
New South Wales 2031
9/2/98

Dear Editor

I refer to Ms Tricia Johnson's letter of 12/1/98.

The establishment of a Diver Support Network, as outlined by Ms Johnson is being supported by the staff of the Hyperbaric Medicine Unit, Prince of Wales Hospital.

In the first instance we would like to hear from persons interested in being part of this initiative in support of divers who have suffered from decompression illness.

Depending on the response it is intended to compile a directory to be used by those who feel the need for some further consultation after the hyperbaric treatments have been completed.

The contact person is John Kershler, Hyperbaric Technician, phone 02-9382-3881 and fax 02-9382-3882.

John Kershler

Key Words
Accidents, letters, stress, trauma, treatment.

DIVING FOR THE DISABLED

Institute for Sports Science
University of Göttingen
Mühlenstraße 3 b
37073 Göttingen
Germany

Dear Editor

For the past two years we have examined the effects of snorkelling and scuba diving on people who have neurological diseases such as paraplegia, quadriplegia, hemiplegia, spina bifida, spasticity and traumatic amputation.

The purpose of our work is to establish snorkelling and scuba diving as new ways to help people rehabilitate themselves as, unfortunately, the rehabilitation system in Germany is being reduced.

At the beginning of the program each patient should develops his, or her, own sensibility for water and its characteristics. By using neoprene clothing and "ABC-equipment" (mask, snorkel, fins) these patients get the most possible independence.

The first breath under water is taken from equipment provided by the therapist. After few lessons the patients get in contact with the scuba diving utilities. Later on, the patients are taught to use their own equipment in a most independent way.

To evaluate the patients abilities and skills there are different exercises to manage: from cleaning the mask under water up to rescue a dive partner who simulates unconsciousness.

Snorkelling and scuba diving by the disabled has shown some effects which should be followed up in the coming years.

Social aspects

Scuba diving is a life-time sport which can be learned at any age.

Scuba diving offers the possibility of mixing with non-disabled people in an equal way.

Scuba diving is rehabilitation without the necessity of a medical centre or therapists.

Medical Aspects

Relaxation of muscles and reducing spasticity.

Relief of the weight support shoulder of wheelchair users.

Inhalation and exhalation training for the diaphragm and the rib muscles.

Increase of the lung's vital capacity.

Are there any similar works or research projects in the South Pacific ? We are very interested in starting international correspondence and co-operation! I can be contacted by telephoning +49-(0)551-45916, faxing +49-(0)551-3898013, or e-mail at MHELLWI@Stud.

BOOK REVIEWS

PROCEEDINGS OF REBREATHER FORUM 2.0
Editors M Menduno and K Shreeves
DSAT (Diving Science and Technology Corp.), 1251 East Dyer Road #100, Santa Ana, California 92705-5605, USA.
RRP $US 29.95.

Although the most common form of "technical diving" involves use of Enriched Air Nitrox breathed via conventional scuba equipment, there is much interest in rebreather technology with a number of rebreather models now becoming commercially available. Rebreathers have considerable attractions for serious technical divers, including extended duration of underwater breathing, conservation of expensive mixed gas, relative silence and, especially for photographers and scientists, lack of bubbles and buoyancy variations with breathing. The high price of rebreathers will severely limit this attractiveness but, for those with the money and enthusiasm to pursue this form of diving, objective evidence upon which to proceed is difficult to find.

The Proceedings of Rebreather Forum 2.0 can be highly recommended to any divers considering venturing into this complex form of diving. Held in California in September 1996, Rebreather Forum 2.0 brought together a range of experts and interested parties including manufacturers, the military, doctors, physiologists, representatives of instructor organisations and technical divers.

While many transcripts of workshops and conferences are difficult to read, this Proceedings has been well edited and is published to provide a comprehensive reference on the state of this "art." The Findings and Recommendations section provides three pages of considered and mostly cautionary advice to manufacturers and potential users. The edited transcript of the Forum and the appended Papers and Articles make it clear that this is very much an evolving field and, particularly for anaesthetists and others with a special interest in gas systems and physiology, the technical detail makes interesting reading. The natural market for this publication is clearly the participants in the forum, manufacturers and instructors, however diving doctors could contribute significantly to diving safety by insisting that any potential rebreather users obtain and read a copy of this publication. As the Findings state: "The military have been successful in managing the risks (of rebreathers) through use of a large supporting infrastructure, a high degree of discipline and training. Comparable infrastructure, discipline and training have not been needed in sport diving until now, and currently don't exist in the market."

DSAT, which is a subsidiary of PADI Worldwide, is to be congratulated on producing this book which so clearly warns of the problems associated with rebreather use and the need for a much higher standard of equipment care and training than is necessary for open circuit scuba using air.

Ian Millar

Key Words
Book review, equipment, physiology, rebreathing.

Dr Ian Millar is Head, Hyperbaric Service, Alfred Hospital, Commercial Road, Prahran (Melbourne), Victoria 3181, Australia.

DIVING MEDICINE FOR SCUBA DIVERS (2nd EDITION)
Carl Edmonds, Bart McKenzie and Robert Thomas
J.L.Publications, PO Box 381, Carnegie, Victoria 3163, Australia. Telephone/fax +61-3-9886-0200.
Price from Publisher $Aust 36.00.

That this book, aimed at the average scuba diver, has required a second edition within 5 years shows that there really are intelligent divers about who want to learn about what happens to our bodies when we go diving. Not only that but they are aware that diving can be dangerous and are seeking information to help them avoid the problems that those uneducated in diving medicine do not foresee. And this is the book they ought to buy. It is well laid out, easy to read, survives handling well, simply but accurately written and is value for money.

This book had a laudatory review when it first appeared (SPUMS J 1992; 22 (4): 212). As much of the book is unaltered those comments still apply. It can safely be recommended to divers who want to learn about diving medicine as it almost free of medical jargon, and the medical jargon that has crept in has been explained at its

first appearance. As those who know the authors would expect opinions are clearly and forcefully expressed.

The major change in the book is the inclusion of a chapter on Technical Diving. The authors and publisher were kind enough to supply portions of this chapter to the SPUMS Journal. These appeared in September 1997 (27 (3): 169-175. This chapter, and its reading list, should be compulsory reading for any diver contemplating starting Technical Diving. It would also help those who have started to use these techniques..

John Knight

Key Words

Book review, diving medicine, technical diving.

UNITED STATES NAVY DIVING MANUAL

Complete set (Volumes 1 and 2 with binders)

Best Publishing Company, P.O.Box 30100, Flagstaff, Arizona 86003-0100, U.S.A.

Price from the publishers $US 89.00. Postage and packing extra. Credit card orders may be placed by phone on +1-520-527-1055 or faxed to +1-520-526-0370.

Another review of the United States Navy (USN) Diving manual might seem unnecessary because the Vol 1 now reviewed is the same edition as was reviewed in 1995 (*SPUMS J* 25 (2): 74) with changes, rather than a new edition. The semantics of when a series of changes becomes a new edition is a Pentagon secret. It is disappointing to note that the changes are accompanied by a number of new typographical errors.

At 2.3 kg, the set qualifies as a weighty tome but it is debatable if most SPUMS members should purchase it. While it has a good medical section, it is a diving manual, intended for divers. Many SPUMS members would find the medical section less detailed than they would like. In some ways the manual advocates procedures that differ from current local practice. For example in treating a patient who did not have a satisfactory response to compression on oxygen to 18 m most local hyperbaric centres would go to 30 m on a 50% oxygen mixture, rather than to 50 m on air as suggested in the USN flow charts. These gas mixtures can be used in the USN, under instruction from a Diving Medical Officer.

The errors in the total decompression times in the air tables, noted in my previous review, have been corrected. The total ascent times now reflect the change from an ascent rate of 60 feet/min (18 m/min)to 30 feet/min (9 m/min) and the air tables are provided with metric equivalents. My other main criticism in 1995 was the lack of an index; this has now been added.

I think a copy of Vol 1 should be available at all treatment chambers for immediate access to the finer points of table usage. For example what do you do if the oxygen supply fails during a treatment? When should you administer oxygen to an attendant who is doing an extended Table 6 as a repetitive dive? The USN Manual tells you. It is also a very good yardstick against which to evaluate your procedures. Either doing it their way, or by developing an argument for doing it your way, provides you with a useful quality test.

Because of the orientation toward the needs of a diver I consider that the manual should be reference reading for diving instructors who are trying to provide high quality tuition.

Vol 2 is devoted to mixed gas and oxygen diving. It should be noted that it does not discuss the topics of open circuit Nitrox or trimix diving as these techniques are not used by the USN. Vol 2 is of interest to people who are involved with deep and mixture diving. Some SPUMS members would find it useful in advising patients who are contemplating deep dives. The check sheets are a useful indication of good practice in deep diving and could be used as a benchmark for other operations.

The main change in Vol 2 is that using the surface supplied Helium-Oxygen decompression table has been simplified. One now enters the table at the required depth and is given an range of acceptable oxygen concentrations, and a series of decompression schedules, for bottom times up to 120 minutes. Previously the first step was to calculate the helium partial pressure and then enter a table of helium pressures. The other change in this table is that for most dives deeper than 200 feet, the decompression stops from 100 to 60 feet (30 to18 m) are now made breathing 40% Oxygen/60% Helium. The tables still require oxygen stops at 50 and 40 feet (15 and 12 m). These stops expose the diver to 2.4 ATA of oxygen when on the mix at 100 feet (30 m) and 2.5 ATA when on oxygen at 50 feet (15 m). This is acceptable to the USN as the helmet used gives the diver protection if he suffers an oxygen convulsion. Divers with less adequate precautions against the consequences of an oxygen convulsion might prefer to use the DCIEM Helium/Oxygen tables which use oxygen at 9 m (30 feet) and air for any deeper stops.

Readers of the manual might note that dove is the American past tense of dive, not a small white bird.

John Pennefather

Key Words

Book review, decompression illness, flow chart, diving operations, treatment, underwater medicine, occupational diving.

John Pennefather is the Scientific Officer at the RAN Submarine and Underwater Medicine Unit.

SPUMS ANNUAL SCIENTIFIC MEETING 1997

ASSESSMENT OF PATIENTS WITH DECOMPRESSION ILLNESS

Richard Moon

Key Words
Accidents, decompression illness, investigations, treatment.

History

The diagnosis of decompression illness (DCI) is made clinically and should be based entirely upon an accurate history and physical examination. Required for the diagnosis are the appropriate circumstances for the condition to occur and signs and symptoms which are consistent with the disease. The patient must have been exposed to a reduction in ambient pressure. In order to experience pulmonary barotrauma and gas embolism a breathhold ascent from 1 metre may be sufficient.[1] For bubbles to form in the body from supersaturated tissues (decompression sickness, DCS or bends) a diver must have been at depth long enough for supersaturation to occur. It was formerly believed, incorrectly, that the disease would never occur in divers whose maximum depth was less than 10 m (33 ft). Symptoms of DCS have been described after a series of breath hold dives.

The history should include the dive profile, rate of ascent, symptom onset time and changes in symptom type or intensity. As an approximate gauge of a diver's inert gas load a dive table can be consulted. Factors which increase the likelihood of bends include missed decompression stops, heavy exertion during the dive, rapid ascent and a previous history of DCI. A series of dives may incrementally augment the inert gas load. A diver's self-reported profile is often inaccurate. If a dive computer was used, some models will permit downloading an objective record of the profile. Unfortunately it is a common belief among physicians not trained in diving medicine is that divers who have stayed within the limits of a computer or table profile will not suffer DCI. This is not true and such physicians must be educated out of their dangerous ways.

For recreational divers reporting DCI of all types to DAN in 1995 the median time of onset was 1 hour, if there had been no altitude exposure. Within 24 hours 95% of all symptoms had become evident.[2] Table 1 has been compiled from the Diver Alert Network (DAN) statistics for 1995 and shows the frequency of presenting symptoms (the first symptom) and of all symptoms, in 590 recreational diving accidents reported to DAN, tabulated by symptoms. There were over three times as many total symptoms as first symptoms, emphasising that many cases have multiple symptoms. The most severe cases presented shortly after surfacing, as reported by Francis and colleagues.[3] They observed that within 10 minutes of surfacing 50% of their divers had developed symptoms and 85% had done so within an hour. Ninety six per cent of those developing cerebral symptoms had them within 60 minutes. Therefore the longer the delay between surfacing and the onset of a symptom, the less likely it is due to DCI. Symptoms beginning after 24 hours following a scuba dive are unlikely to be caused by DCI.

Exposure to even moderate altitudes can precipitate DCI. The most commonly reported altitude exposure is in commercial aircraft (typical cabin altitude 1,500-2,400 m). After a scuba dive the majority of altitude-precipitated bends occur when the flight is within 24 hours of surfacing from a dive.[4] After a saturation dive symptoms may occur during an altitude exposure days after surfacing.[5]

Physical examination

General physical examination should include measurement of pulse and blood pressure and a search for evidence of pulmonary barotrauma (pneumothorax, pneumomediastinum, subcutaneous emphysema) and otic barotrauma (erythema or rupture of the tympanic membrane, blood or fluid in the middle ear). Rarely, in cases of arterial gas embolism, bubbles can be observed in the retinal vessels. In DCI a non-specific skin rash is occasionally seen. Lymphoedema may indicate obstruction of lymphatics by gas. A specific sign of DCI is a marbling of the skin occurring shortly after surfacing, indicating heterogeneous obstruction by bubbles of subdermal blood vessels.

The examination of a patient with pain only bends usually reveals no evidence of joint inflammation and there is rarely pain on movement. A "classic" physical sign is alleviation of pain when a sphygmomanometer is inflated around the affected joint.[6] Similarly, pain in the hips or legs may diminish when the patient stands up, presumably because the resulting increase in local tissue pressure compresses bubbles. These signs are not sufficiently sensitive or specific to exclude the diagnosis of DCI.

Physical examination in suspected DCI should always include a neurological examination. The patterns of abnormality observed in DCI are usually different from those typical of occlusion of major intracranial blood vessels due to intracranial haemorrhage or thromboembolic stroke. In DCI patchy areas of hypaesthesia, isolated urinary sphincter abnormality (usually urinary retention) and ataxia may be the only abnormalities, but these may be missed if only an abbreviated neurological examination is performed. Walking and performing tandem gait (heel-

TABLE 1

FREQUENCY OF SYMPTOMS OF DCI IN 590 RECREATIONAL DIVE ACCIDENTS IN 1995 (MANY WITH MULTIPLE SYMPTOMS SO TOTAL OCCURRRENCES EXCEED 590)

Symptom	Occurrence as a first symptom		Total Occurrences	
	Number	(Percentage)	Number	(Percentage)
Severe neurological				
Unconsciousness	4	0.7	15	2.5
Paralysis	3	0.5	22	3.7
Visual disturbance	5	0.8	39	6.6
Difficulty walking	2	0.3	55	9.3
Semiconsciousness	1	0.2	13	2.2
Bowel control problem	1	0.2	17	2.9
Speech disturbance	-	0.0	16	2.7
Bladder control problem	-	0.0	11	1.9
Convulsion	-	0.0	-	0.0
Total	**16**	**2.7**	**146**	**24.7**
Mild or ambiguous neurological				
Numbness	129	21.9	364	61.7
Dizziness	44	7.5	134	22.7
Decreased skin sensation	1	0.2	39	6.6
Personality change	1	0.2	14	2.4
Reflex change	1	0.2	15	2.5
Weakness	38	6.4	150	25.4
Total	**214**	**36.3**	**340**	**57.6**
Total neurological	**230**	**39.0**	**486**	**82.4**
Pain/skin/nonspecific				
Pain	203	34.4	341	57.8
Extreme fatigue	25	4.2	124	21.0
Headache	35	5.9	146	24.7
Nausea	25	4.2	87	14.7
Itching	21	3.6	53	9.0
Rash	5	0.8	25	4.2
Restlessness	6	1.0	38	6.4
Muscle twitch	5	0.8	33	5.6
Hemoptysis	1	0.2	6	1.0
Total pain/skin/non-specific	**326**	**55.2**	**104**	**17.6**
Ambiguous†				
Hearing loss	3	0.5	6	1.1
Ringing in ears	1	0.2	14	2.4
Cardiorespiratory				
Difficulty breathing	8	1.4	47	8.0
Other††	22	3.6	48	8.1
Total other presentations	**34**	**5.8**	**133**	**22.5**
Total of all recorded occurrences	**590**	**100.0**	**590**	**100.0**

† Could also be due to middle or inner ear barotrauma

†† Includes stiffness (2), hot/cold flushes (2), cramps (1), swelling, (5) pressure sensation (2), lightheaded/confusion (1), fullness (1), muscle ache/soreness (4), bleeding (1), coughing (1), ear blockage (1), erratic heartbeat (1).

to-toe walking) can often reveal abnormalities which are not otherwise apparent. Inability to perform tandem gait forwards and backwards with eyes open or closed on a hard floor while barefoot suggests a neurological abnormality. Inability to stand with eyes closed, arms folded on the chest and one foot in front of the other (sharpened Romberg sign) has been correlated with DCI.[7] A thorough sensory examination may reveal small areas of hypaesthesia. In the absence of a pre-existing problem with micturition, elevated residual bladder volume can be assessed by inserting a urethral catheter after the patient has attempted to void.

Many patients with paraesthesias as the only symptom have no other manifestation of the disease. Therefore absence of neurological signs cannot be used to exclude the diagnosis of DCI.

Diagnostic tests

The plain chest radiograph is the most available and least expensive of all of the various radiographic techniques which have been applied to DCI. Abnormalities on chest radiographs in DCI include pulmonary oedema in cardiorespiratory DCI ("chokes"),[8] focal opacities due to aspiration of water or vomitus, or pulmonary over distension.[9] Discovering these abnormalities rarely changes the management of the patient, however chest x-ray studies may still be useful. However, conditions which can predispose to pulmonary barotrauma, such as bullae,[10] and evidence of barotrauma such as subcutaneous or mediastinal gas may support the diagnosis of gas embolism if neurological symptoms have resolved. The existence of a significant, and increasing, pneumothorax may dictate the insertion of a chest drainage tube before recompression treatment.

Abnormalities have been reported using neuro-imaging techniques such as computed tomography (CT),[11-13] magnetic resonance imaging (MRI)[14,15] single photon emission tomography (SPECT)[16-18] and positron emission tomography (PET).[19] However, their value in the management of DCI has not been demonstrated, except to rule out unrelated conditions such as haemorrhage which require different therapy. Occasionally chest CT can detect predisposing factors to DCI which are not easily detected with plain chest radiography.[10,20]

Abnormal neurophysiological tests such as EEG,[12] brainstem auditory evoked response (BAER) and somatosensory evoked responses (SSEP)[15] have been described in DCI. While they can occasionally be useful in assessing the patient when clinical evaluation is unsatisfactory, they are not sufficiently sensitive to be used routinely. Because audiograms and electronystagmography (ENG)are more accurate than clinical examination, they are extremely useful in following the course of inner ear DCI or barotrauma.[21] Clinical testing of hearing loss by non-specialists, without access to a series of tuning forks with a range of frequencies, is likely to be inaccurate. The nystagmus produced by vestibular injury is characteristically inhibited by visual fixation, but can easily be tracked and quantified with the eyelids closed by ENG.

There are no specific blood markers of DCI, however elevation of serum creatine phosphokinase (CPK) has been described in air embolism,[22] presumably due to muscle injury. Haemoconcentration has been described in both forms of DCI,[23,24] presumably due to endothelial damage and the extravasation of plasma into tissues ("third space" loss). If contaminated breathing gas is suspected because of symptoms or signs of carbon monoxide poisoning, measurement of a blood carboxyhaemoglobin level may confirm the diagnosis. Appropriate blood or urine assays can be used to detect other metabolic causes of encephalopathy such as hypoglycaemia and drug or alcohol intoxication.

Cases of DCI exist in which the only manifestation appears to be a mild encephalopathy observed by the diver, who realises that he is unable to perform a routine, familiar task. Neuropsychological tests have been used to demonstrate abnormalities in DCI which are not apparent after history and physical examination.[25] If trained personnel are available to administer short neuropsychological tests, they could also be useful in following the course of treatment. Additional investigation is needed to determine the appropriate tests to use and their role in the management of DCI.[26]

Differential diagnosis

Onset of pain, rash, dyspnoea or a neurological abnormality after a dive is usually (correctly) assumed to be due to DCI. However, unrelated disease processes may coincidentally become evident shortly after a dive and can therefore complicate the diagnosis. Severe symptoms which begin after more than six hours following decompression without altitude exposure and any symptom occurring more than 24 hours after surfacing should raise the suspicion of an alternative diagnosis. A diagnosis of DCI should also be re-evaluated in a diver who fails to improve despite prompt recompression treatment. Table 2 shows several diagnoses which may be confused with DCI.[27]

Conclusions

The treatment of decompression illness using appropriate first aid measures and recompression treatment should be based upon clinical evaluation. To date, with the exception of inner ear damage (due to barotrauma or decompression injury), in which repeated audiograms and electronystagmograms are the most accurate means of

TABLE 2

CONDITIONS WHICH CAN MIMIC DCI
(Modified from Moon[27]).

Contaminated breathing gas (carbon monoxide)

Headache, nausea, vomiting, impaired consciousness. The diagnosis is suggested if several individuals who obtained their breathing gas from the same source are similarly affected. Diagnosis made by measurement of blood carboxyhemoglobin level or carbon monoxide in breathing gas.

Near drowning and hypoxic brain injury

Impaired consciousness, confusion. May be impossible to differentiate from arterial gas embolism.

Seafood toxin poisoning

Ingestion of large reef fish (Ciguatera),[28-30] Puffer fish (paralysis)[29] or shellfish (paralysis).[29,31] May present with focal neurological signs after ingestion of fish. Nausea, vomiting and abdominal pain frequently precede the onset of neurological symptoms in ciguatera poisoning. Diagnosis suggested if meal companions similarly afflicted.

Envenomation

Cone shell (paralysis),[29] Sea snake[29] or "Sea stroke".[32] "Sea stroke" is respiratory failure after presumed envenomation while swimming in the ocean on the east coast of the USA.

Migraine[33,34]

Focal neurological deficit preceding headache. Diagnosis suggested by a history of migraine headaches.

Guillain-Barré syndrome

Progressive neuromuscular weakness which can lead to respiratory failure. Progression is over several hours or days, unlike severe neurological DCI, which usually progresses over minutes.

Porphyria

Neuropathic pain, frequently in the abdomen, may be accompanied by neurological signs, including impaired consciousness and mental changes. Diagnosis suggested by history of similar attacks.

Sickle cell crisis

Acute onset of severe pain, usually in the limbs. Most common in black people. Diagnosis suggested by history of similar attacks. Microcytic anemia and hemoglobin S.

Multiple sclerosis

Onset of neurological symptoms/signs usually over hours or days. Acute, reversible abnormalities can sometimes be experienced by patients with a history of the disease, apparently triggered by stress such as a change in environmental temperature.

Transverse myelitis

Acute onset of back pain and signs of spinal cord dysfunction. Progression usually over hours, rather than minutes. Abnormal cord MRI.

Spinal cord or root compression

Due to disc protrusion, hematoma or tumor. May be difficult to differentiate from DCI. Poor response to recompression. Diagnosis confirmed by MRI.

Middle ear or sinus barotrauma with cranial nerve compression[35-41]

Isolated cranial nerve (V or VII) abnormality due to "reverse squeeze". Associated with sinus pain or difficulty clearing middle ear on ascent.

Inner ear barotrauma[21,35]

Symptoms of vertigo, hearing loss usually occur during compression, rather than after decompression. Usually a history of difficulty equalizing middle ear pressures and middle ear barotrauma evident.

Stroke

Ischemic[42] or hemorrhagic stroke. May be difficult to differentiate from DCI. Poor response to recompression. Diagnosis confirmed by MRI.

Subarachnoid haemorrhage

Suggested by nuchal rigidity, subhyaloid haemorrhages. Diagnosis confirmed by MRI, lumbar puncture.

Cold water immersion pulmonary edema[43]

Pink frothy sputum, dyspnea, usually occurring early in a cold water dive, before significant depth-time exposure. Chest radiograph demonstrates pulmonary edema. "Chokes" (cardiorespiratory DCI), caused by high levels of venous emboli, also causes pulmonary edema, but is not usually accompanied by expectoration of pink sputum, and requires significant depth-time exposure.

Unrelated seizure, post-ictal state (hypoglycemia, epilepsy)

Seizure in water during decompression or after exiting the water usually attributed to AGE until proven otherwise. Hypoglycemia severe enough to cause unconsciousness and seizure only likely to occur after administration of insulin or other blood glucose lowering medication, or insulinoma.

Functional abnormality[44]

Suggested by history of hysteria or conversion reactions, secondary gain (e.g. compensation for disability) with apparent deterioration days or weeks after good response to recompression.

assessment, there are no laboratory, radiographic or electrophysiological tests which are more sensitive that clinical examination for the diagnosis of decompression illness. A history and physical examination are usually sufficient to make the diagnosis and plan the treatment.

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MECHANISMS OF SPINAL CORD INJURY IN DCI

James Francis

Abstract

After more than a century of research into the mechanisms of spinal cord injury in decompression illness (DCI), there are reasons to believe that neither of the two principal mechanisms which have been proposed apply in all instances. Arterial gas bubble emboli are probably rare in non-lethal presentations and should preferentially injure highly perfused organs because they receive most bubbles. On the basis of blood flow, one would expect the grey matter of the cord to be the principal target, whereas it is in the white matter that the lesions of DCI are normally found. Venous infarction is not only a very rare cause of spinal cord dysfunction but the histology of massive, central infarction differs from the scattered, punctate haemorrhages which are classically seen in DCI. An alternative mechanism, the formation of extravascular bubbles within the substance of the cord, so-called autochthonous bubbles, received little attention until the late 1980s.

An established canine model of severe DCI was adapted to study the acute histology of the disease and to compare it with that found after other insults. Numerous extravascular, non-staining, space-occupying lesions, located principally in white matter, were found soon after the onset of DCI. It is likely that they were caused by the local evolution of gas bubbles. These lesions were not present in undived controls, in dived animals which did not develop DCI or in spinal cords rendered dysfunctional by global ischemia or gas emboli.

Analysis of the size and distribution of these lesions in 5 cords indicated that they were sufficiently numerous to account for the loss of function. Recently, further doubt has been cast on the role of venous infarction, but the role of embolic bubbles remains controversial. It is concluded that no one mechanism can be responsible for spinal cord injury in DCI.

Key Words

Bubbles, decompression illness, hyperbaric research, physiology.

Introduction

Decompression illness (DCI) of the nervous system has been recognised since the early descriptions of the condition by Bauer[1] and Clark.[2] Involvement of the spinal cord was described as having an onset of a few minutes and sometimes a few hours after leaving the caisson or water. This is still a common presentation.[3] Patients presented with numbness, weakness or paralysis of the lower limbs, accompanied, on occasion, by a constricting girdle pain. Frequently there were disturbances of bladder, bowel and sexual function. The upper limbs, although occasionally involved, were usually spared.[4] Blick, who saw over 200 cases of "diver's palsy" among the pearl divers of Broome (60 of them post mortem) early this century, noted urinary retention was such a common consequence of diving that none of the divers would consider his outfit complete without a soft catheter![5] Bert observed that although neurological DCI may recover spontaneously, "Too frequently the paralyses of the lower limbs are persistent.... In none of the cases which we reported was a paraplegia which lasted more than two days ever completely cured."[6]

An aspect of neurological DCI that became apparent during World War II was the different distribution of symptoms in aviators compared with divers and caisson workers. In the latter group the spinal cord was, and is, more frequently involved than the brain and the onset may be without warning.[3,7-19] In aviators the central nervous system (CNS) involvement in DCI was mostly *cerebral* involvement, occasionally associated with "chokes" (cardio-pulmonary DCI) or cardiovascular collapse.[20-27] Spinal cord involvement was rare and usually transient[28-30] although occasional cases have been reported in man[12,31,32] and experimental animals.[33]

An intriguing aspect of this condition is that, despite considerable research efforts over the last century or more, the mechanism by which decompression injures the spinal cord remains far from clear. It is this issue that will be addressed in this presentation.

Spinal cord pathology in acute DCI

In 1870, Bauer described the post mortem examination of a 35 year old caisson worker who had survived for 5 days following the onset of acute neurological DCI.[1] The principal findings in the spinal cord were: hypervascularity of the dura and arachnoid mater, an accumulation of cerebrospinal fluid, the thrombosis of a moderately sized vein near the cauda and some softening of the cord. Spinal cord softening in this and other cases that he observed occurred in "circumscribed portions of the columns". Similar observations plus "small clots of extravasated blood on the external surface of the dura" were made by Clark[2] when commenting on cases which had died in a similar time frame.

Van Rensselaer published a review of 25 post mortem examinations of cases of caisson disease.[34]. The gross findings were essentially the same, with the additional observation of secondary tract degeneration in a man who had survived for 36 days. In this paper was one of the first descriptions of the microscopic findings in the spinal cord. He stated that the white rather than the grey matter was affected and, at the late stage of the disease represented by

these cases, the condition was characterised by the destruction of nerve tissue, increased neuroglia and the loss or atrophy of axis cylinders. He considered this to be degenerative or resemble a diffuse parenchymous "myelitis", with the lower dorsal cord most often affected. He found no evidence of haemorrhage. Sharples made similar observations on a tetraplegic diver who had died 38 days after the onset of DCI. There were scattered lesions throughout the cord, with the greatest tissue destruction at the cervical level.[35]

Many of the early reports of the neuropathology of DCI described cases in which death had occurred many days after the onset of the condition. Brooks described two cases in which death occurred after 3 days and 13 hours respectively.[36] In the first case he described an abundance of cerebrospinal fluid (CSF), patchy softening of the cord and a single, small, 'H' shaped haemorrhage at the level of T8. Microscopically, there was oedema and numerous "lacerations" in the firm parts of the cord and the softened areas presented the familiar appearance of "transverse myelitis". The spinal cord of the second case appeared considerably less softened and, although the spinal canal contained large amounts of blood, only tiny areas of haemorrhage were found within the spinal cord, principally located in the dorsal columns. Oedema, numerous "air lacerations" and microscopic haemorrhages were demonstrated histologically in the white matter of the cord.

Blick performed post mortems on 60 cases of DCI.[5] At the time of examination many had been dead for some time and had begun to putrefy. He also described the frequent finding of blood or blood-stained fluid in the dural canal. He described the cross sections of these cords as:

"It looks as if one had stippled the face of the section with a fine knife or needle, a semi-disintegrated appearance. With this condition is nearly always associated haemorrhage of greater or lesser extent".

Nine of his cases had large haemorrhages which were:

"Practically cutting the cord in two and filling the meningeal tube for over one and a half inches".

Although frequently quoted, this is the only description of such haemorrhages in the literature. Since the early 20th century the above pathological findings in the spinal cord have not been challenged although three additional features have been described. Kitano and Hayashi described a case in which spinal cord congestion was associated with the coagulation of blood in the epidural veins. These veins contained numerous fat droplets that were thought to be of bone marrow origin.[37] They also found, in a diver that had died from acute neurological DCI shortly after surfacing, several small (up to 1 mm diameter), non-staining, round spaces in the white matter of the brain and spinal cord.[38] Similar spaces were described by Waller in the brains of two scuba divers who had died underwater.[39]

Evidence of fat and bone marrow emboli has been found in animals, particularly in the lungs, but not in cerebral or spinal cord vessels.[40] Bubble-like lesions have been described in the spinal cords as well as other organs of dogs[41] and mice[42] suffering fatal decompression illness and in decompressed fingerling salmon.[43] In 1986 Palmer described a thin rim of sub-pial white matter, in the spinal cords of goats with DCI, that was invariably spared.[44]

The pathophysiology of DCI.

By 1891 there were almost as many theories of the mechanism of DCI as there were observers of the condition. Many of these hypotheses were based on scant evidence and conceived in the absence of a clear understanding of physics or physiology. They were fully reviewed by Van Rensselaer[34] who classified them as follows:

1 The theory of exhaustion and cold.
2 The gaseous theory.
3 The theory of congestion with sequelae:
   a "Black blood" (i.e. blood deprived of its oxygen.)
   b Evolution of gas in the blood vessels.
   c Haemorrhage.
   d Acute revulsive anaemia.
   e Comparative stasis.

He dismissed the first theory on the grounds that, in winter, many of the population are exposed to greater and more prolonged cold than caisson workers during their decompression, yet they do not display the signs of caisson disease.

The second theory has its origins in the observations of Robert Boyle[45] who decompressed numerous animals in his "exhausted receiver". One of Boyle's remarkable conclusions deserves quoting in full:

"Whether, and how far the destructive operation of our engin upon the included animal, might be imputed to this, that upon the withdrawing of the Air, besides the removal of what the Airs presences contributes to life, the little bubbles generated upon the absence of the air in the Bloud, juyces, and soft parts of the body, may by their vast number, and their conspiring distension, variously streighten in some places, and stretch in others, the vessels, especially the smaller ones, that convey the Bloud and Nourishment; and so by choaking up some passages, and vitiating the figure of others, disturb or hinder the due circulation of the Bloud? Not to mention the pains that such distensions may cause in some nerves, and membranous parts, which by irritating some of them in convulsions may

hasten the death of the animals, and destroy them sooner by occasion of that irritation, than they would be destroyed by the bare absence or loss of what air is necessary to supply them with."

Paul Bert,[6] in 1878, wrote, after a large series of experiments, mostly on dogs, that:

"Sudden decompression, beginning with several atmospheres, brings on symptoms of varying severity depending upon the degree of compression, the speed of decompression, the animal species, the individuals, and the state of the experimental animal at the time. These symptoms must be attributed to the escape of nitrogen which had been stored up in excess in the organism, following Dalton's law. This gas changes to a free state in the blood vessels, the different organic liquids, and even the interior of the tissues; it may therefore, according to circumstances, check the pulmonary circulation, soften and cause anaemia in certain regions of the nervous centres and especially the lumbar enlargement of the spinal cord, lacerate the tissues, and produce swellings or a more extensive emphysema. The severity of the symptoms depends upon both the seat and the extent of these multiple disorders."

Van Rensselaer criticised Bert's conclusions, saying that findings based on animal experiments, performed at higher pressures than those used in caissons, could not be extrapolated to man.[34] He adhered to the third theory. Although these various congestive mechanisms, if they were considered to be primary rather than reactive, appeared to be supported by the pathological findings, their mechanisms depended upon the erroneous belief that atmospheric pressure is unevenly distributed throughout the body. Not surprisingly Bert's conclusions have formed the basis for one theory of the pathogenesis of DCI involving the spinal cord.

The arterial bubble embolus hypothesis.

In 1903 Hill and Macleod observed the circulation in the vessels of a bat's wing and frog's web during and following decompression.[46] They noticed:

"For about a minute after rapid decompression the circulation continued unaltered, then small, dark bubbles were seen, first one, and then another, and then numbers scurrying through the vessels, and driving the corpuscles before them. In a moment or two the vessels became entirely occupied with columns of air bubbles, and the circulation was at an end." In 1906 Oliver confirmed these results.[47].

Apart from making considerable advances in the design of safe, yet efficient decompression procedures, Boycott, Damant and Haldane explored the possible pathogenic mechanisms of DCI.[48] They eventually selected goats as their experimental animals because they considered that they would have comparable decompression obligations with men. They studied goats which died and some that were killed at varying intervals after decompression. They noted that the presence of bubbles post mortem did not necessarily mean that bubbles had been present in vivo, because they may have formed after death. Their observations pertinent to the spinal cord were:

First, the presence of bubbles in venous blood correlated poorly with symptoms of DCI. Bubbles were found in asymptomatic animals and no bubbles were found in animals in which symptoms would have been expected had they not been killed. Arterial bubbles were occasionally seen, especially in animals that had died slowly.

Secondly, veins contained variable quantities of bubbles, but always more than arteries. Interestingly, they observed few bubbles in the veins of the brain and spinal cord.

Thirdly, they found that, as in human cases, areas of the spinal cord might be softened. The distribution of these areas of softening were most marked in, and usually confined to, the lower dorsal and upper lumbar segments and affected only white matter.

Based on these and other observations, they concluded that:

"The distribution of small bubbles in the arterial stream must be universal. They probably lodge in many places: while they are rapidly pushed forward in the grey matter and in most other tissues, if they lodge among the fatty surroundings of the capillaries of the white matter, or in actual fat, they quickly increase in size to such an extent that their removal becomes impossible.... The cause then of these areas of softening is not ordinary embolism, but embolism which becomes effective to produce infarction by reason of the effect on the size of the embolus of the local conditions of the circulation rather than from any of those peculiarities in the resistance of the different tissues to lack of oxygen, or in the freedom of collateral circulation, which determine the topography of common infarcts."

Since 1908, some experimental work appearing to support the gas embolism mechanism has appeared.[49-52] Other investigators have also observed arterial bubbles in decompressed animals.[53-55]. However, in these studies, arterial bubbles were only seen following their appearance in veins and were associated with severe DCI which was often fatal. These authors concluded that arterial bubbles were rare in less serious forms of DCI.

In none of these studies was the nucleation of gas bubbles observed directly and many of them were performed upon small rodent species that were subjected to near explosive decompression insults in order to generate an injury. As Hills reasoned,[56] it is during rapid

decompression that blood may have time to supersaturate and bubbles to nucleate between leaving the lungs and r eaching the tissues. This could result in the appearance of arterial bubbles that may be absent in the less rapid, yet spinal cord-damaging, decompressions undertaken by man.

The pathological findings in the spinal cord have been described as being compatible with ischaemic necrosis.[57,58] This has been used to support the arterial gas emboli theory.[59,60]

The development of the Doppler ultrasonic probe has resulted in a mass of evidence that intravascular bubbles are associated with DCI in both animals[55,61-65] and man.[66-73] The evidence is that bubbles first appear on the venous side of the circulation and that arterial bubbles are rare and only associated with severe disease.

A further problem with the arterial bubble embolus theory relates to the origin of arterial bubbles. It is widely recognised that, after passage through the lungs, arterial and alveolar gas tensions have equilibrated. Hills calculated that, during an ascent of less than about 6 m/min (20 fsw/min), arterial blood would not reach inert gas saturation in the time taken to travel between the lungs and tissues.[56] He noted that it is difficult to form bubbles in blood even with appreciable degrees of supersaturation and thus the formation of bubbles in arterial blood during decompression at conventional rates is most unlikely.

It is surely pertinent that arterial gas embolism from pulmonary barotrauma invariably presents with cerebral rather than spinal symptoms. DCI is common and gas embolism is rare in caisson workers and saturation divers who generally experience a controlled decompression.

It has been known for some time that arterial bubbles may arise from the paradoxical embolism of gaseous, venous emboli.[74,75] Post mortem a detectable foramen ovale (PFO) is found in about 35% of cases.[76] When accompanied by a Valsalva manoeuvre, a shunt was detectable by contrast echocardiography in 18% of adults. At rest, this figure dropped to 5%.[77] The effect of the Valsalva is important since, in the event of significant intravenous bubbling, the pressure in the right side of the heart is increased,[78- 80] which may provoke shunting in a similar manner to a Valsalva manoeuvre. Additional factors which may increase right-sided blood pressure and thereby increase right-to-left shunting in divers are: immersion, cold and negative-pressure breathing.

It has been shown that divers with a history of DCI have a higher prevalence of PFO than divers without DCI or non-diving controls.[81,82]. There has been recent interest in MRI findings in the brains of divers. One study of sports divers showed an increased prevalence of hyperintense “lesions” in divers compared with non-diving controls[83] and a more recent study has found that, in sports divers, these “lesions” are associated with a PFO.[84] None of the divers studied had had an episode of overt DCI. This has been used to imply that divers with a PFO are at a greater risk of DCI than those without.[85]. Unfortunately, the prospective trials which are required to test this hypothesis have yet to be undertaken. So far there is no evidence that a PFO is associated with lesions in the spinal cord. The argument for an embolic injury mechanism in the cord is less compelling than for the brain because the evidence is that both solid and gaseous emboli invariably embolise the latter.[86-89]

Wilmshurst has reiterated the hypothesis first proposed by Boycott et al.[48] that bubbles which embolise spinal white matter grow and obstruct the circulation whereas those which embolise the brain (or, presumably, the spinal cord grey matter) do not because of the relatively rapid washout of gas in these structures.[90] While this may occur there is, as yet, no experimental evidence to support it. The theory also does not explain the latent interval between decompression and the appearance of venous bubbles. It is difficult to reconcile this with very short-latency disease. Equally, with the spinal cord white matter having an estimated time constant of about 9 minutes,[91] this mechanism is unlikely to account for longer latency disease because the cord will have washed out its surplus inert gas by the time it is embolised. This does not mean that embolic bubbles arriving then will cause no spinal cord dysfunction, since bubble emboli are capable of disrupting the function of cords with ambient inert gas tensions.[92] It is therefore unnecessary to invoke bubble growth as a mechanism for spinal dysfunction.

Bubbles can appear in arterial blood if venous bubbles traverse the pulmonary filter. This may not occur with much frequency as the lungs have been shown to be a most efficient filter of beads[93] and gas emboli[94-98] in the size range of bubbles measured in the venous blood of decompressed dogs.[99] In the presence of massive intravascular bubbling the filtering capacity of the lungs may be exceeded.[80,96] However, this process is time-consuming[100] and accompanied by pulmonary symptoms. Thus this mechanism is unlikely to be relevant to cases of DCI where the onset occurs either during or shortly after decompression. As with other embolic mechanisms, there should be simultaneous clinical or subclinical cerebral emboli.

There is a question whether an embolic-ischaemic mechanism compatible with the pathological appearance of spinal cord DCI. There is evidence that it is the grey rather than the white matter is preferentially injured by both ischaemia[101] and emboli[92,102] although, in acute disease, the changes are subtle. The histology of spinal cords in which there was long-latency DCI (30 minutes) showed no evidence of the white matter haemorrhages which are a consistent finding in short-latency disease.[91] This indicates that the mechanisms involved are likely to be different and

possibly compatible with the minimal acute histological changes seen with ischaemia following bubble embolism.

Recently Marzella and Yin have questioned whether ischaemia playa a significant role in the pathophysiology of DCI involving the cord.[103] Using a rat model of DCI (and consequently a dive profile which would probably prove lethal to larger species), with microspheres to measure regional blood flow, they showed that lumbar spinal cord blood flow increased rather than decreased during the onset of spinal cord injury. It is unclear whether the lumbar cord in these animals was involved in the disease. Furthermore, the techniques would have been unable to detect areas of focal ischaemia which may occur in DCI. Nonetheless these results indicate that, in this model, an ischaemic/embolic mechanism is unlikely to be responsible for spinal cord dysfunction.

Other embolic theories.

End proposed that an initiating event in decompression sickness (DCS) is the agglutination of formed blood elements that lose their common revulsion by some undisclosed mechanism during decompression.[104,105] He proposed that these aggregates then act as emboli. Certainly, rheological changes in blood occur in DCI. An increased haematocrit and a loss of plasma volume are commonly found in both humans and animals with DCI.[40,78,106-110] This tends to increase blood viscosity and reduce tissue perfusion. The aggregation of blood components such as platelets[109,111-115] and leucocytes,[113] the formation of rouleaux,[108] and the finding of endothelial cell,[113,116] fat and bone marrow emboli[38,116-120] have all been described. However, these phenomena may be secondary to the nucleation of bubbles in blood or bone marrow and need not be primary events in DCI. Furthermore, the sludging of blood occurs in other conditions without resulting in the manifestations of DCI.[121] An example is disseminated intravascular coagulation (DIC) in which many of these haematological events occur on a considerable scale. However the more common consequences of DIC (haemorrhagic necrosis of the gastrointestinal mucosa, congestion of the abdominal viscera and microscopic occlusion of capillaries by thrombi with surrounding secondary, focal necrosis) are not typical of DCI. Furthermore, spinal cord involvement in DIC is most unusual.

Bubble oxygenators, part of the bypass technique for open heart surgery, impose massive rheological changes on the patient. These include the denaturation of plasma proteins, the clumping of formed blood elements and the generation of fat emboli.[123] Another complication is gas bubble embolism[124,125] which affects the brain rather than the spinal cord. Even if rheological changes were an initiating event in DCI, it is unlikely that they could account for spinal cord injury.

The dramatic improvement in DCI that is often seen with recompression, especially if applied within minutes, is difficult to explain using a theory based upon the impaction of solid emboli as the principle pathological event. If embolic phenomena are responsible for the condition, this observation would be more readily explained by compressible, gaseous emboli.

The venous infarction hypothesis.

Haymaker and Johnston raised the theoretical possibility that, under conditions of extreme DCI, bubbles in the epidural vertebral venous plexus (EVVP), combined with back pressure from bubble-laden lungs transmitted through venous anastomoses between the spino-vertebral-azygous and pulmonary vasculature, may cause venous engorgement of the spinal cord.[12] Haymaker[22] developed the hypothesis by noting Batson's observation that the EVVP is a large, valveless, low-pressure system that would make it a favourable site for the formation of bubbles.[126,127]

Hallenbeck et al. reasoned that gas bubbles are not inert in the blood stream but, as a result of a 40-100 Å layer of electrokinetic forces at the blood-gas interface, they cause structural alterations to plasma proteins.[128] This may result in the activation of the coagulation, complement and fibrinolytic cascades, the release of kinins and complex alterations to haemodynamics. They demonstrated that one of these systems, coagulation, was accelerated by the presence of bubbles.[129]

Another argument they developed was that embolic mechanisms for spinal cord injury in DCI could be criticised on the grounds that the distribution of CNS lesions appear to be unique. In other clinical, embolic conditions such as subacute bacterial endocarditis, fat embolism and mural thrombus of the left atrium, it is the brain that is the principle target organ. They quoted Blackwood's observation that arterial embolism of the cord is extremely rare. Of the 3,737 autopsies he reviewed on patients that died with neurological diseases, he found not a single case of spinal cord embolism.[130]. If emboli are responsible for the pathological findings in DCI, it is the brain rather than the spinal cord that should be preferentially embolised, since it constitutes some 98% of the mass of the human CNS and receives 75-85 times the blood flow of the spinal cord.[131] They performed a number of elegant experiments, including the direct visualisation of the spinal cord venous drainage in an animal model of DCI, that demonstrated many elements of the hypothesis that bubbles accumulate in the venous drainage of the cord and their presence, combined with their activation of the clotting mechanism, results in a slowing and eventual cessation of venous outflow. This, they observed, causes congestion and, ultimately, venous infarction of the spinal cord.[78,89,132-134] They considered that the scattered, punctate, mainly white matter haemorrhages of DCS were

compatible with Henson and Parsons' description of venous infarction of the spinal cord.[135]

This theory also has its shortcomings. First, there is some doubt that the characteristic lesions of spinal cord DCS are compatible with a venous infarction mechanism.[136] In rats, for example, obliteration of the EVVP is associated with vasogenic oedema of white matter, but not frank infarction,[137] although Martinez-Arizala et al. described haemorrhagic tissue necrosis as occurring at 24 hours and involving the grey matter more than the white.[138] Again, in monkeys, it is principally the grey matter which is involved.[139] In man, when haemorrhage in the spinal cord is associated with venous obstruction, the haemorrhage tends to be massive, centrally located, involving both the grey and white matter.[140] Venous infarction of the spinal cord is a very rare pathology[141] and this may be because the EVVP, being an extensive plexus, is difficult to obstruct. If this plexus was completely blocked at any given level, it is probable that the resulting venous congestion and infarction would be more extensive than that seen in DCI. Even obstruction at the level of the radicular veins might be expected to result in one or more lesions with a segmental distribution. Such a distribution is not typical of the lesions of DCI.

Another problem with the venous infarction mechanism relates to the frequent finding of "silent" intravascular bubbles in asymptomatic divers[67,142,143] and cases of chokes, particularly in aviators, who are free from spinal symptoms.[144] Why should "silent" bubbling, which presumably provokes similar rheological changes to symptomatic bubbling, fail to compromise spinal cord drainage? While it may be argued that such bubbling fails to exceed some arbitrary threshold, it is difficult to understand why aviators with sufficient venous bubbling to cause "chokes" do not also invariably suffer spinal cord injury.

Complement activation in DCI.

Studies in both rabbits and man show that the activation of the complement system may be an important event in the generation of the symptoms of DCI.[145-148] However cardiopulmonary bypass has been shown to activate complement in a similar manner to decompression,[149] yet without generating a syndrome similar to DCI. Furthermore, treatment of rats with a soluble complement receptor sCR-1, which has been shown to be beneficial in complement-dependent disease, failed to prevent DCI.[150] It has been claimed that variation in susceptibility to DCI in both rabbits and man correlates with the sensitivity of the complement system to activation by bubbles.[146,151]. However, others have questioned the validity of these conclusions because the extent of complement activation varies greatly over time and so predicting susceptibility to DCI on the basis of a single measurement can not be justified.[152] Furthermore, in a recent study of human repetitive dives, no association between the activation of complement in vitro and DCI was found.[153] Thus, although the activation of complement may occur in DCI, its role in the development of the manifestations of the condition is far from clear. It has never been shown how the activation of complement could result in the characteristic spinal cord lesions of DCI.

The autochthonous bubble hypothesis.

Another possible mechanism for spinal cord injury in DCI is through the liberation of a gas phase in situ (autochthonous bubbles). Indeed, it is possible to interpret the conclusions of both Boyle and Bert as proposing a role for autochthonous bubbles. However, this is rarely done. Even Boycott et al.[48] who published camera lucida drawings of a goat spinal cord showing evidence of massive autochthonous bubble nucleation, concluded that the principal mechanism involved in spinal cord injury in DCI was the embolism of arterial bubbles.

In 1916 Keyser proposed an autochthonous bubble mechanism.[154] During the discussion of a clinical case of DCI he reasoned:

"Vernon has demonstrated that fat dissolves more than five times as much oxygen and nitrogen as water.[155] The myelin of the white matter of the cord belongs to the group of fats and would, therefore, be a most common site of bubble formation in common with the fat of other parts of the body. Minute gas bubbles, which would be of no significance in the fatty tissues of the omentum or abdominal wall, would cause definite symptoms if they occurred in the cord.... From purely theoretical considerations and also the location of the lesions, it seems more probable that the bubbles form in the white matter itself rather than the blood stream."

In 1982 Hills and James, after a study of the mechanical properties of the spinal cord, proposed that spinal cord ischaemia could result if, during decompression, sufficient gas bubbles nucleate to increase spinal cord volume by 14-31%.[156] They argued that such a volume increase would raise the tissue tension sufficiently to collapse the arterioles and cut off the blood supply.

The major problem with the autochthonous bubble theory has been that, until the late 1980s, except for the observations of Boycott et al. in the goat[48] and vague references to "air lacerations"[36] or "stippling" of the white matter in early descriptions of the human pathology,[5] extravascular bubbles in the spinal cord had rarely been described. The evidence in animals was limited to the finding of bubbles scattered throughout the spinal cord white matter of six out of sixteen dogs with fatal decompression illness[41] and in the cords of decompressed fingerling salmon.[43] In man, non-staining, round spaces were

described in the cerebral and spinal cord white matter of a diver who died shortly after taking only 20 minutes to surface from a four hour dive to a depth of 40 m.[37] Numerous similar lesions were described in the cerebral white matter of two scuba divers who had apparently died prior to being brought to the surface from 42 m (140 ft). Sadly, the spinal cords were not examined.[39]

A possible reason why autochthonous bubbles have so rarely been demonstrated is that their presence in the cord may be transitory. Sykes and Yaffe examined the spinal cords of dogs that had been perfusion-fixed following recompression treatment for DCI (3 or more hours after the diagnosis).[157] Although they described abnormalities of myelin that may have been a consequence of local bubble formation, no overt bubbles could be demonstrated by light or electron microscopy.

In the mid 1980s we adapted a well established canine model of severe DCI, which had been employed for the assessment of treatments, for the investigation of the acute pathology.[158] The recording and measurement of spinal somatosensory evoked potentials (SSEP) were computerised to enable a rapid diagnosis of the onset of DCI affecting the cord. Fixation of the tissue within about twenty minutes of the diagnosis of the condition, using a rapid perfusion technique, enabled us to demonstrate very early changes.

We found that, by embedding the tissue in epoxy resin, non-staining, space-occupying lesions (NSSOL) were found at histology in the white matter of cords afflicted with DCI but not in undived controls or dived dogs with no loss of function. When paraffin wax was used as the embedding material, occasional artefactual NSSOL were found caused by the section tearing as it was cut. The size of the decompression-induced NSSOL ranged from 20 µm - 200 µm in diameter. We inferred that these lesions were likely to have contained gas in vivo because the surrounding tissue appeared to be compressed as would occur with an expanding bubble of gas. Similar findings from another canine model of DCI which employed a less stressful dive profile were reported by Burns et al.[159] They demonstrated that these lesions were gas- filled by immersion fixing the tissue in formalin at different pressures and showing that the size distribution of the NSSOL varied in accordance with Boyle's Law.

The question is how these lesions provoke tissue dysfunction. To assess this, the cords of 5 animals which had shown evidence of a loss of spinal cord function within 4-6 minutes of surfacing were rapidly fixed at the time of diagnosis (when the amplitude of the SSEP fell below 80% of baseline), but before the minimum amplitude of the SSEP was reached. This was to preserve the size of the NSSOL. From previous experience with the model[160-164] we knew that the minimum amplitude of about 20% of baseline was reached within 15-20 minutes of diagnosis, when the cords in this study would have been fixed. The cords were then serially sectioned from the conus to T12 (at which level the SSEP were measured) and submitted to computerised morphometry. Although the proportion of spinal cord white matter occupied by bubbles was small (always less than 0.5%) we concluded that autochthonous bubbles would account for the loss of cord function if between 30% and 100% of the fibres which were displaced by them were rendered non-conducting. The means whereby they might achieve this are:

1 Destruction of axons at the site of bubble formation. It was estimated that this effect would account for only 1% of the functional deficit.
2 Stretching and compression of axons around the growing bubble. This neurapraxia is an attractive mechanism because the onset is very rapid (unlike ischaemia in the cord) and reversible.[165-168] It could account for the most fulminant presentations of the condition and the improvement which is commonly seen if recompression is undertaken early or the more gradual spontaneous recovery which is often seen.
3 A biochemical insult akin to the complex interaction between blood and bubbles. If this effect were limited to those axons adjacent to the bubble surface, we calculated that this would, at most, account for 50% of the loss of function. If there is such an effect, it is unlikely to be the sole cause of the loss of function.

Another mechanism by which the cord may be injured in DCI was raised by Broome.[169] He correlated functional outcome, in pigs, with the extent of haemorrhage into the tissue, which he showed to occur after early recompression. Expanding bubbles in spinal white matter may not only disrupt axons but also the delicate microcirculation. Lacking connective tissue support, these vessels may be uniquely vulnerable to such an insult. The resulting haemorrhage can be expected to be punctate in distribution.

A degree of supersaturation is necessary to provide the number of molecules necessary for bubbles to form and grow. In a study of the spinal cords of 18 animals which were saturated for 4 hours at a fixed pressure and cardiac arrest induced prior to decompression, it was found that few bubbles formed at a saturation pressure of less than 3.6 bar, equivalent to diving to 26 m (86 ft). This indicates that bounce dives to depths much less than this are unlikely to provoke autochthonous bubble formation. The intact cord will off-gas increasingly with time following a dive. Unless bubbles form early, the probability of their formation decreases with time. In only two of our animals was the onset of spinal cord dysfunction observed more than 30 minutes after surfacing. In these, examination of the cord showed no evidence of autochthonous bubbles. The appearance of the cords in these cases closely resembled that of bubble embolism.[91]

Since the description of autochthonous bubbles in the spinal cords of canines with DCI, they have been found by other investigators[170,171], although in the first of these studies the number found was not considered to be sufficient to account for the observed loss of function.

Conclusions

1 Given the range of latency of onset of spinal cord injury in DCI from during decompression to as long as 48 hours after surfacing, it is most unlikely that any single mechanism will apply in all circumstances.

2 The onset in fulminant cases, which tend to occur after deeper dives, requires a local mechanism which rapidly interferes with white matter conduction. Autochthonous bubbles meet these requirements.

3 A purely embolic mechanism is an attractive explanation for cases in which the onset is delayed by a few minutes after completing the decompression (perhaps 10-20 minutes, or 1-2 time constants of intact spinal cord gas exchange) or which arise from dives to less than about 25 msw. The mechanism of amplification of embolic bubbles by the diffusion of excess tissue gas, as proposed originally by Boycott et al., although lacking direct experimental evidence, may occur slightly earlier than this, particularly in divers with significant right-to-left shunting through a PFO or other atrial septal defect.

4. The role of the venous infarction hypothesis is unclear. It is unlikely to provoke a fulminant loss of function requiring, as it does, obliteration of most of the flow in the epidural vertebral venous plexus, which appears to take time. The role of this mechanism in cases with a delayed onset will be determined only when the pathology of that presentation is clearly defined as the histology of venous infarction and spinal cord gas embolism are quite distinct. The evidence, from only two experiments reported by Francis,[91] is that, in his model, an embolic mechanism was likely to be responsible for loss of function 30 or more minutes after surfacing from the provocative dive.

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DECOMPRESSION ILLNESS IN SPORTS DIVERS THE UK EXPERIENCE

James Francis

Abstract

The development of the diving accident database at the Institute of Naval Medicine in 1990 has facilitated the study of decompression illness (DCI) in the UK. Although the collection of data remains incomplete, largely because participation in the scheme is voluntary, the situation is improving. The data from 595 cases of DCI arising from diving which were collected from 1991-1995 are reviewed and summarised. Four hundred and eighty two (81%) of the cases were recreational divers and 499 (84%) were male. In 434 cases (73%) the diver was aged under 40 years. In the cases where depth was recorded the average depth of the preceding dive was just over 30 m. The nervous system was involved in 457 cases (77%). Only 77 (13%) presented with what has classically been labelled Type I, or pain-only, decompression sickness (DCS). Only 325 divers (55%) were reported to have made a complete recovery following one recompression treatment. The remainder had residua of some kind. With a second treatment the cure rate improved to 68% (404 divers) and with a third to 74% (440 divers). One hundred and seven divers (18%) still had symptoms and signs after their hyperbaric treatments. In 8% (48 divers) there was no indication of the extent of recovery in the case report.

Key Words

Accidents, decompression illness, diver emergency services, recreational diving, sequelae, treatment.

Introduction

In 1990, Dave Smith, Rob Hills and I set up a diving accident database at the Institute of Naval Medicine (INM). The primary objective was to attempt to better define the syndrome of decompression illness. This turned out to be a considerably more involved task than was originally thought. The problems included software development, our initial choice of Advanced Revelation proved to be an error and the entire thing was rewritten in Fox-Pro in 1994 by Mike Ralph. We needed the cooperation of as many chambers in the UK as possible and, quite coincidentally, this requirement coincided with the founding of an organisation, which is now titled the British Hyperbaric Association, which has been a tremendous help. We had to persuade a lot of doctors to fill in our forms, legibly. This was not easy for some as it meant an end to the three line case report, dive profile, diagnosis and treatment table, which abound in the archives at INM and, I suspect, elsewhere. Instead, they were forced into recording a detailed description of the condition, its evolution, management and outcome. Last but not least, we needed a team of people to nurture the database by performing the essential tasks of data audit and entry. With each report containing over 100 fields, and with over 1,000 records now in the database, I am particularly grateful to Dave Smith, Mike Ralph, Peter Benton and Paul Kelleher who manfully took on this task.

Over the years, data collection has become more complete as more chambers have agreed to use our forms but it is still not universal. Each year the British Sub-Aqua Club (BSAC) collate data on UK diving accidents and it is evident that the INM database only contains about two thirds of cases identified by the BSAC. Those which are treated in an Accident and Emergency department or by family practitioners, and those who receive no treatment or who die, tend to be omitted. Nonetheless, the database now gets reports on over 90% of cases which attend a hyperbaric treatment facility. Unfortunately, a proportion of reports are still submitted with incomplete data fields, some do not get posted, some are still illegible and all of these need to be chased up. Thus, although the data set is incomplete, all this effort has permitted us to take a better look at DCI in the UK than has been possible previously and I will now summarise the most recent five years of data which are available to me. I searched the database in February 1996, at which time the 1995 data set was incomplete.

Results

Table 1 shows that the case load varies considerably from year to year. This is a familiar pattern. In 1989 there was over 200 cases yet in 1990 there was less than half that number. There is no satisfactory explanation for this variation. It would be of interest to compare the incidence of DCI with the number of dives conducted. Unfortunately there is no such record in the UK and thus it is not possible to determine whether there is a fairly constant incidence of DCI per 1,000 dives and the variation in incidence reflects variations in the number of dives undertaken or whether it is the incidence per 1,000 dives which varies. This is a problem which is universal in recreational diving. The planned launch, in 1998, of DAN's Project Dive Safety, in which the denominator will be known for a large sample of divers may make such an analysis possible.

TABLE 1

Year	Cases of DCI
1991	127
1992	98
1993	92
1994	170
1995	108
Total	**595**

Where these cases receive treatment is of some interest (Table 2). Much of the recreational diving in the UK is undertaken in the South West and off the West coast of Scotland. Inland, a lot of diving takes place at Stoney Cove in Leicestershire. It is therefore not surprising that a high proportion of cases are seen at the Diving Diseases Research Centre (DDRC) in Plymouth. Military chambers in the West of Scotland help out Aberdeen with the Scottish patient load, whose representation is artificially low in these data because it is only since 1994 that they agreed to send case reports to the INM. Whipps Cross Hospital, being in London, serves a large population of recreational divers but tends to get cases which develop some time after the provocative dive and also receives the growing number of cases which arrive by air at Heathrow or Gatwick from diving holidays around the world. INM used to receive just about all the cases which occurred on the South Coast. However, since the Chamber at Poole opened in 1994 a growing proportion are treated there.

Most of the cases are male (500 or 84%). Recreational divers provided 482 cases (81%). Military divers contributed only 36 (6%) cases to the database, the remaining 60 (10%) being occupational. The apparent safety of military diving has little to do with the advanced technology of their equipment but results from close adherence to procedures, with the supervisor exercising strict control of the divers in the water, and a conservative use of tables. It will be interesting to see if things change with the introduction of a set in 1997 with a deep (80 m) bounce-dive capability which will necessitate the military diver having greater control over his dive profile.

Despite the rapid growth of the dive computer industry during this period, when the information was recorded, 44% of decompressions were calculated using tables, commonly BSAC 88. The age distribution of cases is shown in Table 3. Table 4 shows that the dive preceding the onset of DCI tends to be quite deep, on average almost 30 m. Not surprisingly, many of these (43%) involved a staged decompression. This figure is likely to grow in the future as the popularity of deep, mixed-gas diving increases.

As we have published previously, the majority of cases presented with multiple manifestations, the most prevalent of which were neurological.[1,2] Dividing the manifestations into broad categories of: neurological (excluding pain), pain (predominantly limb pain), constitutional (including headache, malaise, fatigue, nausea and vomiting), skin, pulmonary and lymphatic, only 35% of cases presented with manifestations in a single category. The frequency of reporting each category is shown in Table 5 and a breakdown of the frequency of neurological involvement is shown in Table 6.

TABLE 2

CASES BY TREATMENT CENTRE

Diving Diseases Research Centre (DDRC) Plymouth, South West England	131
Defence Research Agency and INM Portsmouth area, South coast England	95
Other military chambers	58
Whipps Cross Hospital, London	62
Poole, Dorset, South West England	47
Dunstaffnage	27
Aberdeen, North East Scotland	26
Stoney Cove, Leicestershire, Midlands, England	24
Lancashire Constabulary, North West England	14
Fort William, North West Scotland	14
Great Yarmouth, East Anglia	13
Northumbria, North East England	12
Craigavon	9
Cork Naval Base, Eire	2
Other	61
Total	**595**

TABLE 3

AGE DISTRIBUTION OF CASES

Age	Divers	%
Under 20	24	4.0
20-29	194	32.6
30-39	214	36.0
40-49	107	18.0
50-59	24	4.0
60-69	2	0.4
Unknown	30	5.0
Total	**595**	**100.0**

TABLE 4

DEPTH OF DIVE PRECEDING ONSET OF DCI

Depth (m)	Number	%
0-9	31	5.2
10-19	117	19.6
20-29	174	29.2
30-39	153	25.7
40-49	65	10.9
50+	40	6.8
Unrecorded	15	2.6
Total	**595**	**100.0**

Remarkably, what I was once taught as being the most common presentation of DCI, the old 'Type 1" or pain-only DCS was seen in only 77 divers (13%). I can offer no firm explanation for this apparent disparity. It could be that the disease has changed. Advances in equipment, notably the thermal protection afforded by modern diving suits and larger capacity bottles mean that deeper and longer dives can be undertaken and this may influence the presentation of the DCI which results. It could also be that doctors are getting better at eliciting neurological symptoms and signs. Personally, I have found that being required to complete an accident form has improved the completeness of my history taking and the thoroughness with which I record my clinical observations.

A finding from these cases questions another piece of classical teaching, namely that DCI recovers with adequate treatment. In fact, only 325 cases (55%) in this series made a complete recovery after the first treatment. This increased to 404 (68%) after the second treatment and to 440 (74%) after the third. Further treatments reap a diminishing return in terms of complete recovery with 107 divers (18%) having residual manifestations at the completion of their hyperbaric treatments. Surprisingly, in only 77% of cases with limb pain was this completely resolved after the first treatment.

A final feature of the management of DCI in the UK which has changed over the past few years is the first aid management of the condition. In particular, the use of oxygen is becoming more common. Although the first-aid section of the diving accident reporting form is one which is generally completed less fastidiously than others, the available figures support my own observations. In 1991 only 6% of cases were recorded as receiving oxygen, 4% oral fluids and 3% both. In 1995 this had improved to 28%, 18% and 16% respectively. There are two principal reasons for this: the Coastguard now routinely carry oxygen and use it when recovering injured divers. More importantly, more dive boats and clubs are equipped with oxygen and trained in its administration. The BSAC now provides an oxygen administration course for sports divers which includes a three hour session on: the medical aspects of diving; symptoms and signs of diving-related injuries; resuscitation and the theory of oxygen and fluid administration. This is followed by a 2 hour practical session using a "Resusci®Anne" and then theory and practical examinations. It is too early to tell how this will affect the outcome of DCI, but it is unlikely to be detrimental.

TABLE 5

CATEGORIES OF MANIFESTATIONS IN 595 DIVERS

Category	Cases	%
Neurological	458	77
Pain	369	62
"Pain only"	77	13
Constitutional	196	33
Cutaneous	71	12
Pulmonary	36	6
Lymphatic	6	1

Note that many patients had more than one type of manifestation. See text for description of categories.

TABLE 6

NEUROLOGICAL MANIFESTATIONS IN 458 DIVERS

Manifestation	Cases	%
Sensory abnormalities	350	76.6
Motor weakness	160	35.1
Coordination abnormalities	97	21.2
Special senses	74	16.2
Higher function abnormality	49	10.6
Altered level of consciousness	42	9.1
Loss of sphincter tone	16	3.5

Note that many patients had multiple neurological manifestations.

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DCI IN SPORTS DIVERS DAN USA EXPERIENCE

Richard Moon

Key Words
Accidents, decompression illness, diver emergency services, recreational diving, research.

The Divers Alert Network (DAN) in the USA was set up some years ago, primarily with the idea of providing emergency medical consultation for divers with decompression illness. There was at that time, and there still is, a fairly small number, proportionately, of physicians who are familiar with decompression illness and we continue to this day to provide that service as our major function.

In 1996 there were 13,000 information calls during the course of the year. These are non-emergency "Can I dive if ...". Now that we are on the Internet we are receiving an increasing number of e-mail enquiries, 1,000 last year. We also had roughly 2,000 emergency calls.

We enter all the recreational scuba diving decompression illness data we receive in the DAN database. The number of cases reported to DAN has been steadily increasing. This is not, I suspect, because the number of cases has been going up, but our ability to acquire information has gradually improved. In the same period we have actually recorded about 600 cases per year in the decompression database. We receive forms on about two thirds these. Unfortunately many of them cannot be used because of incomplete data. We actually record about 600 cases per year in the decompression illness database.

We are a little bit schizophrenic in the way we classify decompression illness. We use the traditional classification. Type I includes pain, fatigue, skin, lymphatic symptoms and signs. Type II decompression sickness includes neurological or cardio-respiratory. We still use the term arterial gas embolism (AGE), being the pathophysiological classification of pulmonary barotrauma with intravascular gas, separately. Using that classification, approximately a quarter of our cases are recorded as Type I by the treating physician. About two thirds as Type II decompression sickness, and about 8% AGE. We have seen the proportion of AGE steadily decreasing over the last 10 years. It started off somewhere around 18% of cases and is now down around 8%. I suspect that this is actually not a reduction in the disease, but rather the decreasing frequency with which it is diagnosed.

It is interesting that there is a very high incidence of misclassification. The treating physicians classified 97 patients who had neurological symptoms as Type II. Twenty of these were reclassified as severe neurological cases and 77 as mild. Also a number of pain only cases, as reported by the diver himself, were reported as Type II decompression sickness or arterial gas embolism. We are

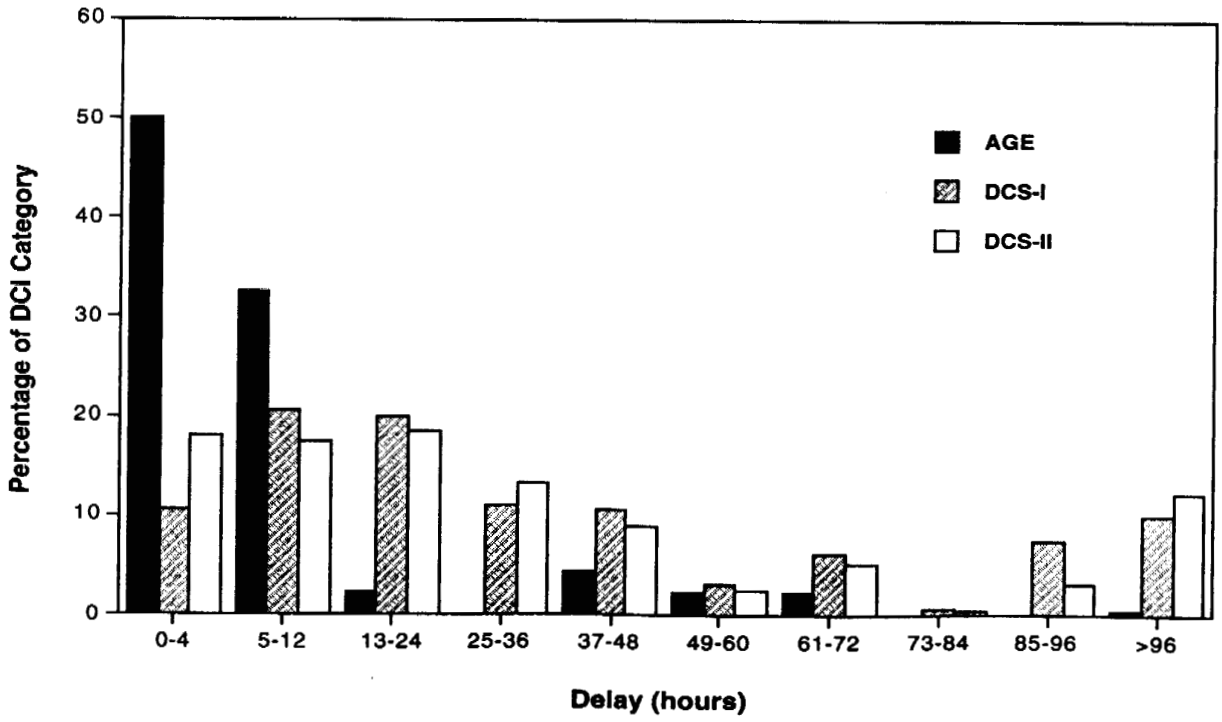

Figure 1. Delay from symptom onset to recompression therapy.

slowly getting rid of this classification in our database. It is kept only because it is so entrenched in the literature and we keep getting enquiries about the proportion of cases in these various categories.

It is interesting that a reasonably high proportion of divers with decompression sickness have previously reported DCI. Approximately 10 - 20% of cases between 1990 and 1992 had previously experienced decompression illness. This suggests to me that these " repeat offenders " represent a high risk subset of divers with an intrinsically high risk of DCI.

Chris Lawrence told us that it usually takes 24 hours from the time of death to starting an autopsy. That is very close to the time it takes our divers get to the recompression chamber. Figure 1, taken from DAN data, shows the delay in hours from symptom onset to recompression. Arterial gas embolism is in shown in black, Type I decompression sickness is cross hatched, and Type II is white. The median delay was about 24 hours, part of which is due to the long distances we have, very similar to the ones in Australia, but also because the divers do not inerpret their symptoms correctly or report them to anyone. Figure 2, also from DAN data) is delay from onset of symptoms to calling for assistance. Most of the AGEs get reported fairly quickly, but there is very a long tail on this distribution and sometimes people do not do anything for several days.

It is unfortunate that our educational efforts are not taken to heart. It turns out that many divers with decompression illness actually experienced symptoms prior to their last dive. That is, they made a dive even in the face of symptoms, some of which are rather worrying. Nine divers in 1995 had severe neurological symptoms before their last dive and another 5 had difficulty breathing. I am not sure what this behaviour represents. It could be group pressure or denial.

We vigorously promote the use of oxygen as an initial treatment for DCI and have done so for many years. Unfortunately US divershave been fairly unreceptive so far. Only a small proportion of divers actually use surface oxygen, and many of these applications are very brief. In 1995 only 190 of 590 divers with DCS (32%) used oxygen. A couple of summers ago I was treating a diver , and he told me that he had used surface oxygen. I asked him "How long did you breath it for?", and he replied "About 5 minutes". I said, "Why only 5 minutes?", and he said, "Well, there were several other divers on the boat who had symptoms and they were sharing the tank".

We also collect information on fatalities. In the bad old days of the 1970s there was a rapid increase in the number of fatalities per year, reaching a peak of 147 in 1976. Those were the days when untrained divers were doing things like cave diving and getting into serious trouble. The number of fatalities in recent years has been around 100 and it seems to be fairly stable. We are also experiencing other patterns of diving besides the traditiona lcompressed air breathing scuba. Divers are doing all kinds of fairly technically difficult and challenging dives. There is a lot of nitrox use and some of these divers having decompression accidents. One of the unfortunate things is that the quality

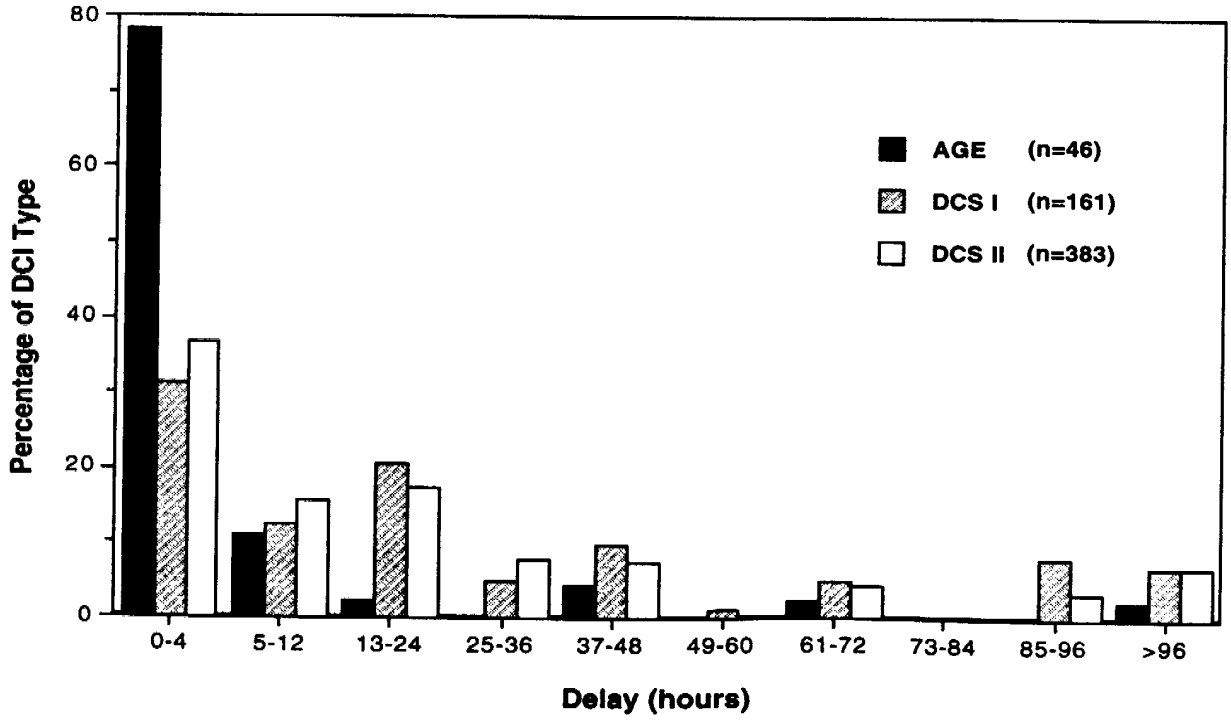

Figure 2. Delay from symptom onset to calling for assistance.

control on the gas mix is very variable and some divers know they are breathing nitrox but they have absolutely no idea what percentage.

We have now over 3,000 cases in the DAN database, and one of our major tasks which did not exist at the beginning of DAN but certainly does now, is research to try to promote diving safety and investigate pathophysiology. There are several ongoing projects in addition to analysis of the database. For instance in 1995 there were 590 accidents (DCS and CAGE). Analysis showed rapid ascents (170), buoyancy problems (90), dives outside the limits of computer or table(50) and equipment problems (77) were associated with nearly two thirds of diving accidents. There were also 35 divers who did not use a computer or tables. Adding them brings the total up to 422 divers or 71.5% with avoidable causes. Perhaps if we could change this behaviour, we might substantially reduce the number of decompression incidents.

Dick Vann is doing a project on flying after diving. A number of volunteer divers have been in our chambers, made a dive, followed by a pre-flight surface interval and then a simulated flight to an altitude of 8,000 feet. He has looked at a number of dive profiles at 60 fee as well as some 100 foot exposures. It appears that the guidelines that we currently operate under, which which call for a surface interval before flight of 12 to 28 hours, seem to be correct. After 12 hours for a single dive, and beyond 22-24 hours for a multiple dive, the risk of decompression sickness is fairly low.

Dr Vann is also working on a long term project to collect depth-time profiles on enough dives to be able to assess the probability of DCI for any reasonable dive within the framework of recreational scuba. Project Dive Exploration is expected to take up to 10 years to complete and require actual recordings of about a million dives. Primary data collection is expected to start in 1998.

Other projecs include field studies of blood glucose in divers with insulin-dependent diabetes mellitus, assessment of venous gas embolism in recreational divers and studies of the relationship between oral contrceptives, menstruation and the risk of DCI. These are in parallel with the statistical analysis of the ever enlarging data base.

Figures 1 and 2 are reprinted from the 1997 DAN Report on Decompression Illness and Diving Fatalities by kind permission of the Divers Alert Network.

More details about the 1995 recreational scuba diving injuries and deaths reported to DAN can be found in the 1997 edition of the DAN report on Decompression Illness and Diving Fatalities which is available from DAN, the Peter B Bennett Center, 6 West Colony Place, Durham, North Carolina 27705, USA.

Professor Richard E Moon was one of the Guest Speakers at the 1997 Annual Scientific Meeting at Waitangi, New Zealand. His address is Department of Anesthesiology, Duke University Medical Center, PO Box 3049, Durham, North Carolina 27710, USA. Phone +1-919-681-5805. Fax +1-919-681-4698. E-mail moon0002@mc.duke.edu .

DES AUSTRALIA EXPERIENCE

Michal Kluger

Key Words

Accidents, decompression illness, diver emergency service, first aid, recreational diving, transport.

Australian diving accident statistics are even less well collected than the statistics in the United Kingdom and the USA. The Diver Emergency Service, usually known as DES, is primarily a phone contact for medical advice to divers who have had diving accidents. It advises divers and tells them how to contact the appropriate authorities in their area who will organise their treatment. It is also contacted by medicos wanting diving medicine advice. There is no national data base of diving accidents in Australia.

More about DES

The Diver Emergency Service's primary function is to be the first contact in diving emergencies. Advice about diving medicine is very much less important. DES is accessed by phone. We have a free phone within Australia (1-800-088-200), which any diver can access at any time of the day or night. Outside Australia it is a user pays number (+61-8-8373-5312), which comes through to the same physicians 24 hours a day.

When a diving emergency occurs, divers phone the free phone number, or the number from outside Australia, and reach the St John's Ambulance switchboard in Adelaide. Immediately one of the five diving medicine consultants is paged. Our set up is unlike some of the other call networks around the world, as divers have immediate access to a physician, trained in anaesthesia, intensive care and diving medicine, within 3-5 minutes.

The DES workload

In 1996 there were approximately 506 calls. Approximately, because like all reporting statistics, not all the cases which phoned were recorded. Not surprisingly, most of the calls came from heavily dived areas, such as Queensland and New South Wales. Interestingly, we got

several calls from Alice Springs, right in the middle of Australia where the only permanent water is in swimming pools, divers who had been on a round Australia tour and had flown from say, Cairns, straight to Alice Springs and developed symptoms there. As Alice Springs is 1,350 km from the nearest hyperbaric facility we often had significant problems about what to do with them.

Besides 452 Australian calls we had 52 overseas calls. We had several calls from areas that are commonly dived by Australians in the immediate vicinity, such as New Guinea, the Solomons, Vanuatu, Fiji, and Guam. However, we also got some calls which were geographically very distant such as the United States, Cyprus (this was a diver who had complications 5-6 days after a dive and who did not really know who to contact in Europe), Hong Kong (this was a patient who three weeks after a dive decided he was worried about his symptoms at 10 o'clock at night and decided to phone me, unaware of the time zone difference and at 2 in the morning decided to tell me about his nagging shoulder pain); one from the Philippines and one from the United Arab Emirates.

TABLE 1

ORIGIN OF CALLS

Location	Number
Australia	
Queensland	158
New South Wales	131
Victoria	66
South Australia	39
Western Australia	37
Northern Territory	7
Tasmania	7
Australian Capital Territory	7
Total Australian calls	452
Near-by Pacific Islands	
Papua New Guinea	14
Solomon Islands	12
Vanuatu	6
Fiji	5
Total near-by Pacific Island calls	37
Other overseas calls	15

Presenting symptoms

We record the presenting symptoms of all those referred to DES over the DES phone. Pain, paraesthesia, headache, fatigue, this is just all the symptoms grouped together. Marine envenomation is put separately. About two years ago the specific emergency telephone service Stinger Line stopped functioning, so now we take the primary calls for marine stingers around Australia. Not surprisingly most of the envenomations come from Queensland, although there were a couple from South Australia, and a queried account from Western Australia. Of these, 4 were *Chironex*, one a sea urchin, one a crown of thorns, one a stone fish and one was not an envenomation but was poisoning from eating fish which gave GI symptoms.

TABLE 2

PRESENTING SYMPTOMS

Presenting symptom	Number
Pain	189
Paraesthesia	106
Headache	97
Fatigue	82
Nausea	65
Vertigo	63
Concentration reduced	32
Weakness	28
Visual problem	17
Loss of consciousness	12
Skin rash	11

Primary diagnoses

Twelve of the primary diagnoses made by the physicians receiving DES calls in 1996, were definite diagnoses, over the phone, of decompression illness (DCI). Another 132 were diagnoses of possible decompression illness. There are approximately 400 cases of decompression illness per year around Australia. Obviously not all of these go through the DES network. Many get referred straight to the local hyperbaric centres.

Other primary diagnoses were sinus problems, oral barotrauma or simple musculo-skeletal problems. Other included anxiety or panic, pulmonary barotrauma, which is usually manifest as subcutaneous emphysema. Another less specific subgroup, head injury, was interesting. A man who

TABLE 3

PRIMARY DIAGNOSES

Diagnosis	Number
Decompression illness (DCI)	112
Possible DCI	132
Aural barotrauma	34
Musculoskeletal injury	20
Not dive-related	12
Anxiety	11
Pulmonary barotrauma	9
Viral illness	8

was filming underwater on the east coast of Australia banged his head on the camera. He felt completely well, but reported it just in case it was a problem later.

Symptoms of DCI

The symptoms follow the same pattern as the numbers from the United States and the United Kingdom. The largest group is neurological symptoms including paraesthesia and paralysis, followed by pain only and the other ones. A symptom we see commonly, and we were not sure how seriously to take it, is the excessive fatigue that divers with decompression illness often experience. This is often one of the most difficult symptoms to get rid of when treating them post-recompression.

There were a few who were unconscious and a few with visual disturbances. Twenty five out of 240 cases of definite or probable decompression illness had a past history of decompression illness. That is about 10%, again consistent with UK and USA numbers. Twenty four of these divers presented with the same symptoms as decompression illness. Of these we thought 14 had decompression illness again. We referred 113 of our callers to other hyperbaric chambers around the country.

First aid treatment.

If first aid is bad in the United Kingdom and America, we are certainly not doing very well In Australia. Forty seven divers were given 100% oxygen and a further 26 had a variable percentage of oxygen. That is 73 (30%) of 242 cases who were given oxygen. As 70% were not given oxygen, it is clear that oxygen is not available at all dive sites. Fluid was not generally given. We can understand intravenous therapy not being commonly given, but it does seem that oral fluids have not been aggressively advocated in Australia.

In-water oxygen

Carl Edmonds will be pleased to see that in-water oxygen was used by two divers. One was an American, sailing in Micronesia and diving off their boat. He got symptoms and phoned DES. The consultant who took the call was less than happy at in-water oxygen, but the diver went ahead and did it. The diver phoned back when he had got so much better that they continued their sailing around Micronesia. Certainly, from that case in-water oxygen seemed to have a beneficial effect. Two divers had EAR and then subsequent CPR. Anybody who has tried to do in-water EAR will recall how difficult that actually is. In fact in-water EAR is almost impossible to do in a real life situation.

TABLE 4

FIRST AID AND OXYGEN USE

Treatment	Number
Oxygen (100%)	47
Oxygen (unknown percentage)	26
Oral fluids	15
Intravenous fluids	6
Supine	33
In-water oxygen	2
Expired air resuscitation (EAR)	2
Cardiopulmonary resuscitation (CPR)	2

Deaths

Six deaths were reported through the DES desk. We had two forms which were either incomplete or illegible. Two were asking for post mortem advice. One was from Christmas Island, who said, “This diver has died, we can’t get him off the island for two days. Is it okay to stick him somewhere? How about the walk-in freezer?” It seemed like a good idea to us and two days later he flown down to the Cocos Islands.

One diver died 10 days after a rapid ascent. He was completely well during that 10 day interval, but we were phoned up to see if this death could possibly be due to diving. It was difficult to make any suggestions.

The only definite death due to diving which we recorded, was a novice diver in Queensland who made an uncontrolled ascent from about 10 metres and had an acute neurological DCI and gas embolism.

Retrievals

We recommended retrievals in 33 cases. They were Australian and international retrievals. One example, an Australian citizen, who had been snow skiing in Denver, came back via Fiji. He developed acute neurological decompression illness after a very benign dive profile, became almost quadriplegic and was put in the chamber in Suva. He was given two RN 62s and then we were phoned in Adelaide to give some advice about this diver who really wasn’t doing very well. We asked, “Was the diver insured?”, and he was not. So we then had to organise a retrieval. The retrieval was quoted to us as $35,000 to get this diver from Fiji to Melbourne. A very irate father phoned me and said “Can you justify spending $35,000 dollars. Can you assure me that this will make any difference to outcome?” Which is very difficult to do over the phone. But if one has somebody with neurological symptoms, failing to resolve

with two treatments, the Suva chamber is not the place to have them. But sometimes expectations are quite high.

Non-emergency calls

General medical enquiries using the free phone number make up a significant proportion of DES calls. Ninety one (18%) calls were general medical enquiries and this group seems to be increasing. DES was set up to provide advice for divers involved in diving accidents. The medical service is provided free by five unpaid consultants who do this service in addition to their normal duties. DES was not set up to provide a toll free number for doctors to get advice during diving medicals. There is a phone number (08-8222-5116) available for such advice in the Hyperbaric Medical Unit at the Royal Adelaide Hospital.

Commonwealth Government funding for DES was discontinued during the past two years. Now after a period of difficulty in funding the DES phone is being funded by DAN Australia South East Pacific.

In conclusion, looking at the 1996 statistics, DES still provides a valuable service to divers in Australia and for Australian divers who are travelling overseas. We can understand why doctors find it useful for medical advice. In this user-pays worlds it would reduce our operating costs if doctors used the Hyperbaric Unit line for advice.

I would like to thank Steve Goble, the Senior Hyperbaric Technician at the Royal Adelaide Hospital, for his assistance in preparing the data.

Dr Michal Kluger, FFARCS, Dip DHM, was on the staff of the Hyperbaric Medicine Unit, Department of Anaesthesia and Intensive Care, Royal Adelaide Hospital, for some years. His address is now Department of Anaesthesia, Auckland Hospital, Park Road (Private Bag 92024), Auckland 1, New Zealand.

DIVING MEDICINE COURSE

The School of Public Health and Tropical Medicine, James Cook University, Townsville, will be conducting a course in Diving Medicine from Monday 6th to Saturday 10th of October 1998.

For further details contact
Dr Peter Leggat, Senior Lecturer
School of Public Health and Tropical Medicine
James Cook University,
Townsville, Queensland 4811

Telephone 07-4722-5700

DECOMPRESSION ILLNESS IN NEW ZEALAND DIVERS: THE 1996 EXPERIENCE

Karen Richardson, Simon Mitchell, Michael Davis and Marie Richards

Key Words

Decompression illness, diver emergency services, recreational diving, sequelae, transport, treatment.

Introduction

Two hyperbaric units, one located at Auckland in the Royal New Zealand Navy (RNZN) Base, and one at Christchurch, previously located at Princess Margaret Hospital and now at Christchurch Hospital, provide treatment for injured divers in New Zealand. From 1967 to 1983 the average number of patients seen at the RNZN unit was less than 2 annually, but from 1984 to 1990 this rose to a mean of 15 per year.[1] There were 24 cases in 1990, 31 in 1991, 55 in 1992, 68 in 1993, 48 in 1994 and a record 100 cases in 1995.[2] The Christchurch Hyperbaric Unit (CHU), treated an average of 6 divers per year from 1979 until its temporary closure in May 1994. The unit reopened in February 1996 and its 1996 caseload is included in this review.

During the 1996 calendar year 76 cases of decompression illness (DCI) following diving were treated in New Zealand: 57 at the RNZNH Slark Hyperbaric Unit (SHU); and 19 at the CHU. Demographic data describing this patient population is presented in this review.

Methods

Relevant data describing patients diagnosed as having DCI and treated by recompression at both units during 1996 were entered on a Microsoft Access 2 database. One case of DCI induced by extreme altitude exposure in an unpressurised aircraft was excluded from this review. Most data was gathered prospectively by patient interview and examination, but some was obtained retrospectively from clinical records. The collection of data at the SHU was aided by use of a baseline clinical data form designed for use in a randomised prospective double blinded trial of lignocaine in the treatment of DCI which is currently underway. The relationship between incomplete recovery at discharge and a variety of putative prognostic factors was assessed using a Chi square test.

Results

SEASONAL INCIDENCE

The peak incidence of DCI was in the warmer months October to April, while there were very few cases in July.

The number of cases by month is plotted in Figure 1.

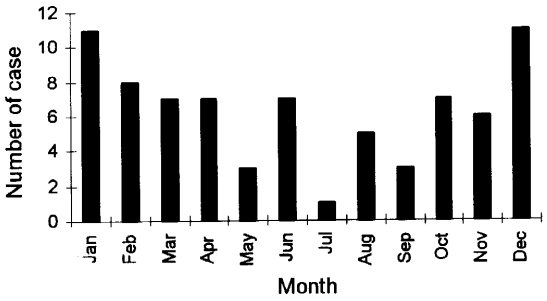

Figure 1. Numbers of DCI patients treated in New Zealand by month during 1996.

AGE AND GENDER OF DIVERS

The age of divers ranged from 14 to 51 years, with a mean of 31.5 (8.6 SD). Fourteen divers (18%) were female (21 to 37 years, mean 27.6) and 62 (82%) were male (14 to 51, mean 32.6)

PAST DIVING HISTORY

Seven divers had received no formal diving training. Three divers suffered DCI during training for their initial diving qualification. The training history was not recorded for five divers. All others held recognised qualifications. Table 1 records the number of divers trained to each qualification level and the number of divers awarded their highest qualification by each major training agency.

In the 57 patients treated at the SHU, diving experience before suffering DCI was variable with the number of dives ranging from 0 to 8,000 with a mean of 614 (SD 1,420). The deepest previous dive ranged from 9 m to 72 m (mean 37 with SD 14). Twenty two divers admitted to diving deeper than 40 m on at least one occasion, including 7 of those trained to instructor level and 2 of the untrained divers. The percentage of divers treated at the SHU with fewer than 20 and with 100 dives before their episode of DCI was 21% and 44% respectively.

TABLE 1

HIGHEST DIVING QUALIFICATION AND THE ISSUING AGENCY FOR THE HIGHEST QUALIFICATION

Highest qualifications	**Number**	**%**
Open water diver	30	39
Advanced open water diver	6	8
Rescue diver	5	6
Divemaster	5	6
Instructor	10	13
Under training	3	4
Commercial	3	4
Diving Medical technician	2	3
No training	7	9
Not recorded	5	6
Issuing agencies	**n**	**%**
PADI	36	47
SSI	11	14
NZUA / CMAS	10	13
NAUI	2	3
Royal Adelaide Hospital	2	3
No qualifications	7	9
Unknown	8	11

NATURE AND LOCATION OF DIVING

All cases of DCI developed after air scuba diving except for one using surface supplied air , two which occurred in chamber attendants and one which occurred in a snorkel diver (see Discussion). Seventy cases (92%) were diving for sport or pleasure, while 6 were engaged in occupational activities. Forty nine divers were diving in waters around New Zealand's North Island, 19 in South Island waters, 2 in Australia and 4 off various South Pacific Islands. Two DCI patients were diving in recompression chambers as attendants.

REFERRAL AND TRANSPORT

The majority of patients were referred for assessment either by their local doctor or by themselves. Most referrals were made via the dedicated Diver Emergency Service (DES) telephone line funded by New Zealand Underwater and maintained at the SHU. Common forms of transportation to the treating unit included fixed wing, one bar pressurised air ambulance, helicopter ambulance and private vehicle. Further details of referral and transport modalities are given in Table 2. The time from surfacing after the last dive to arrival at the treatment facility ranged from 30 minutes to 24 days, mean 67 hours (SD 113).

DEPTH, TIME AND OTHER RISK FACTORS

Thirty two divers used standard tables to assess their decompression status, 23 used dive computers while 3 used a combination. Eighteen divers used no form of assessment and for 3 divers the means of decompression status control was not recorded. Thirty eight divers (50%) reported dive profiles within the limits set by their table or computer, while 35 (46%) either did not use a means of decompression status control or dived outside the no decompression limits. Compliance with their own table could not be assessed for the 3 patients whose means of decompression status control was not recorded. Only 19 divers (25%) reported profiles within the limits specified

TABLE 2

THE SOURCE OF DCI REFERRALS TO THE SHU AND MEANS OF EVACUATION FOR ALL CASES

Source of referral to SHU (57)	**Number**	**%**
Local doctor	27	47
Self	17	30
Hospital	6	11
Coast guard	1	2
Dive instructor	1	2
Charter boat crew	1	2
Ambulance	1	2
Other	3	5
Means of transport (76)	**Number**	**%**
Private vehicle	24	31
Fixed wing ambulance	18	24
Helicopter ambulance	16	21
Road ambulance	12	16
Helicopter and fixed wing ambulance	2	3
Other	4	5

TABLE 3

THE MEANS OF DECOMPRESSION STATUS CONTROL EMPLOYED

Means of decompression control	**Number**	**%**
Computer	23	30
PADI Recreational Dive Planner	19	25
US Navy Air Diving Table (including recreational agency derivatives)	7	9
DCIEM Sport Diving Table	6	8
No decompression status control	18	24
RNZN 63 treatment table	1	1
18:60:30 treatment table	1	1
Control unknown	3	4

Note. 3 divers reported a combination of computer and table control of decompression status, the snorkeller is not included, therefore n = 78.

TABLE 4

RISK FACTORS FOR DCI IDENTIFIED IN THE HISTORY

Risk factor	**Number**	**%**
Previous DCI - known	5	7
- suspected	9	12
Repetitive diving	52	78
Second dive deeper than first	28	37
Consecutive days diving	27	35
Strenuous diving	14	18
Equipment failure	3	4
Out of air	11	14
Multiple Ascents	18	24
Rapid ascent	27	36
Flying within 24 hours of diving	9	12
Ascent to >300m after diving (other than flying)	10	13

TABLE 5

FREQUENCY OF PRESENTING SYMPTOMS AND SIGNS

Symptom or sign	**Number**	**%**
Pain	52	67
Fatigue	41	54
Tingling	35	46
Headache	35	46
Numbness	26	35
Weakness	20	26
Cognitive difficulty	19	25
Dizziness	15	20
Ataxia	13	17
SOB	9	13
Itch	7	10
Visual disturbance Rash	6	8
Loss of consciousness	4	5
Cough	2	3
Urinary dysfunction Other	1	1
Other	9	13

by the Canadian Defence and Civil Institute of Environmental Medicine (DCIEM) table. Divers are grouped according to the method/s of decompression status control used in Table 3.

A thorough analysis of other risk factors for DCI has not been performed here since the DIMS database compiled at Adelaide includes these patients and will be reported by Dr Chris Acott in due course. However, a retrospective notes review was conducted to determine the incidence of known risk factors for DCI (other than provocative depth/time profiles). These data are presented in Table 4.

PRESENTATION OF DCI

First symptom latency varied from zero (present on surfacing) to 54 hours after diving, mean 7.3 (SD 21 hours). Musculoskeletal pain was the most frequently reported symptom with headache, fatigue and tingling also common. The percentage incidence of presenting symptoms is

recorded in Table 5. Objective signs were found in 43 patients (57%).

TREATMENT

Fifty nine patients were compressed to a maximum pressure of 2.8 bar (18 m) during the initial treatment. With the exception of 8 patients (see discussion), all of these completed US Navy (USN) treatment table 6[3] with or without extensions. Seventeen patients were treated according to deeper treatment tables after initial compression to 2.8 bar failed to achieve adequate resolution of symptoms and signs. At the SHU where the lignocaine trial protocol[4] is followed, this is defined as less than 80% recovery in subjective symptoms or less than full resolution of objective signs. It should be noted that not all symptoms and signs are "followed" for the purposes of decision making during treatments.[4] The frequencies of use of the various initial treatment tables are given in Table 6.

Daily retreatments with an 18:60:30 table[5] were given until the patient either made a full recovery or experienced no sustained improvement over two consecutive days. Thirty two patients required retreatments. The number of retreatments ranged from 1 to 12 with a mean of 2.6.

One patient suffered central nervous system oxygen toxicity, manifested as a convulsion. The episode occurred 23 minutes into the first oxygen breathing period of a third and final 18:60:30 retreatment. The convulsion resolved spontaneously after which the chamber pressure was reduced to 2.4 bar and the treatment completed on 0_2.

OUTCOME

Fifty seven (75%) of the 76 patients treated for DCI in 1996 were discharged with no sequelae. Omitting 6 cases from the SHU and 2 cases from the CHU in which the diagnosis of DCI was considered highly equivocal, the proportion fully recovered at discharge increased to 81% overall (84% for the SHU; 71% for the CHU). The groups achieving full and incomplete recovery (exclusive of equivocal cases) are compared with respect to a variety of factors postulated to be predictive of outcome in Table 7.

TABLE 6.

THE RECOMPRESSION PROTOCOLS EMPLOYED IN 1996

Treatment table	Maximum depth	Treatment gas	n=
18:60:30	18 msw	O_2	7
USN 5	18 msw	O_2	1
USN 6	18 msw	O_2	51
RNZN 1	30 msw	Nitrox / O_2	2
RNZN 1A	30 msw	Heliox / O_2	7
RN 63	50 msw	Air / O_2	1
RNZN 63	50 msw	Heliox / O_2	4
RNZN 63 Modified	50 msw	Heliox / O_2	3

Discussion

The total of 76 DCI patients represented a decline in annual cases compared with the 100 patients in 1995. The SHU caseload was particularly affected since this unit treated all 100 divers in 1995, whereas 19 of the reduced 1996 total of 76 divers were treated by the CHU. There is no clear explanation for the decline in total cases.

As in 1995, a large proportion of the 1996 patients were trained to Professional Association of Diving Instructors (PADI) Open Water level,[6] and used the PADI Recreational Dive Planner (RDP)[7] to control decompression status. These data reflect the PADI market share in diver training and cannot in any way be interpreted as an indication of a poor standard of training.

An impressive proportion (50%) of divers with DCI reported profiles within the limits of the dive table they were using. Moreover, retrospective calculations revealed that 25% of the reported profiles were within the limits of the DCIEM table. While it is acknowledged that reported profiles are unreliable, these data illustrate that DCI does occur despite adherence to dive tables, a fact that is still poorly appreciated among recreational divers.

The presenting symptoms and signs in the 1996 patients were quantitatively and qualitatively similar to those reported in 1995[2] and in other series.[8] There was a 60 hour discrepancy between the symptom latency (mean 7.3 hours) and delay to presentation (mean 67 hours), although there was a large standard deviation for both parameters. Nevertheless, even if an average evacuation time of 6 hours is allowed, these data indicate that many divers tolerate symptoms for a significant period before seeking help.

Selection of treatment tables at the SHU during 1996 was dictated by the protocol for the lignocaine trial.[4] Initially, this protocol included randomisation to treatment on a heliox or nitrox/air table in the event of the treatment being deepened beyond 2.8 bar, hence 30 and 50 msw nitrox/air tables appear in Table 6. This attempt to retain elements of our earlier "heliox trial" was abandoned because of statistical concerns after only a short period and now all patients requiring deep treatment are treated according to a 30 or 50 msw heliox table. Seven patients were initially treated according to an 18:60:30 table, which is an unconventional approach. In one case, the symptoms of

TABLE 7

NUMBERS OF PATIENTS MAKING COMPLETE AND INCOMPLETE RECOVERY BY DISCHARGE FACTORED AGAINST VARIABLES OF PUTATIVE PROGNOSTIC IMPORTANCE

Variable	Complete Recovery		Incomplete recovery		p
All cases	**n = 55**		**n = 13**		
Age					
≤ 40	45	(80%)	11	(20%)	n.s.
> 40	10	(83%)	2	(17%)	
Gender					
Male	43	(81%)	10	(19%)	n.s.
Female	12	(80%)	3	(20%)	
Delay to onset of symptoms					
≤ 1 hour	29	(81%)	7	(19%)	n.s.
> 1 hour	26	(81%)	6	(19%)	
Delay to presentation					
≤ 24 hours	28	(82%)	6	(18%)	n.s
> 24 hours	27	(79%)	7	(21%)	
Compliance DCIEM tables (n = 65)*					
Yes	15	(88%)	2	(12%)	n.s.
No	37	(77%)	11	(23%)	
SHU Data Only	**n = 43**		**n = 8**		
Objective signs at admission					
Present	21	(75%)	7	(25%)	<0.05
Absent	22	(96%)	1	(4%)	
Previous DCI					
Known or suspected	7	(70%)	3	(30%)	n.s.
None	36	(88%)	5	(12%)	

Note. Eight cases (6 from the SHU and 2 from the CHU) where the diagnosis of DCI was highly equivocal were excluded leaving 68 cases for analysis.

* The two recompression chamber attendants and the snorkeler are excluded from analysis of compliance with the DCIEM table.

decompression illness were minor and of secondary importance to a salt water aspiration syndrome. The treatment was truncated after resolution of symptoms to minimise both patient stress and any pulmonary oxygen toxicity. The others were cases of equivocal DCI where the compression was conducted as a diagnostic manoeuvre.

Nineteen patients (25%) were recorded as having made an incomplete recovery despite recompression therapy. This number fell to 13 (19%) with exclusion of 8 patients for whom the diagnosis of DCI was equivocal. The true treatment failure rate in this series therefore lies somewhere between 19 and 25%. This is similar to failure rates previously reported by the Royal Australian Navy facility at HMAS Stirling during the period 1984-88 (20%)[8] and the SHU in 1995 (30%).[2] It is significantly lower than failure rates in the range 40-60% previously reported by other Australasian units.[1,9-10] Of the factors tested for association with incomplete recovery at discharge, only the presence of objective signs at admission was shown to be significant, although non-compliance with the DCIEM tables and a history of previous DCI generated strong numerical trends. It is notable that these two factors were shown to be significantly related to poor outcome in a previous series of similar size.[11] As in 1995,[2] we have again failed to demonstrate any prognostic significance for delay to presentation. However, this may simply be a reflection of the tendency for the more severe cases to present early.

Three specific cases are worthy of mention.

Case 1

A male of 24 years developed chest pain, dizziness, nausea, and upper motor neurone weakness on the left side including the face while snorkelling. An ECG revealed no evidence of myocardial ischaemia, and a chest X-ray was normal. The weakness almost completely resolved with recompression therapy (table RNZN 63 plus three 18:60:30 retreatments). Unfortunately, the patient discharged himself before treatment was complete and before further investigations could be carried out.

Two cases of DCI arose in nurse chamber attendants after treatment tables.

Case 2

The first followed an uneventful RNZN 63 treatment table. Symptoms, including rash, itch, nausea and multifocal limb pain, arose approximately 30 minutes after completion of the table and were rapidly progressive. Complete recovery was obtained after an extended USN Table 6 and four 18:60:30 retreatments. There was no known predisposition to DCI and a bubble contrast echocardiogram did not detect an inter-atrial shunt. The RNZN 63 table was subsequently modified to include stops between the 50 and 18 msw depths and an extended period of oxygen breathing for the attendant at 9 msw.

Case 3

The second followed an uncomplicated 18:60:30 table, despite the nurse breathing oxygen for the entire ascent. Moderate right elbow pain was noted 5 minutes after completion of the treatment and the nurse was recompressed some 15 minutes later according to USN Table 6. The elbow pain was slow to resolve and the table was extended, resulting in symptomatic pulmonary oxygen toxicity. All pain had resolved by the end of this treatment and there were no sequelae. It was suspected that holding the arm in a tightly flexed position throughout the ascent (to hold the oxygen mask on), with consequent reduction of circulation, may have contributed to this event.

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MAX E NOHL AND THE WORLD RECORD DIVE OF 1937

John R Kane

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December 1, 1937 was a momentous day in the history of diving. Not only was a new world record set at 420 feet; it was also the first significant use of a helium-oxygen breathing mixture in an open water environment outside the confines of a dry hyperbaric facility.

The participants

Max Eugene Nohl was 27 years of age at the time of the dive. He was the son of Mr and Mrs Lee F Nohl (a prominent Milwaukee attorney). In 1929, the year Max Nohl graduated from the Milwaukee University School, he made his first dive in a swimming pool with an open helmet made from a pail. During his college years, he attended the Massachusetts Institute of Technology. Upon his graduation in 1933 Nohl returned to Milwaukee. He later purchased a standard hard hat rig from the friend of a diver who had died in an accident while using it. Nohl began to dive regularly and from this experience he was on his way towards the development of a new diving apparatus.

Dr Edgar End, of the Marquette University School of Medicine in Milwaukee, was the person responsible for calculating the helium-oxygen breathing mixtures and the decompression schedule used for the world record dive. Dr End first met Max Nohl in April 1937.

Captain John D Craig (Danger is My Business book and TV series) also played an important role in terms of financially backing and participating in the experiments conducted at the Milwaukee County Emergency Hospital Recompression Chamber in the months prior to the 420 foot (127 m) dive. Captain Craig and Max Nohl had dived together on a shipwreck (the John Dwight) in the summer of 1935 and had kept in contact with each other to work on new equipment designs since that time.

Experimentation

In 1937, the use of a helium-oxygen breathing mixture for an actual dive was seen as a revolutionary step. However, the actual theory was first proposed nearly two decades earlier. Professor and inventor Elihu Thompson first theorised, in 1919, that helium might be used to replace nitrogen in a diver's breathing mixture, thus avoiding the narcotic effect of air at greater depths. The United States Bureau of Mines and the United States Navy (USN) conducted joint experiments in 1924 with helium-oxygen breathing mixtures. By 1927, the USN was running its own tests at the Experimental Diving Unit in Washington, DC.

In the spring and summer of 1937, Max Nohl, Captain John Craig and Dr Edgar End participated in three experiments conducted by Dr End. The recompression chamber at the Milwaukee County Emergency Hospital was the site of the experiments.

The three experiments were conducted in the same manner. Max Nohl and Captain Craig entered one compartment of the chamber and used rebreather apparatus. Each unit had a spirometer, soda lime canister and a mouth-piece. Max Nohl and Captain Craig would breathe helium-nitrogen-oxygen and helium-oxygen mixtures (premixed) through each apparatus. Dr End was in the other compartment and breathed air during each experiment. He controlled the experiments and observed Nohl and Craig through a small window in the compartment door.

In each experiment the chamber was pressurised to an equivalent of 90 feet (27 m) of water. In experiment 1, Nohl and Craig breathed 21% oxygen, 52.5% nitrogen and 26.5% helium. Dr End breathed air in a separate compartment. The participants remained for one hour at the 90 foot (27 m) level. Because of the reduced nitrogen content, decompression was greatly reduced. Less nitrogen had been absorbed into the divers' bodies. Decompression took only 8 minutes. Because of the reduced nitrogen, it was as if the two divers had been at the 60 foot level. Dr End required a longer decompression due to his breathing air.

In the second experiment Max Nohl and Captain Craig breathed a mixture of 21% oxygen, 26.5% nitrogen and 52.5% helium. Once again the participants remained for one hour at a 90 foot (27 m) equivalent. Dr End wanted to cut the decompression time in half. Decompression was conducted as planned at a uniform rate. Within four minutes Nohl and Craig were back at atmospheric pressure with no side effects.

The third and final experiment employed a mixture of 79% helium and 21% oxygen. After one hour at a 90 foot equivalent, decompression was carried out within two minutes. No ill effects occurred. The only problem noted was the change in pitch of the divers' voices as they breathed helium-oxygen mixtures.

New equipment

The diving equipment used by Max Nohl on his record dive was completely self-contained in terms of its breathing supply. The only connection to the surface was made via a telephone cable and a life line. Both life line and cable could be disconnected at the diver's discretion or cut by the diver if need be in any emergency situation.

The helmet was very sophisticated in terms of its form and design. It was made of polished aluminium and weighed approximately 45 pounds (20.5 kg). No breast plate was to be used. A 360 degree faceplate offered the diver an excellent field of vision in any direction. Into the top of the helmet's interior was fitted a depth gauge, watch, compass, pressure gauges for the back mounted cylinders, and a container for liquid food. The overall appearance of the helmet can be compared to a scaled down lighthouse dome. When using this helmet Nohl wore a leather football helmet. This was to offer protection from bumping his head on the inside of the diving helmet, but also held the earphones in place for communication purposes.

The diving dress was made of rubberised canvas. It was designed to keep the diver dry. The helmet and diving dress were connected together by two metal bands which were drawn tight once the collar of the dress was stretched up over the bottom of the helmet.

The breathing mixture was to be carried in cylinders on the diver's back. A three cylinder unit was available, but only a two cylinder rig was used for the world record dive.

According to Dr End, "In one of the steel cylinders is carried a respirable gas which the diver admits into the suit to equalise increasing water pressure as he descends. The other cylinder contains oxygen which enters the suit at a rate carefully controlled by the diver to satisfy his metabolic requirements."

Inside the diving suit Max Nohl wore an oro-nasal mask which was connected to a rubber device looking like a hot water bottle. This was filled with soda lime to remove carbon dioxide from the diver's exhalations. Valves were arranged so that when Nohl inhaled the breathing mixture would come from what was inside the suit and helmet. When Nohl exhaled the carbon dioxide was effectively removed by passing through the soda lime, while the exhaled helium would reenter the suit to be used again.

One of the great advantages of this design was that Nohl could completely control the flow of oxygen. Decrease it at greater depths and increase it at lesser depths. Therefore, the partial pressure of oxygen could be approximately that of atmospheric air.

On the day of the dive Max Nohl wore a one-piece zippered suit of thick wool. This was complemented by wool socks and mittens. Over the wool undergarment he wore a thin suit of chamois leather.

Assistants help Nohl suit up
in preparation for the record dive.

Standard diving boots were also worn with the diving outfit. Each boot weighed approximately 18 pounds (8 kg). These could be removed by the diver in case of emergency.

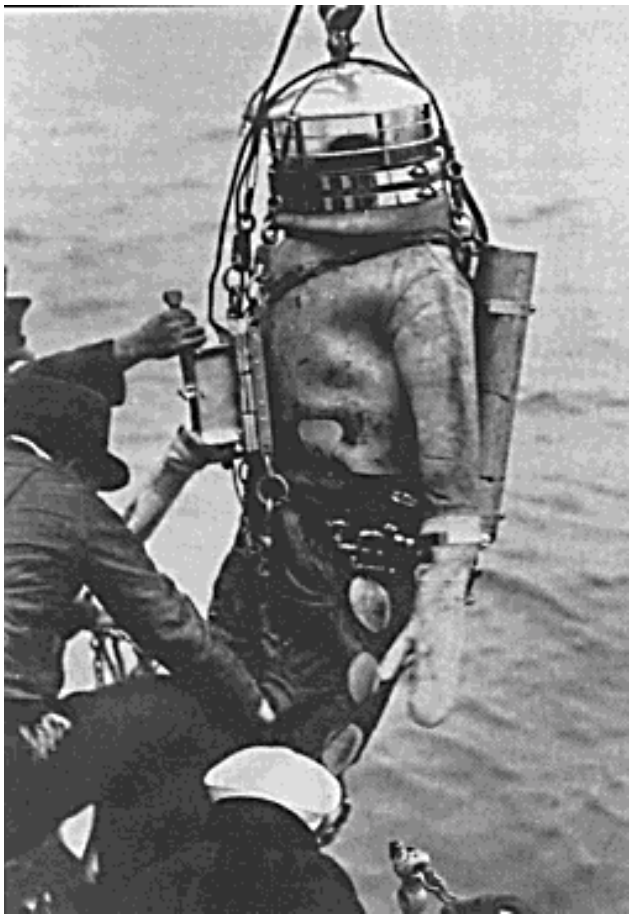
Attached to the outside of the diving suit at chest level was a braking device. Through it passed the descending line. At any time during the dive Nohl could stop or slow his descent.

The cylinders, helmet, and braking device were all chained together to form a harness. At the top of the helmet was a lifting ring to facilitate getting the diver in and out of the water. Once out of the water the diver's weight would be supported by the chain harness.

No weight belt was needed or used with the Craig-Nohl diving dress.

The record dive

During the autumn months of 1937, Max Nohl made a series of dives to test his diving apparatus and Dr End's decompression schedules. Each dive was deeper than the



Max Nohl is lowered over the side of the *Antietam* to begin the dive. Note the braking device on the diver's chest to help retard his decent.

preceding one. By late November, Nohl was confident that he could establish a new world's record.

On Wednesday, December 1st 1937, the US Coast Guard cutter *Antietam* reached its destination by noon. The Lake Michigan dive site was about 25 miles (40 km) north east of Milwaukee and approximately 12 miles (19 km) east of Port Washington, Wisconsin. The commanding officer of the *Antietam*, Lieutenant E C Whitfield, ordered depth measurements to be taken. Both by sounding lead and gauge the depth registered was 420 feet (127 m). This would also be verified later by Nohl's descending line and telephone cable.

Max Nohl began to suit up for the dive and was assisted by Mr Ive Vestrem, an associate, and Mr. Carl Fischer (Chief Engineer of Milwaukee Country Institutions). Dr End watched and was near the diver's telephone. Once ready, a lifting cable was attached to the top of Nohl's helmet and he was swung over the side of the *Antietam* using a lifeboat davit.

Nohl entered the water at 12:50 pm. Within three minutes he had reached a depth of 200 feet (60 m), where he paused to equalise his ears and make adjustments to his breathing mixture.

A slight mishap occurred during this part of the dive. One of the surface crew did not hold onto the telephone cable. The cable continued to feed out on its own weight from the cable already in the water. This was noticed and corrected, but the cable in the water had formed into a large loop. As Nohl resumed his descent he passed through the loop and became entangled. He worked his way to 240 feet (72 m), but this took 26 minutes. Nohl could have cut his telephone cable and continued his descent, but it was thought wiser to return him to the surface and start the dive over again.[20]

At 1:25 pm. Max Nohl re-entered the water after being untangled and reached the bottom without incident within 9 minutes. At the 400 foot (120 m) level he had paused momentarily. Ive Vestrem asked Nohl to come up, but Nohl replied that he had better go as far down as he could. He also said that the temperature was just above freezing and that he could see his white diving suit mittens in front of his helmet.

Moments later (1:34 pm.) Max Nohl reported, "I've hit bottom." The crew on the *Antietam* started to shout and cheer. The cutter's whistle was blown. Nohl spent nine minutes on the bottom, walking and crawling. He was hoping to find a small stone or a rock to bring up, but could not find any due to the clay and mud-like bottom conditions. Nohl was in constant communication with Mr Vestrem and Dr End. Visibility on the bottom was reported by Nohl to be only 5 to 6 inches at best.

At 1:43 pm. the ascent to the surface began. Nohl was raised towards the surface at a slow rate. On the way up he vented excess helium-oxygen from his suit to avoid a blow up. Large bubbles were noticed on the surface as Nohl carried out this procedure. He reached the 200 foot (60 m) level at 1:55 pm. and ten minutes later was at the 30 foot (9 m) level. Nohl's breathing mixture up to this stage of the dive had been 80% helium and 20% oxygen. Once having reached the 30 foot (9 m) level, Nohl vented all helium-oxygen out of the suit and replaced it with pure oxygen. This was also done at the 20 foot (6 m) and 10 foot (3 m) levels as well.

Nohl remained at the 30 foot (9 m) level for 22 minutes and at the 20 foot (6 m) level for 28 minutes. His longest decompression stop was for 46 minutes at the 10 foot (3 m) level. It took Nohl a total of 118 minutes to reach the surface. Dr End was not in a hurry to return him to the surface quickly and considered the decompression schedule used to have a wide margin of safety.

At 3:41 pm. Nohl reached the surface. His helmet was removed and according to the newspaper accounts Nohl's first words were. "What's that funny smell?" His assistants replied, "That's fresh air."

Conclusion

Max Nohl's dive was a great success. His equipment worked very well as did Dr End's decompression schedule. Nohl showed no sign of mental alteration during or after the dive. Having cold feet were his only complaint. Dr. End examined Nohl once he was out of his diving suit and was unable to find anything physically or mentally wrong with the diver.

Once inside the warm cabin of the *Antietam,* Nohl sat down to eat a ham sandwich and sip coffee. He spoke with reporters and smiled for the cameramen as the Coast Guard cutter headed back to Milwaukee.

Max Eugene Nohl had set a new world's record. He had reached a depth that no other diver had ever attained in a flexible diving suit. Most importantly, Nohl and Dr End had clearly demonstrated the practical use and enormous benefits to be gained from helium-oxygen as a breathing mixture for deep diving. The door to the future was now open.

Max Nohl had intended to modify his diving suit following the December 1937 dive. He had also planned to join with Captain John D Craig in an effort to dive the sunken ocean liner "Lusitania", off the coast of Ireland. For reasons unknown the planned dive on the "Lusitania" never took place. This may have been because Max Nohl and Jack Browne, with the help of Milwaukee businessman Norman Kuehn were in the process of founding DESCO (Diving Equipment and Salvage Company) in 1937. In 1960 Max Nohl was killed in an automobile accident which also claimed his wife.

Acknowledgments

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The author

John Kane, who dedicated this paper to the memories of Max E Nohl and Dr Edgar End MD, grew up in Erie, Pennsylvania. He became interested in diving at an early age and received his certification as a scuba diver in 1970. His exploration and study of Lake Erie shipwrecks has continued to the present time. John has a Masters Degree in history, specialising in the history of Japan. He teaches at Campbell University in North Carolina and at Wake Technical Community College in Raleigh, North Carolina. John has an extensive collection of Savoie diving helmets, including a rare flip-up model and a Savoie mixed gas recirculator/$C0_2$ scrubber. His other interests include the study of Japanese history, the history of diving and undersea exploration and other (art related) collecting activities.

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