The Editor's Offering

By the time this reaches our readers the New Year will be well past and most will have converted to writing 2000 instead of 1999. Just before the end of the penultimate year of the second Millennium the International Standards Organisation recommended that shortened dates should be written year/month/day and Standards Australia took this up and issued an Australian Standard to conform with the recommendation. This explains the date format to be found in the reports of Minutes of meetings.

Another change to be seen in this issue is the availability of back copies of the Journal in electronic format. Adobe .pdf documents can be read by almost every computer whatever its operating system as long as it has Acrobat Reader installed. PDF stands for portable document format. These cannot be edited but can be read on screen or printed off. Of course this comes at a cost. While the .pdf documents are much smaller the originals when the originals are large, as the Journal is, the .pdf is large as well. As this is typed this issue is up to 3 MB and that is not the final size. Because of the illustrations the .pdf will be well over 2 MB. This makes it too big for a floppy disc and too big for easy e-mailing. The transmission time is far too long. However there is a solution. Some years ago the Iomega Zip drive was introduced. This can be used with both Windows and Mac operating systems. The great advantage is that the discs hold 100 MB. Of course they don't, the available space is about 95 MB and one is advised not to completely fill the disc. However with the discs being about $30 or less this is the way to get your back numbers to 1993 and copies of the first twelve volumes (1971-1982). We will be able to offer more as the conversion from paper to electronic continues. Luckily Macs accept Windows discs and can put .pdf documents on them. So those who want back numbers should send the Editor a Zip disc formatted for their computer and he will return it with the back numbers. To cover costs there will be a charge of $10.00 for the service. Unfortunately in July, when the 10% GST comes in, the cost will have to rise to $11.00.

The Editor's dream of being able to produce a CD with all the back numbers of the Journal and the Journal Index from 1971 to the year of production looks a lot closer. Only 10 years, of 4 Journals a year, remain to be completed.

The Christmas issue of the Medical Journal of Australia carried a Water Hazards section with eye catching titles: *Dead in the water: how safe are our water sports?* (Robyn Walker); *Patterns of drowning in Australia, 1992-1997* (Ian Mackie); *Snorkelling deaths in Australia 1987-1996* (Carl Edmonds and Douglas Walker); and *Scuba diving medical examinations in practice; a postal survey* (Graham Simpson and David Roomes). The last paper was largely a comparison of what the results of applying the medical from AS4005.1 (Recreational Diving Training) to a series of 15 cases, most of whom would have been referred for specialist opinion or failed according to the Standard, with what 52 doctors, who were on the SPUMS Diving Doctors list, thought should have been done. There were wide variations from the Standard's recommendations, mostly on the side of allowing the trainee to learn to dive. The author's concluding paragraph recommended a "move towards risk assessment and education of the prospective diver, with emphasis on "informed consent", and with the diver accepting responsibility for this or her actions". This shift in SPUMS official thinking took place at the 1995 Annual Scientific Meeting (ASM) on Castaway Island. The problem is that the diving training organisations want a clear yes or no answer and definitely prefer "Yes". The Editor recommends that all those attending the 2000 ASM, again on Castaway Island, should read or reread these Christmas presents as they all bear on fitness to dive.

Last year's Undersea and Hyperbaric Medical Society (UHMS) Annual Scientific Meeting had a whole day devoted to air embolism. The proceedings will not be published but the Journal has been allowed to reprint an article, written by Bill Hamilton (a guest speaker at the 1996 SPUMS ASM), which appeared in Pressure (the Newsletter of the UHMS). This report, which provides a lot of information about air embolism and its occurrence, can be found on pages 55-57. One speaker believed that diver behaviour was a much more important cause of air embolism than lung status. To add information about the topic on page 60 there is the abstract of a recent paper in Undersea and Hyperbaric Medicine (1999; 26 (4); 213-217) on the risk of pulmonary barotrauma in submarine escape training. Although there is a statistically valid association of barotrauma with smaller than predicted forced vital capacity (FVC) "it is insufficiently specific for low FVC to serve as an exclusion criterion for submarine escape training".

In the original papers Bob Wong draws attention to the occurrence of decompression illness in breath-hold divers, an unusual, but not unheard of,
occurrence. Geoff Taylor shares his knowledge and experience of toxic fish spine injury. Placing the affected limb in 45°C water, within an hour of injury, and keeping it at that temperature of 30 minutes gives almost immediate pain relief and uneventful healing. Later treatment usually results in indolent ulceration at the entry wound. Very few punctures require exploration, only those still tender after hot water treatment.

And of course there are the papers from the 1999 ASM by Robyn Walker, Richard Moon, Chris Acott (2) and David Doolette. On page 19 readers will find the provisional program for the 2000 ASM at Castaway Island where the topic is ***DIVING IN THE NEW MILLENNIUM.***

ORIGINAL PAPERS

BREATH-HOLD DIVING CAN CAUSE DECOMPRESSION ILLNESS

Robert Wong

Key Words

Breathhold diving, decompression illness, history, risk.

Summary

The widely held belief that breath-hold divers do not develop decompression illness is incorrect. There is historical and modern (medical) evidence to the contrary.

With the introduction of submarine scooters BH divers can now do deeper repetitive dives that have led to DCI.

While DCI is uncommon with BH diving, cases occur. Breath-hold divers should consult their medical practitioners if they experience symptoms after diving.

With the exception of the aggressive mode of diving, associated with Taravana, of some of the pearl divers from the Tuamotu Archipelago in French Polynesia, most professional BH divers do not experience decompression illness (DCI). This is almost certainly due to the fact that they generally limit their dive times and use longer surface intervals.

Introduction

For centuries, humans have performed breath-hold (BH) diving as a profession. However, since the introduction of compressed air diving, the number of professional BH divers has declined. In northern Australian waters, in the middle of the nineteenth century, mother-of-pearl shell harvesting, for the production of buttons and furniture inlays, was initially done by BH divers. With the advent of compressed air diving in the later 1800s BH divers were replaced by hard hat divers, usually Japanese.[1] At about the same time, in the Aegean Sea, compressed air diving also displaced the traditional BH diving for sponges. In both areas the increased underwater achievements were paid for by horrific incidences of decompression sickness (DCS) when the men worked in deeper water.[2]

In the Tuamotu Archipelago, in French Polynesia, BH diving for pearls was still being carried out in 1957 with a number of divers becoming paralysed or dying after BH dives.[3] More recently the introduction of cultured pearls and the availability of scuba gear has eliminated the need for BH diving in this area. But again at the cost of DCS.

Nevertheless, BH diving for a living still exists in a few places, most notably Japan and Korea. Professional BH divers in Korea are predominantly women (known as hae-nyo for sea women), whereas, in Japan, men outnumber the women (ama for sea women and katsugi for sea men). For convenience they have all been referred to as Amas in most publications.

It is a general misconception amongst most recreational divers, that decompression illness (DCI) does not occur with breath-hold diving, unlike compressed air diving where most divers are vaguely aware of the risk of DCI.

Taravana

In 1958, in the Tuamotu Archipelago, pearl divers used a weight (4-6 kg) to assist their descents to depths of from 9 to over 40 m. The dive time was 30-60 seconds and surface intervals were 3-10 minutes. The working day was some 6 hours. In the Hikueru Lagoon, in one day of diving, 47 (20%) of the approximately 235 divers developed symptoms of “Taravana”. Symptoms described included vertigo, nausea and mental anguish. Some divers became paralysed, either partially or completely. Two fatalities occurred, one underwater after about 18 to 20 dives to 39 m (130 ft). This death may have been due to hypoxia during his ascent.[4] However the other victim was pulled into the boat semi-conscious and died two hours later, which does not fit in the clinical picture of ascent hypoxia. Mangareva pearl divers using the same diving technique but with 12-15 minutes on the surface did not develop Taravana.[3]

Compressed air divers reporting similar symptoms after a dive in Australia would be considered as, at least, equivocal cases of DCI.

The Ama

For centuries, the Japanese and Korean breath-hold divers, generally known as Amas, have been diving without apparent incidence of DCI. This may be because both the depths and the number of dives have been inadequate to produce sufficiently high nitrogen partial pressures to cause DCS. An alternative explanation is that such diving accidents are regarded as part of the job and are not reported due to a general lack of awareness of the problems.

Teruoka published the first scientific study of BH diving in 1932.[5] He described the diving patterns of assisted (Funado) and unassisted (Cachido) Ama. Using a

counterweight, which was hauled up by the boatman when dropped, one Funado descended to a depth of 20-25 m at an average speed of 1.2-1.5 m/sec, but occasionally as fast as 1.8 m/sec. After about 28 seconds on the bottom, the counterweight was dropped and Funado ascended at 1.6 m/sec, being pulled up to the boat. The total time underwater was less than 1 minute. Also described were two Cachido who descended under their own power to 5 -11 m. Both ascent and descent were at about 0.6 m/sec. Travel time took an average of 17.4 seconds for the 5 m dive and 38 seconds for the 11 m dive. Total dive time was 30 seconds for 5 m dive and 45 sec for 11 m dive. Observations on a large group of Ama indicated that the typical dive was for a total of 30 seconds to 5 m and was repeated 60 times in an hour. Exceptional Cachido Ama were able to perform sustained repetitive dives for around 60 seconds each. The Funado Amas managed to perform repetitive dives for 60 second duration to 15-20 m with 60 seconds between dives. Before wetsuits were introduced Amas dived either in minimal briefs or in cotton shirts and trousers.[6]

In 1965 Teruoka's study became available in English when it was reproduced in *Physiology of Breath-hold Diving and the Ama of Japan* by Rahn and Yokoyama.[6] The diving pattern of the Amas was governed by the need for the divers to get out of the water and rewarm at intervals. By the 1980s, when wearing wetsuits had become common among the Ama, more time was being spent underwater as rewarming was not needed so often.[7]

Since the introduction of wet suits and fins, the pattern of diving has remained similar to the that of the cotton suit era. The ascent, however, is about 60% faster with the use of fins. The bottom time is 16.5 seconds for the 5 m dive and 12 sec for the 10 m dive. The use of wet suits has lengthened the working time substantially. By 1985 Amas were diving for 3 hours in summer as opposed to an hour in a cotton suit. In winter they dived for 2 hours a day instead of 30 minutes.[7]

In 1955, well before the introduction of doppler ultrasound, Schaefer observed foam in venous and arterial blood drawn immediately after breath-hold divers surfaced from single dives, lasting approximately 1.5 minutes, to 27 m (90 ft).[8] Subsequent samples drawn 10 seconds after surfacing did not show bubbles. This was a demonstration of a short lived presence of bubbles, presumably due to supersaturation in the blood, after single breath-hold dives.

Lanphier, using theoretical calculations, suggested that enough N_2 could be taken up by the body to cause DCS if deep repetitive dives were separated by short surface intervals.[9]

Doppler studies have demonstrated the presence of venous gas emboli after repeated breath-hold dives in Ama divers.[10,11] However, no cases of DCS had been reported until recently.

In 1992 nitrogen accumulation in venous blood was demonstrated in repetitive breath-hold diving in Korean female divers, however the level of N_2 accumulation was thought insufficient to cause DCS.[12]

In 1998 two cases of multiple cerebral infarctions in Ama divers, who developed neurological symptoms after more than 3 hours of repeated BH diving to 15-25 m, were reported.[13] These divers used a 15 kg weight to assist their descents but ascended unassisted. The dives lasted about 1 minute with surface intervals of 1-3 minutes. Symptoms included euphoria, disturbed conscious state, dizziness, diplopia and nausea.

The incident dive of the first diver, which led to the investigation, was performed in 1990 when, after a series of dives in the morning, the diver complained of diplopia and nausea. About 10 minutes later, he was unable to speak and had weakness and numbness over the right side of the body. His speech recovered after some 20 minutes, but the hemiparesis and paraesthesia persisted for about 1 and 4 weeks respectively. Two weeks after the incident, he was transferred to a hospital for investigation. The delay was because the diver lived a long distance away from any hospitals. CT scan showed no abnormalities but MRI demonstrated 2 small lesions, one in the left internal capsule and the other in the left frontal lobe.

The second diver performed similar dives and had done so for 20 years. His incident dive occurred in 1986. After diving for 4 hours continuously, he took a break for lunch, but within 10 minutes, he developed hemiparesis and sensory disturbances over his left side. Within 2 hours he was unable to walk and within 3 hours he lost consciousness but recovered gradually over several hours. Four years later, he reported to hospital for examination. Physical examination revealed no abnormalities, but MRI showed lesions in the subcortical and subependymal areas.

While the only evidence of their previous problems are the MRI lesions, the clinical histories of rapid onset neurological problems soon after a series of deep immersions and the slow but complete recovery suggest that DCI was the likely diagnosis. There are numerous such case histories to be found in Paul Bert's *Barometric Pressure*.[14] Although these 2 divers developed DCI in 1986 and 1990, their problems only recently came to the attention of knowledgable medical practitioners.

Perhaps, DCI is far more common in BH diving than many doctors believe, but has not been reported because it is accepted as part of the job. In 1974 a study of 301 out of 400 Japanese compressed air, shell-fish divers, living in an isolated village, was published.[15] Between 1966 and 1968 "each year three to five men died from accidents or from decompression sickness. 'The bends' were very common; they almost ignored them, thinking them unavoidable and treating them with baths and by drinking alcohol". If these

compressed air divers phlegmatically accepted paralysis as a frequent, unavoidable occurrence in their lives, as they did, it seems likely that BH divers might accept the occasional occurrence of symptoms after a day's diving in the same way.

This supposition is supported by Kohshi et al.'s interviews with the breath-hold divers from the same village. Eight of 15 divers had previously experienced some neurological disorders during or after a dive.[13] The divers did not consider their symptoms as abnormal and had not sought medical treatment, which was, in any case, not available in their remote villages.

A year later Kohshi et al. reported two more BH divers who experienced neurological symptoms.[16] Again both dived with a 15 kg weight but with unassisted ascents. The dives were to 15-25 m for 1-1.5 minutes with 1 min surface intervals. They usually dived for 5 to 6 hours. One diver, who experienced dizziness and blurred vision, had a MRI performed 4 days later which showed 2 cerebral infarcts. The 2nd patient, 39 years old, was admitted to hospital, after similar dives, with a mild hemiparesis and numbness on the right side. He had also experienced left hemiparesis after breath-hold diving at the ages of 17, 25 and 27.[16] Three cerebral infarcts were demonstrated on MRI. The fact that this diver had experienced and recovered from 3 incidents of hemiparesis since the age of 17 suggests that he had accepted this paralysis as a "normal" consequence of diving and so had not sought medical treatment.

Again in Japan, Kohshi et al. interviewed 15 divers on the island of Mishima, Yamaguchi Prefecture.[17] Seven admitted to having experienced previous diving accidents, with unilateral weakness and sensory disturbances. Eleven divers experienced dizziness, nausea and/or euphoria. None experienced the joint pains commonly seen after compressed air dives. One diver had lost consciousness, which is most likely to have been the result of hypoxia of ascent. Most of the divers spent 4 -5.5 hours in the water. Depths were to between 10-15 m, with some to 20-30 m. They averaged 20 to 30 dives per hour, but did up to 40 dives per hour in shallower water (8 to 12 m). The onset of symptoms was always immediately on reaching the surface.

The above reports are reminiscent of the symptoms reported by Cross.[3] Compressed air divers reporting similar symptoms after a dive in Australia would be considered as, at least, equivocal cases of DCI.

Recreational divers

There are many breath-hold recreational divers who spear-fish and do repetitive dives. Medical attention has been focussed on those who hyperventilate before the dive and die from hypoxic unconsciousness, followed by drowning, on their way to the surface.[4] Other recreational divers attempt BH dive records, although they do not do repetitive dives. Nonetheless, great depths have been achieved. In October 1999 Umberto Pelizziri set a world record for sled-assisted free diving by reaching 150 m.[18] These deep dives last around 3 to 3.5 minutes. These durations expose the divers to the hazards of hypoxia on ascent.

Two BH divers who experienced nausea, vertigo and headache, neither of whom complained of joint pain, were diagnosed as DCI by Wong.[19] One was recompressed with complete resolution of symptoms. The other recovered over time without treatment.

There is a new sport in the Mediterranean Sea off the Balearic Islands. Here there are many BH divers who regularly make use of submarine scooters to achieve quick descents and ascents. Some have dived to 63 m (210 ft) and back in a matter of 2 or so minutes, with surface intervals of 2 minutes or less. They do between 15 to 20 dives per hour for between 3 to 8 hours. Batle reported 25 cases of neurological DCI in such divers whose symptoms appeared immediately on surfacing.[20] They were all treated using either USN Table 5 or 6 with complete resolution of symptoms. The depths of such dives using submarine scooters and the rate of ascent would suggest that supersaturation would occur and produce bubbles on ascent.

Magno et al. described 6 separate incidents of neurological problems which occurred in four BH divers.[21] The dives included a single deep weight and buoyancy assisted dive to 120 m (400 ft), 3 assisted dives to depths ranging from 35-90 m (115-300 ft) and multiple unassisted dives to 25-30 m (89-100 ft) over a period of 2-4 hours. Symptoms and signs appeared within minutes of surfacing and included hemiplegia, ataxia, dysarthria, diplopia and colour blindness. Some were recompressed whereas others received no therapy, nonetheless, all made complete recovery.

Fanton et al. described a spear fisherman who did a series of dives to 40 m consisting of some 14 dives per hour for 3 hours.[22] He was unconscious on surfacing. With resuscitation and oxygen administration he woke up but exhibited slow mentation and disorientation. This is unusual with anoxic unconsciousness of ascent in the absence of near drowning. Subsequent EEG and MRI showed focal cerebral injury. The diver admitted that he had experienced several episodes of weakness in the arms during earlier diving activities. These cannot be explained by ascent hypoxia.

While the above cases affected divers who performed deep repetitive dives, Bayne and Wurzbacher described a BH diver who twice attempted to swim across a 25 m (25 yard) pool at about 1.8 m (6 ft) deep.[23] After the second

attempt, he surfaced complaining of headache, dizziness and tingling all over. He also complained that "his lungs were hurting". Minutes later, he suffered a Grand Mal fit, became pulseless and apnoeic. Autopsy showed bleeding under the visceral pleura, mediastinal emphysema and large amount of air in the right heart and cerebral vessels (the left heart was not opened at autopsy). Bruch has also reported 2 divers who developed mediastinal emphysema during BH diving to 4.5 m (15 ft).[24] Such cases are seldom mentioned in diving medicine textbooks.

Personal communications, as yet unpublished, include a diver who experienced headache, dizziness, blurred vision, numbness and weakness of all four limbs after repetitive BH dives over a three and half hour period to a depth of only 8 m and a BH diver who did repeated dives for some 4 hours to depths of 6-8 m and complained of tiredness, headache, joint pain and paraesthesia in his upper limbs. This diver was treated in a recompression chamber with resolution of all symptoms.

Breath-hold diving after compressed air diving

Paulev, a submarine medical officer, described his personal experience.[25] After spending 8 minutes at 20 m as an attendant in a recompression chamber, he performed a number of repetitive BH dives in the submarine escape training tank to depths of 20 m for about 5 hours. He suffered from nausea, dizziness and belching. Later, he also developed pain in his left hip, the right knee, the right arm was weak and the right arm felt tired. There was also paraesthesia and blurring of vision. He was recompressed and made a full recovery. Three similar cases of BH diving following previous hyperbaric exposure also presented with symptoms of DCI and were similarly treated with complete resolution.

Discussion

It is a general misconception, and a myth, that DCI does not occur with BH diving. It is probable that symptoms are often ignored or misinterpreted, both by the divers and by any doctors who were consulted, because of the general lack of appreciation of the pathophysiology of this condition.

However, the cases mentioned above show that, under certain circumstances, DCI is possible after BH diving. Hemiplegia has occurred after a single breath-hold dive.[26]

Given the right circumstances (repetitive dives for prolonged periods and short surface intervals, perhaps with excessive ascent rates), it is possible for the body to absorb sufficient inert gas to cause supersaturation and bubble formation on ascent. Short surface intervals also do not allow time for the body to eliminate much inert gas between dives.

In the cases above the following factors were present

1 repetitive dives in excess of some 3 to 4 hours;
2 depths of dives were generally in excess of 15-25 m;
3 surface intervals were generally short and
4 ascent rates were usually rapid.

In the cases of CAGE, one would have to assume that during the dive, air was trapped in a closed off part of the lungs before ascent. It is hypothesised that air trapping during diving can cause local distension of the lung and lung rupture on ascent producing air embolism.[27]

It has also been suggested that forceful inhalation to total lung capacity (TLC) might cause lung rupture by over-distension of a weak area in the lung without pressure changes.[28]

Conclusions

BH diving is not as benign as one would like to think.

Decompression illness, both DCS and CAGE, can occur with BH diving. The incidence is considered to be low. In most cases DCS is due to a combination of repetitive deep diving, short surface intervals and fast ascents. Since it is reputed that 30% of the population might have a Patent Foramen Ovale, it could be considered surprising that few BH divers present with symptoms after repetitive dives. The low rate of symptoms is probably because few breath-hold divers seek medical attention for symptoms which commonly disappear spontaneously.

Deep repetitive BH diving immediately after compressed air diving can cause DCI.[24] Breath-hold diving, to recover an anchor, after completing an appropriate decompression for a compressed air dive has led to sudden death on more than one occasion.[28]

It is highly recommended that any diver who experiences unusual symptoms after BH diving should seek medical advice, as recompression can speed their recovery if the symptoms are due to DCI.

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TOXIC FISH SPINE INJURY LESSONS FROM 11 YEARS EXPERIENCE

Geoff Taylor

Key Words

Injury, marine animals, toxins, treatment.

Abstract

The most important intervention in toxic fish spine injury is the application of heat. This should be continued until the pain is relieved and for a minimum of 30 minutes. Heat treatment applied within one hour of injury prevents the indolent ulceration usually follows these injuries. Local anaesthetic infiltration and exploration are usually unnecessary, except with injuries to toes, and should only be done if pain and tenderness persists after heat treatment.

Introduction

The town of Exmouth (Latitude 21° S) on North-West Cape is close to the Ningaloo Marine Park and is a popular tourist destination, especially in winter months. The author was a General Practitioner in the town for 11 years from 1982 to 1993. Boating, fishing and diving are popular recreations.

Toxic fish spine injuries were extremely common, and an almost weekly occurrence, especially during the winter tourist season. Many patients presenting with severe pain and a puncture wound were unaware of the cause. The majority of the cases, approximately one patient a week presenting at the local hospital, were presumed stingray spine injuries to the feet and ankles. Some patients had felt the slimey feel of the fish under their feet or had seen the ray. Other fish spine injuries were from fish caught on a line or handled in a trawl net. These included injuries from the Scorpion fish family (*Scorpaenidae*) such as Butterfly Cod, Bearded Ghoul, Bull Rout; Rabbit Fish (*Siganidae*) and Cobbler or Catfish (*Siluroidei*) also provided patients. These injuries were normally to the upper limb, usually the hand or fingers. Many cases of simple fish spine penetration were also seen but will not be discussed here.

Two cases of Stonefish injury were also encountered and will be discussed separately.

Toxic fish spine injuries

With stingray spine injuries, the commonest presentation was of a puncture wound on the dorsum of the foot or around the ankle, which was sometimes bleeding. The patient complained of severe local pain. There were no cases with systemic symptoms. There were some more unusual presentations, such as a baby that had crawled across the lawn impaling a detached stingray spine in the knee, which had then penetrated further with every movement to a depth of one centimetre. There was also the case of a prawn trawler crewman who in trying to extricate a large stingray from the trawl net, was stabbed by the massive spine in the back of the thigh, severing his sciatic nerve.

Experience has shown that the toxin injected with these injuries is heat labile and will be destroyed by heat treatment with hot water. In my earlier years in Exmouth patients were managed according to the recommendations of Edmonds.[1] Heat treatment was applied and the wounds explored, under local anaesthetic, for the presence of a foreign body.

In 1982 a protocol was drawn up for management of stingray spine injuries at the local hospital and a file started in the hope of conducting a study into the effectiveness of treatment. It was anticipated that patients would self-select themselves on the basis of the time delay from injury to presentation for treatment.

Unfortunately for the project, but not for the patients, nursing staff became so proficient at treating these injuries that medical help was not always requested. As a result follow-up of these injuries was not routinely organised and the study did not eventuate. However over an 11 year period much experience was gained. While the author cannot present accurate statistics, his experiences are reported here. He also gained considerable personal experience of such injuries and the efficacy of the treatment recommended below.

Management

The initial management of all foot and hand injuries was to immerse the whole affected part in water at approximately 45° C. Edmonds[1] recommended a temperature of 50° C, but it was found that most patients could not put their feet into water above 45° C because it felt too hot. This treatment brought almost immediate relief of pain in most cases and analgesia was rarely needed. Treatment was usually initiated by nursing staff. The hot water needed to be topped up or replaced approximately every 10 minutes to maintain an adequate water temperature. Experience showed that heat treatment was necessary for at least 30 minutes, after which pain recurrence was rare. With a shorter duration of heat treatment, pain recurred when the limb was removed from the hot water.

In the earlier years, many wounds were explored. Local anaesthetic was infiltrated and the wound probed to determine if a foreign body was present. Infiltration with local anaesthetic brought immediate relief of pain in most

instances. Exploration seldom produced any foreign bodies. In later years exploration was restricted to cases where experience showed that a foreign was likely. The reasons for this change in policy are discussed below.

Results

As anticipated, patients self-selected themselves into two groups, those requesting immediate treatment and those who delayed. All patients who presented early, within approximately one hour, had a satisfactory outcome from their treatment. Their puncture wounds healed without sequelae.

This was in marked contrast to those patients where there was delay in initiating heat treatment. These people usually developed ulceration at the puncture site, which often took several months to heal. Antibiotic therapy had little influence on these ulcers.

Embedded stingray spines

It was rare to find stingray spines broken off in the wound. No spines were found in the wounds around the ankle or in the body of the foot nor were spines found in situ with toxic spine injury to the hands.

Spines were only found in wounds of the toes. Pulling these embedded spines out through the entry puncture is very difficult because of the barbs. The spine must be advanced and removed through an incision over its tip, so creating a second wound. Wounds which continue to be tender after the initial pain has been removed by heat treatment awere considered an indication for exploration. However this was rearely necessary.

Stonefish envenomation

Two cases of stonefish envenomation were seen. Both had occurred in a mangrove creek where the species was common and a positive identification was made in the second case.

The first case was a 16 year old girl who presented screaming and hysterical from the severity of the pain. She had impaled her big toe on the spine of a fish in the water. There was a puncture wound on the tip of the toe. The whole toe was cyanosed and swollen, and there was oedema of her foot. Large doses of pethidine failed to control her pain. A ring block was inserted in the toe and Stonefish antivenene was administered, with good effect. She was discharged next day and did not suffer sequelae.

The second case was a 35 year old man who trod on a stonefish wearing thick-soled boots. A spine penetrated the sole of the boot producing a shallow puncture wound. He was treated with heat (hot water) treatment and analgesia. There was no sign of local or systemic spread of the toxin and he did not require antivenom.

Discussion

Toxic Fish Spine injury is a common occurrence throughout the north of Australia. There is significant morbidity from inadequate treatment of such injuries, the patient suffering a festering ulcer that refuses to heal for several months.

Exploration of most injuries was found to be unnecessary. Heat treatment of these wounds, by immersing the affected part in hot (45°C) water, is of paramount importance. Although infiltration of local anaesthetic brings immediate relief of the pain, once the wound is anaesthetised, it is impossible to "titrate" the duration of heat treatment against the patient's pain. It is vital that, if local anaesthetic is infiltrated, the heat treatment should continue for at least 30 minutes.

Heat treatment itself is successful at relieving pain in almost all instances, and should be continued until the patient can remove their limb from the hot water without recurrence. Most patients could not stand treatment with water above 45°C on their feet, but heat tolerance was better in the hands.

The presence of persistent tenderness beneath the puncture wound, after the initial pain has been removed by heat, indicates the need for exploration. Exploration is more often required with injuries to the toes, where it is much commoner that spines are broken off. It is essential in cases where spines are removed, and pain has been relieved by local anaesthesia, that the heat treatment is also carried out to prevent the later indolent ulceration.

Stonefish spine injuries are distinguishable by the extreme severity of the pain and the local effects around the wound, swelling and cyanosis, which occur rapidly. While the toxin of Stonefish may be heat labile, it spreads rapidly, and antivenom treatment is usually required to prevent long-term sequelae.

References

1 Edmonds CE. *Dangerous Marine Animals of the Indo-Pacific Region*. Newport, Victoria: Wedneil, 1975; 24-78

Dr Geoff Taylor's address is 6 Park Way, Busselton, Western Australia 6280. Phone/Fax +61-(0)8- 9754-2772. E-mail <jaqtayl@iinet.net.au>.

THE WORLD AS IT IS

LINE DANCING AND THE BUDDY SYSTEM

Bob Halstead

Key Words
Buddies, diving safety, solo diving.

I recently saw a TV promotion for a country music festival. A special attraction was a horse that was shown line dancing in step with a family group all togged up in cowboy gear. I think viewers were meant to be astonished at how smart the horse was and rush off to see this phenomenal animal perform.

To me the horse looked quite ordinary, perhaps a bit bored. I wondered just how smart the line dancers were. We sometimes forget that, by definition, half the population have an IQ less than 100 but this does explain line dancing, TV soap operas and Queensland Workplace diving legislation.

Before you get too upset I have to say that divers are usually quite smart simply because they have to pass a sort of intelligence test in order to get certified. That is correct I think , isn't it PADI? . . . . NAUI?. . . .NASDS? . . . .Hello, where are you?

Well I might as well go the full Monty and offend everyone. I do not know whether it is because I am 190 cm tall and so have a clearer view of the world, but I do tend to see things differently. The Jabiluka mine is just a big clean up operation as far as I am concerned, what would worry me is if they were putting nuclear material into the place instead of taking it out. I consider that pet dogs and cats are colonial vermin, we Australians should be going for walks with Skippy on a lead not Fido. I also think that all religions should be made to prefix their absolute pronouncements with the phrase "It is highly unlikely that this it true, and it is really quite whacko, but we believe ..... "

The reason I am rambling on here is that a very silly diving practice has risen from the dead and is being touted again. This is the practice of buddying inexperienced divers with experienced divers. The argument is of course that inexperienced divers are vulnerable (true) and that they therefore will be much safer paired with experienced divers (doubtful). This ignores three things, one, how much less safe this makes the dive for the experienced diver, two, this is instruction NOT using an instructor, and three, it assumes human nature is different from what it actually is.

In other words, "They're Dreamin'!"

I know that the uneven buddy system does not work because I have tried it. The experienced diver either has to sacrifice a dive to care for the inexperienced diver, or leads a dive too advanced for the inexperienced diver. If you actually go diving regularly you know that each dive is incredibly valuable. Generally you have to expend a lot of time, effort and money to get to do a dive and even then nature can work against you with rough seas and poor visibility. The thought of finally successfully getting to the dive site and having to baby-sit a beginner, is an unnatural act. The theory assumes that people behave in good and unselfish ways, and like I said, "They're Dreamin'!"

We train Instructors and Dive Masters to teach, and look after, beginner divers. It is a job, a tough and skilful one at that, and people get paid to do it, as they jolly well should, this is no place for amateurs.

I am no great admirer of the buddy system. It is one of the great mistakes, along with no-decompression diving, that we made in the early days of diver training. We should have ditched it years ago and instead promoted self-sufficient diving and surface support. Alas, it is too late now, the legal risks are too great for instructor organisations and Governments to make the changes.

Nevertheless, if you are going to buddy, it is essential that the buddy be of equal standard and interest. In fact I first defined buddy diving many years ago as follows:-

The buddy system is the situation which occurs when two divers of similar interest and equal experience and ability share a dive, continuously monitoring each other throughout the entry, the dive and the exit, and remaining within such distance that they could render immediate assistance to each other if required.

I am flattered that several authors have since borrowed this definition. Many people seem to think that just by putting two divers together they create a buddy system, but a little thought will expose this for the stupidity it is.

After observing some near catastrophic so-called buddy dives, I put my own theory into practice and always tried to buddy inexperienced divers with other inexperienced divers. It worked a charm, the divers did not dive deep, they did not stray far from the boat, they did not have unrealistic expectations about their buddy's ability to rescue them, they surfaced from the dive proud of their own achievement and eager to learn and gain more experience.

I also allowed experienced divers to solo dive, although it must be said this does not mean diving alone

because we always provided excellent surface lookouts and rescue capability. Solo diving was, and is, extraordinarily popular with experienced divers, and particularly marine life underwater photographers, since it allows close experiences with many wild marine animals which would swim away when confronted with pairs or groups of divers. It is an interesting fact that ONLY solo divers ever had close contact with wild dugongs during all the time I was running our dive boats Solatai and Telita.

Mike Ball Dive Expeditions is the only Australian dive operator who can honestly say he caters for all styles of diving at his training facility in Townsville and on board his live aboard fleet, Paradise Sport in PNG, Spoil Sport and Water Sport out of Townsville and Super Sport out of Cairns. If you are a beginner you can get instruction from well qualified professional instructors. If you are qualified but inexperienced you can get a dive guide who is a qualified Dive Master or Instructor to take you diving and be your buddy. That is their job and they get paid for it, you will not be spoiling their dive. If you wish to buddy dive and have a suitable buddy, and the crews will help you meet with like minded divers on board if you came alone, you may do so. And if you are an experienced diver and have a redundant spare air supply such as a pony bottle with independent regulator, you may solo dive.

This is incredible service dedicated to giving divers the best possible experience commensurate with their ability and interest. It provides choice instead of treating every one at the lowest common denominator, and it promotes excellence rather than mediocrity. Mike Ball is a smart operator and I do not think you will ever find him line dancing, with or without a horse.

Reprinted, with some modifications, by kind permission of the Editor from DIVE LOG 1999; 132 (July): 52-54.

Bob Halstead is a diving instructor and provided wonderful diving in New Guinea waters from the Telita for many years. His address is PO Box 141, Earlville, Cairns, Queensland 4870, Australia. Phone +61 (0)7 4095 8155, Fax +61 (0)7 4095 8156.
E-mail <halstead@internetnorth.com.au>.

The above paper has been published to encourage debate about diving practices. Readers are encouraged to write to the Editor with their comments, not only on the buddy system but about all diving procedures.

SPUMS

ANNUAL SCIENTIFIC MEETING 2000
will be held at
Castaway Island, Fiji from May 6th to 14th 2000

Guest speaker Professor David Elliott

Convenors are Drs Vanessa Haller and Guy Williams.

Members wishing to present papers should contact
Dr Haller at
55 Two Bays Crescent,
Mount Martha, Victoria 3934.

The travel agent is Allways Dive Expeditions.
168 High Street
Ashburton, Victoria 3147, Australia

Tel +61-(0)3-9885-8863
Toll Free 1800-338-239
Fax +61-(0)3-9885-1164
E-mail allways@netlink.com.au

HTNA Y2K

8TH ANNUAL SCIENTIFIC MEETING ON DIVING AND HYPERBARIC MEDICINE

Mercure Inn, Brisbane, Australia

Presented by the
Hyperbaric Technicians and Nurses Association
with the
Australian and New Zealand Hyperbaric Medicine Group

Guest Speakers will include

Professor William Zamboni, Las Vegas, Nevada
Dr Simon Mitchell, Brisbane, Australia
Valerie Larson-Lohr, San Antonio, Texas
Richard Durnford, Seattle, Washington

For further information contact HTNA
Phone +61-(0)7-3371-6033
Fax +61-(0)7-3371-1566
E-mail <htna.y2k@wesley.com.au>

"FIT TO DIVE"

A 2-day meeting on
MEDICAL ASSESSMENT OF FITNESS TO DIVE
is being arranged by Biomedical Seminars
in association with The Medical Subcommittee of the
European Diving Technology Committee
at
The Royal Society of Medicine, London
8th & 9th April, 2000

In 2000, the annual "Fit to Dive" meeting will be held at the Royal Society of Medicine. With the subsequent retirement of the organisers, Nick McIver and David Elliott, this may be the last in this series that has survived 20 years and so we are planning it to be the best yet.

Five years have passed since the Biomedical Seminars meeting "Medical Assessment of Fitness to Dive" which was sponsored by the HSE in Edinburgh. It was a significant step towards new medical guidance on fitness assessment which was then issued to all HSE Approved medical examiners of divers. The new Diving Regulations in the UK have ensured that these annual medical assessments are applied to the wide range of working divers, from those in the offshore oil and gas industry to diving scientists and the professional instructors of recreational divers.

The Edinburgh meeting recognised that there was no great place for pass/fail criteria but that each diver needed individual assessment related to their work and that, to achieve this, the judgement of the medical examiner is paramount. Since then, training objectives for diving doctors have been approved by the European Diving Technology Committee. This 2-day meeting is for all medical examiners of divers including those Approved in the UK by the HSE. It will focus on areas of continuing controversy, such as late onset diabetes in relation to the established but inevitably ageing diver. The resumption of diving after illness, injury, surgery or a diving-related incident will be highlighted as perhaps the examiner's most challenging assessment. The medical subcommittee of the European Diving Technology Committee hopes that the output of this meeting will lead to a greater international harmonisation of standards.

The academic program will start at 0900 on Saturday 8th and end at 1700 on Sunday 9th April, 2000. The Registration fee of £180 (if paid before 31 December) will include lunch on both days. From 1 January registration will be £210.

Further details from
BIOMEDICAL SEMINARS, 7 Lyncroft Gardens, Ewell, Surrey, KT17 1UR, England.
Telephone (+44) 181 393 3318 : Fax (+44) 181 786 7036
E-mail: <Karen@biomedseminars.demon.co.uk>

UNDERSEA AND HYPERBARIC MEDICAL SOCIETY MEETING IN STOCKHOLM

June 18th to 22nd 2000

The Undersea and Hyperbaric Medical Society (UHMS) will hold its 2000 Annual Scientific Meeting in conjunction with the 3rd Karolinska Postgraduate Course: Challenges in Faciomaxillary Reconstructive Surgery from June 18-22th. The meetings will be held at the Stockholm Conference Centre, Stockholm, Sweden.

The UHMS portion of the program will include a program of symposia, poster sessions, original papers and discussion. One of the goals of this meeting is to integrate the fields of hyperbaric and diving medicine. A joint introductory lecture will be scheduled each morning followed by a panel discussion. Topics will include: regulation of the microcirculation in health and disease, reperfusion injury and new aspects of oxygen toxicity.

The Karolinska Postgraduate Course will have a program dealing with radiation effects on bone and soft tissue, the role of oxygen in problem wounds, evidence-based hyperbaric medicine in treatment and prophylaxis, advances in molecular biology and nerve regeneration, ischaemia-reperfusion injury, osseointegrated implants, resorbable osteosynthesis and other sciences, strategies and controversies in jaw reconstructive surgery.

For those interested in diving physiology, Dr David Elliott will chair a one and a half day pre-course on Acute Management of Decompression Accidents. The course will be held on 18-19th June. The course will cover current controversies: the assessment and classification of diving casualties; immediate treatment and options; recompression on site versus evacuation to distant hospital-based chambers; the consequences of delay; recompression algorithms and treatment failures; treatment results and audit; and post-decompression dysexecutive syndrome.

Immediately following the meeting, there will be a four-day post-congress symposium on Diving in Cold Waters. The symposium will be held on an island close to Trondheim, Norway, and will give participants both practical and theoretical experience in the particular problems related to cold water diving.

Further information may be obtained from
Jane Dunne
Undersea and Hyperbaric Medical Society
10531 Metropolitan Avenue
Kensington, Maryland 20985-2627
Tel. +1-301-942-2980 extension 102.
Fax +1-301-942-7804.
E-mail: <jdunne@ix.netcom.com>, www.uhms.org or
www.ki.se/org/oxygen .

DIVING MEDICAL CENTRE
Scuba diving medical examiner's course

A course for doctors on diving medicine, sufficient to meet the Queensland Government requirements for recreational scuba diver assessment (AS4005.1), will be held by the Diving Medical Centre at:

Bond University
Gold Coast, Queensland.
Easter weekend 2000.

Previous courses have been endorsed by the RACGP (QA&CE) for 3 Cat A CME Points per hour (total 69)

Phone Brisbane (07)-3376-1056 for further details

Information and application forms for courses can be obtained from

Dr Bob Thomas
Diving Medical Centre
132 Yallambee Road
Jindalee, Queensland 4047
Telephone (07) 3376 1056
Fax (07) 3376 4171

ROYAL AUSTRALIAN NAVY MEDICAL OFFICERS' COURSE IN UNDERWATER MEDICINE

November 27th to December 8th 2000

The course concentrates on diving physiology, fitness to dive, and emergency management of diving injuries.

Practical involvement includes opportunity to dive with different types of equipment and a recompression chamber dive.

The course fee for 1999 was $1,330.00. The 2000 fee is expected to be about the same plus GST but is yet to be determined.

For information or to enrol contact

Officer in Charge
Submarine and Underwater Medicine Unit
HMAS PENGUIN
Middle Head Road
Mosman, New South Wales 2088

Tel: (61) 2 99600333
Fax: (61) 2 99604435
E-mail : <Robyn.Walker.150150@navy.gov.au>

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.

Key Words
Qualification.

MINUTES OF THE 1999 ANNUAL GENERAL MEETING OF THE SOUTH PACIFIC UNDERWATER MEDICINE SOCIETY

held at Layang Layang Island Resort
on 7/5/99 at 1500 local time.

Present

All members attending the Annual Scientific Meeting.

Apologies

Tim Wong, David Davies, Des Gorman, Gareth Long.

1 Minutes of the Previous Meeting

The minutes of the previous meeting have been published (SPUMS J 1998; 28 (4): 198).

Motion that the minutes be taken as read and be accepted as an accurate record. Proposed Dr Ian Seppell, seconded Dr John Knight. Carried.

2 Matters Arising from the minutes

None.

3 President's Report

This was printed on pages 207 and 208 of the December 1999 issue.

4 Secretary's Report

This was printed on page 208 of the December 1999 issue.

5 Annual Financial Statement and Treasurer's Report

The Financial Statement is still in the hands of the auditor and will be published when available.

Treasurer's report (presented by Dr Robyn Walker in the absence of the Treasurer, who was unable to attend the meeting owing to injury.) summarises the total Revenue and Expenditure and includes 1997 and 1998 comparisons for the various accounts operated by the Society. There has been a 17% increase in the income of SPUMS, and a 26% increase in expenses.

Total income for 1998 was $136,811.79, a 17% increase. Of this, 45.5%, or $62,277.10 of the Society's income came from membership subscriptions from 1,200

members. 38% was derived from the 1998 SPUMS ASM at Palau. The conference resulted in a profit of $4,356.23.

Total expenses for 1998 were $139,544.25, a loss of $2,742.46, compared to a profit of $14,824.30 for the previous year. The 26% increase in expenditure was mainly due to increased costs of the Journal printing, $7,389.54, and secretarial costs of the Editor, Secretary and Treasurer of approximately $3,900.

Although as a single category the major expense was the 1998 ASM, with costs of $49,557.82; in fact the major SPUMS expense is the cost of Journal production, which was $59,160.22 in 1998.

SPUMS ACCOUNT BALANCES

Access account	
Opening Balance 1 January 1998	$33,170
Closing Balance 31 December 1998	25,973
Loss	-7,200
Investment Account	
Opening Balance 1 January 1998	$80,789
Closing Balance 31 December 1998	75,000
Loss	-5789
ASM Account	
Opening Balance 1 January 1998	$1,740
Closing Balance 31 December 1998	11,985
Credit	10,245

Closing balance includes registrations for the 1999 ASM.

In total, the Closing Balance of SPUMS accounts was a positive balance of $112,957.40, a loss of $2,742.46 compared with the balance in 1997.

SPUMS held a successful Scientific Meeting in Palau and continued to publish the Journal. Membership has continued to increase, and the subscription rate has been pegged at $100 for full and corporate members, and $50 for associate membership. For 1999 there will again be an expected increase in the Society's expenditure. There will be upgrades in the Secretary's computer system; purchase of the multi-media projector used at the 1999 meeting; upgrade and development of the SPUMS database; website development; and purchase of a new computer system for the newly created position of the SPUMS secretariat.

Finally I would like to thank the Committee and members of SPUMS of the past two years for their help with the challenging position of Treasurer. Unfortunately, due to personal and professional pressures, I will not be seeking a third term, but will be more than happy to serve on the committee of SPUMS.

Tim Wong
Treasurer of SPUMS

Motion that the Treasurer's report be accepted. Proposed Dr John Knight, seconded Dr Chris Acott, carried.

6 Subscription Fees for the forthcoming year

The Treasurer recommended that the fees remain the same, at $100 for members, $50 for associates and $100 for corporate members.

Motion that SPUMS membership fees be $100 for members, $50 for associates and $100 for corporate members. Proposed Dr Robyn Walker, seconded Dr Neil. Carried.

7 Election of Officer Bearers

This year there was an election to choose three Committee Members from the five candidates. All other nominations were unopposed.

The SPUMS Committee for 1999 will be

President	Robyn Walker
Secretary	Cathy Meehan
Treasurer	Peter Dupont
Editor	John Knight
Education Officer	David Griffiths
Past President	Guy Williams
Public Officer	Guy Williams
Committee Members	Chris Acott
	Simon Mitchell
	Douglas Walker

8 Appointment of the Auditor

With the change of Treasurer, our Treasurer's residence is moving from Brisbane to Adelaide. As yet, there has not been time too appoint a new auditor in Adelaide, however we will advise the membership when the process has been completed.

9 Business of which notice has been given

Alterations to the **SPUMS Statement of Purposes and Rules**.

Under the heading **Definitions**

Alter rule 2.(a) by changing the words *30th June* to *31st December*.

Under the heading **Committee**

Insert new rules

21.(d) The Australian and New Zealand Hyperbaric Medicine Group is a Sub-Committee of SPUMS.

21.(d) (i) Its members must be members of the South Pacific Underwater Medicine Society Incorporated.

21.(d) (ii) Its Chairman shall have a place on the Committee.

Under the heading **Officers of the Committee**

Alter rule 22.(a) by adding the words, *the Chairman of the Australian and New Zealand Hyperbaric Medicine Group* after the words the New Zealand Chapter of the South Pacific Underwater Medicine Society Incorporated.

22.(a) will then read

The Committee shall consist of a President, Immediate Past President, a Secretary, a Treasurer, Public Officer, the Editor of the Journal, an Education Officer, a representative appointed by the New Zealand Chapter of the South Pacific Underwater Medicine Society Incorporated, *the Chairman of the Australian and New Zealand Hyperbaric Medicine Group* and three other members of the Association entitled to vote.

22.(b) to be renumbered 22. (d) this reads

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.

22.(c) to be renumbered 22. (e) this reads

In the event of a casual vacancy in any office referred to in sub-clause (a), the Committee may appoint one of the Association's members entitled to vote to the vacant office and the member so appointed may continue in office up to and including the conclusion of the annual general meeting next following the date of that person's appointment.

Insert new rule

22.(b) All officers of the Association, except those detailed in 22.(c), shall be elected by postal ballot if the number of candidates exceeds the number of vacancies.

Insert new rule

22.(c) The Editor, the Public Officer, the representative of the New Zealand Chapter of the South Pacific Underwater Medicine Society Incorporated and the Chairman of the Australian and New Zealand Hyperbaric Medicine Group shall be appointed to their positions. The first two by the Committee, the others by the New Zealand Chapter of the South Pacific Underwater Medicine Society Incorporated and the Australian and New Zealand Hyperbaric Medicine Group respectively.

Under the heading **Publications and Publicity**

Alter rule 41 by adding the words *The Chairman of the Australian and New Zealand Hyperbaric Medicine Group is the Association's official spokesman on Hyperbaric Medicine matters.* after the first sentence.

Rule 41 will then read

Public statements in the name of or on behalf of the Association shall only be made by the President, Secretary or by another member of the Association specifically designated by the Committee to speak on any particular matter. The Chairman of the Australian and New Zealand Hyperbaric Medicine Group is the Association's official spokesman on Hyperbaric Medicine matters.

Insert new heading **Board of Censors**

Insert new rules

42. The Committee shall appoint a Board of Censors

42 (a) The Board of Censors shall be composed of the Education Officer, the President of the Society and a Director of a Hyperbaric Medicine Unit in Australia or New Zealand.

42 (b) The role of the Board of Censors is to advise the Committee on all matters of education in diving and hyperbaric medicine.

42 (c) A Diploma of Diving and Hyperbaric Medicine may be awarded by the Society, on the recommendation of the Board of Censors, to a member who fulfils the requirements set down by the Board and published in the SPUMS Journal from time to time.

Motion that the constitutional changes as proposed and printed above be accepted. Proposed Dr John Knight, seconded Dr Ian Seppell. Carried.

Meeting closed at 1524.

Key Words

Constitutional amendments, meeting.

A NEW WAY TO WRITE THE DATE.

In the year 2001 confusion between the short dating conventions will increase and remain until 2032 when the year will be a higher number than the longest month so making its position quite clear. It is confusing enough that the United States uses month, day, year while much of the world uses day, month, year. For thirty one years no one is going to be able to be certain that the year really is the last entry unless we adopt the method laid down in AS/NZS 3802, which is identical with the international standard ISO 8601.

In these standards the year (in full) comes first, then the month (two digits) and then the day (again two digits). This is described as YYYYMMDD by the standard. In this way dates logically follow each other. In current Australian practice many computers store dates by the first number of the day. Using American practice dates are sorted by the first number of the month. Neither method allows easy tracking of dates by year.

From now (2000/1/2) on the SPUMS Journal will be using the international standard for dates.

MINUTES OF THE SPUMS EXECUTIVE COMMITTEE MEETING

held in Adelaide after
the Hyperbaric Technicians and Nurses Association
(HTNA) meeting
on 1999/8/29

Opened at 0900

Present

Drs R Walker (President), G Williams (Immediate Past-President), C Meehan (Secretary), P Dupont (Treasurer), J Knight (Editor), D Griffiths (Education Officer), C Acott, S Mitchell, D Walker (Committee Members), M Bennett (ANZHMG Representative), V Haller (Co-convener 2000 ASM).

In attendance

Mr Steve Goble (SPUMS administrator).

Apologies

Dr M Kluger (NZ Representative).

1 Minutes of the previous meeting (5th and 7th May 1999)

Moved that the minutes be accepted as a true record. Proposed Dr John Knight, seconded Dr Mike Bennett, carried.

2 Matters arising from the minutes

2.1 Indemnity Policy Update.
The indemnity policy has been finalised and is being held by Dr Guy Williams. A copy of the policy will be held by the Secretary. The policy will be reassessed yearly

2.2 Job description of the Convener.
This has been written and circulated. It will be discussed at the next meeting. It will be available to members electronically or printed.

2.3 Protocol for lending of Audiovisual Equipment.
Requests should be made to the President or the Secretary.

2.4 Update on the SPUMS website.
The Diving Doctors List (DDL) will be posted on the SPUMS Home Page in the near future. Overseas members will be listed. The list will be updated every year when subscriptions are renewed and changes can be made at any time. When the DDL is available on the web the fact will be announced in the Journal. Printouts of parts of the DDL will be available from the Administrator, who will take on some of the responsibilities of maintaining the site. It is intended that the SPUMS Diving Medical will be available on the website.

2.5 Revision of the SPUMS medical.
The medical was finally revised and will be distributed with the December Journal.

2.6 All risks insurance policy for SPUMS equipment.
The Secretary will compile a list of all SPUMS owned equipment, with serial numbers, for the insurers.

2.7 Update on the ANZCA SIG, and ANZHMG.
Dr M Bennett informed the Committee that there would be another meeting of the SIG in Cairns at the Scientific Meeting of the Australian Society of Anaesthetists. A course in diving and hyperbaric medicine is planned at the Prince of Wales Hospital in 2000.

2.8 Hand over to the new Treasurer.
Dr P Dupont has recently been handed the laptop computer and paperwork. He will finalise a report of the ASM 1999 Layang Layang account. In future the Treasurer will produce a quarterly financial report.

2.9 Board of Censors.
The Board consists of three members, the Education Officer (Dr David Griffiths), the President of SPUMS (Dr R Walker) and the Head of an Australasian Hyperbaric Unit (currently Dr Mike Davis). The board will formalise the conditions that apply to the "SPUMS Grant".

2.10 Job conditions of the administrator.
This is being attended to.

2.11 Updating the new database.
The Administrator (Steve Goble) has been correcting the database and will make any changes he sees necessary.

2.12 It was decided to have the SPUMS administrator attend the ASM in an official capacity. He would be assist the convenor and work the audiovisual equipment starting with the 2001 ASM in Madang, PNG. SPUMS would only pay for his airfare and accommodation.

3 Annual Scientific Meetings

3.1 1999 ASM, Layang Layang.
Final figures for profit and loss are still to be provided.

3.2 2000 ASM, Castaway Island, Fiji.
All is proceeding well and the conference is already heavily booked. There has been a further call for abstracts.

3.3 2001 ASM, Madang, PNG.
Geoff Skinner, of Allways Travel, has carried out a site inspection and the venue is suitable. It was suggested that the theme could be "Respiratory issues: The Lung and Diving." It was been suggested that, if possible, a suitable dive guide be found in Cairns to reduce travel costs. Malaria prophylaxis and travel vaccinations will have to be advised.

4 Treasurer's Report

No Treasurer's report was available owing to the late hand over.

5 Correspondence

5.1 Letter from Dr Jen Ch'ng.
The Secretary to reply.

6. Other Business

6.1 SPUMS affiliation with the HTNA.
The Committee approved starting these negotiations.

6.2 Index Medicus listing of the Journal.
An application for this listing has been submitted and will be considered in October.

6.3 GST.
The Treasurer is to investigate and report to the next meeting on the effects, on SPUMS, of the recent GST legislation.

6.4 Confidential Investigation proposed. Dr D Walker to present a protocol to the Committee.

6.5 Dr M Bennett briefed the Committee of the campaign to persuade UHMS to hold its 2003 Annual Scientific Meeting in Sydney.

6.6 The next face-to-face Committee meeting will be held at the Brisbane HTNA meeting in 2000.

6.7 It is hoped that the Treasurer's audited report for 1998 will be available at the next Committee Meeting.

Closed at 1400

MINUTES OF THE SPUMS EXECUTIVE COMMITTEE TELECONFERENCE

held on 1999/12/19

Opened at 0800 Eastern Standard Time

Present

Drs R Walker (President), G Williams (Immediate Past-President), C Meehan (Secretary), P Dupont (Treasurer), J Knight (Editor), D Griffiths (Education Officer), C Acott, D Walker (Committee Members), M Bennett (ANZHMG Representative) and V Haller (Co-convener 2000 ASM).

In attendance

Mr Steve Goble (SPUMS administrator).

Apologies

Drs S Mitchell (committee member) and M Kluger (NZ Representative).

1 Minutes of the previous meeting (1999/8/29)

Moved that the minutes be accepted as a true record. Proposed Dr John Knight, seconded Dr Peter Dupont, carried.

2 Matters arising from the minutes

2.1 Job description of the Convener.
This has been written and circulated. It will be discussed at the next meeting. It will be available to members electronically or printed.

2.2 Protocol for lending Audiovisual Equipment.
Requests should be made to the President or the Secretary in writing. Conditions of lending are held by Dr R Walker.

2.3 Update on the SPUMS website.
The DDL will soon be posted on the website and will be updated as required. The SPUMS dive medical will also go onto the website. Mr Steve Goble will take on the responsibility of keeping the website up to date.

2.4 Revision of the SPUMS Medical.
This has been completed and will be distributed with the December Journal. Dr C Meehan gave a brief overview of the recent Standards Australia meeting AS 4005. This draft is being finalised.

2.5 All risks insurance policy for SPUMS equipment.
All committee members need to send full details of electronic equipment to the Secretary in order to insure them.

2.6 Board of Censors.
Dr D Griffiths has informed the Committee that, due to his work load, he is unable to continue as education officer. Dr R Walker is making inquires as to another suitable candidate. The Committee thanked Dr Griffiths for all his hard work to date. Dr Griffiths informed the committee that he would be posting in his resignation.

2.7 Job conditions of the administrator.
This is being attended to by Dr R Walker.

2.8 Update on the Journal.
Correspondence from the Executive Editor of MEDLINE/Index Medicus was discussed. Unfortunately the SPUMS Journal has not been successful in its request to be indexed by the National Library of Medicine. The Editor will explore other indexing agencies.
The Journal index for 1999 will soon be added to the index available on the SPUMS home page, which will then cover 1971 to 1999. In time it is hoped to have all back numbers of the Journal available as "pdf" documents and to produce a CD bearing both the index and all past Journals.

2.9 GST.
Because SPUMS has an income of over $50,000 SPUMS has not only to pay GST but also to register for GST. In view of the GST liability a 5% cost was added to the renewal subscription to cover the GST payable on the subscription portion from July to December 2000. As SPUMS will be a GST registered organisation, GST charged by its suppliers can be reclaimed from the Federal Government. SPUMS must apply for a business number. The Treasurer is attending to all issues involved.

2.10 AGM minutes were held over to the next meeting.

3 Annual Scientific Meetings

3.1 1999 ASM, Layang Layang.
Final figures for profit and loss are still to be provided.

3.2 2000 ASM, Castaway Island, Fiji.
All is proceeding well and the conference is already heavily booked. There has been a further call for abstracts. There are still many delegates with outstanding registration fees. It was noted that no booking is confirmed until the registration fee has been paid. Dr P Dupont will transport the Audiovisual equipment from Adelaide.

3.3 2001 ASM, Madang, PNG.
It was suggested that the theme could be "Respiratory issues: The Lung and Diving." Suitable guest speakers will be discussed at the next teleconference. It is essential that the timing of the meeting is such that it does not clash with other relevant meetings. A suitable dive guide in Cairns has been suggested and Dr Williams will make contact with him shortly. Malaria prophylaxis and travel vaccinations will have to be advised. A discussion was entered into with regard to the reliability of the airline schedules.

3.4 Future ASM.
Dr M Bennett requested that SPUMS consider what involvement it would envisage in the event of a Sydney UHMS meeting. This was to be further discussed at the next meeting.

4 Treasurer's Report

No Treasurer's report was available at this time. An auditor in Adelaide has been found.

5 Correspondence

5.1 Discover Scuba for Children with Disabilities. Although SPUMS as an organisation cannot be involved, assistance has been given to locate a suitably qualified medical practitioner who will undertake the health assessments.

5.2 Proposal from Dr Henrik Staunstrup with regard to distributing the SPUMS Journal to members of the Danish Aerospace and Diving Medical Society. The Committee agreed that a suitable fee would by $25 per issue for each member of the Danish society receiving the Journal. Dr J Knight will reply to Dr Staunstrup.

6. Other Business

6.1 BSAC and UK Sport Diver Medical Form discussed. There are some significant differences between this and the SPUMS medical.

6.2 The New Zealand Chapter of SPUMS is inactive. Letter from Dr M Kluger, Chairperson of the Chapter, presented to the committee. Dr R Walker will reply. If the chapter is dissolved any monies will be sent to the Treasurer. The future of the library will be discussed.

6.3 Letter from Mr Terry Cummins requesting consideration for full membership of SPUMS. Notice has been given for this to go before the membership at the AGM in May 2000.

Closed at 1000
Dr V Haller and Mr S Goble departed at 0815
Dr G Williams and Dr C Acott departed at 0915

ERRATA

Edmonds C. Snorkel diving: a review. (*SPUMS J* 1999; 29 (4): 196-202)

Reference 3 was printed as "Edmonds C and Walker D. Australian snorkelling deaths. *Med J Aust* 1999, In press".

It should now be read as

3 Edmonds CE and Walker DG. Snorkelling deaths in Australia. *Med J Aust* 1999; 171: 591-594.

Owing to an Editorial error a table was omitted from the same paper and is printed below.

CAUSES OF SNORKELLING DEATHS

	Before 1987	**1987-1996**
Numbers	90	45
Average age	30	45
Male	98%	75%
Female	2%	25%
Drowning	52%	45%
Cardiac	3%	30%
Hypoxia	10%	20%

NOTICE TO MEMBERS AND ASSOCIATES

The December 1999 Journal was sent to over 1,100 Members and Associates.

At the end of the first week of March 2000 only 795 Members and Associates had renewed their subscriptions.

<u>This is the last Journal that will be sent to those who do not renew before the end of May 2000.</u>

2000 ANNUAL SCIENTIFIC MEETING
PROVISIONAL PROGRAM

DIVING IN THE NEW MILLENNIUM

Monday May 8th

Professor David Elliott (Guest Speaker).
Why Fitness ?
Dr Robyn Walker (Royal Australian Navy).
Long-term Effects on Divers.
Drew Richardson (PADI).
Title to be announced.
Dr Henrik Staunstrup (Denmark).
Yo-yo diving II.
Dr Guy Williams (Victoria).
Case Studies.
Dr Vanessa Haller (Victoria).
Case Reports.

Tuesday May 9th

Professor David Elliott (Guest Speaker).
Fitness for what ?
Dr Paul Langton (Western Australia).
General Health of Divers.
Mr Chris Coxon (Health and Safety, Queensland).
Resort Course Medicals.
Dr Mike Bennett (Prince of Wales Hospital).
Medical Support of a Major Tunnel Project.
Dr Mike Logan (Dubbo, New South Wales).
Living on Compressed Air.
Dr Malcolm LeMay (Western Australia).
Vision.

Wednesday May 10th

Professor David Elliott (Guest Speaker).
In-water Restrictions for the So-called Unfit.
Dr Simon Mitchell (Wesley Hyperbaric Unit, Brisbane) and Dr Lynn Taylor (New Zealand).
Diabetes and diving.
Dr David Taylor (Melbourne).
Hyperbaric Effects of Pseudoephidrine and Antihistamines.
Dr Jürg Wendling (Switzerland).
Screening for Patent Foramen Ovale.
Dr Rees Jones (Whangarei, New Zealand).
Title to be announced.
Mr Geoff Skinner (Allways Dive Expeditions).
A Decade of Dive Mastering.

Thursday May 11th

Professor. David Elliott (Guest Speaker).
Some Fitness Case Histories.
Dr Paul Langton (Western Australia).
Prevalence of Asthma in Scuba Divers.
Dr Paul Thomas.
Measurement of Peak Flow in the Hyperbaric Chamber.
Dr Guy Williams (Victoria).
Case Presentations and Panel Discussion.
Dr Mike Davis (Christchurch, New Zealand).
Case Studies.
Mr Graham Pollard (South Australia).
The Abalone Industry

Friday May 12th

Professor David Elliott (Guest Speaker).
Resumption of Diving after a DCI Incident.
Professor Des Gorman (Auckland, New Zealand).
Health Surveillance in the 21st Century.
Dr Mike Davis (Christchurch, New Zealand).
Breath-hold Divers.
Dr Jürg Wendling (Switzerland).
Disabled or Handicapped Divers.
Panel Discussion on Fitness to Dive and
Formulation of Policy Statements.

Saturday May 13th

Annual General Meeting.

JOURNAL BACK NUMBERS

All Journals from 1993 to 1999 are now available as PDF documents, designed to be read by all computers using Acrobat Reader, which is free from the Adobe web site.

Unfortunately the documents are too large to be sent over the internet or on floppy discs. However send a Zip disc, which holds 100 MB, formatted for your computer to the Editor and it will be returned with the Journals on it. If reader's require 1999 back numbers send two Zip discs as the six years will not fit on one disc.

LETTERS TO THE EDITOR

THE NEW ZEALAND CHAPTER OF SPUMS: IS THERE A FUTURE ?

From the Chairman and Secretary/Treasurer of
The New Zealand Chapter of SPUMS

2000/1/15

Dear Editor

The role of a separate New Zealand Chapter of SPUMS, with its own meetings, has recently been questioned.

There appears to be great inertia among members of the New Zealand Chapter of SPUMS to become involved in activities in New Zealand. The hugely successful 1997 meeting of SPUMS in New Zealand, organised by Mike Davis, was supported by few registrants from New Zealand. Local annual meetings have had between 6-15 attendees.

Over the past 12 months we have tried to organise a meeting in Tutukaka and asked for expressions of interest. Basically there has been none. What does this mean for SPUMS members in New Zealand? Presumably, most consider the trip overseas to the SPUMS Annual Scientific Meeting and/or the Journal to be their main reason for membership.

Now the time has come to discuss where we should go from here. We would be grateful for any feedback from the New Zealand members on this issue. We would put forward the following questions.

1 Do you want a separate New Zealand Chapter of SPUMS?
2 If YES, what do you want from it?
3 If NO, what should we do with the assets in our account?

Please contact us either by phone or e-mail. If we have not had any positive replies by the middle of the year then we will wind up the New Zealand Chapter of SPUMS and send all assets to the SPUMS main office in Australia.

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BOOK REVIEWS

HYPERBARIC OXYGEN: WOUND HEALING, SAFETY, COST EFFECTIVENESS

Bakker DJ, Péchon JC and Marroni A. Editors.
Holywell Neopren – Hyperbaric Medical Centre, Belgrade, Yugoslavia.
202 pages, hardcover, 1998.

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

Price from Best Publishing Company $US 39.50. Postage and packing extra. Credit card orders may be placed by phone on +1-520-527-1055 or faxed to +1-520-526-0370. E-mail <divebooks@bestpub.com>.

The main title of this book might suggest that it is a text with a major focus on the role of hyperbaric oxygen in wound healing. It is, in fact, the proceedings of the First European Workshop on Hyperbaric Medicine, organised under the auspices of the European Committee for Hyperbaric Medicine and run in Belgrade in association with the First International Postgraduate Course of the International Postgraduate School of Baromedicine at the University of Belgrade.

Three separate workshops were held, one on each of the hyperbaric medicine themes that make up the title. The first, "Oxygen and Wound Healing," focussed upon oxygen physiology and the measurement of oxygen tension in tissue with only two clinical papers which provide a review and recommendations regarding hyperbaric oxygen and its roles in wound healing and in post-traumatic infection. The second workshop considered "Safety at Work in Hyperbaric Medicine" with presentations providing a variety of international perspectives. This section concludes with the European Committee for Hyperbaric Medicine's proposed recommendations for the Prevention of Fire in the Hyperbaric Environment. Unfortunately for readers without a sound grasp of the French Language, these are printed in the original French, an odd inclusion for a book that is otherwise in English but perhaps understandable in the context of using the Workshop Proceedings as a means of disseminating a set of draft recommendations to the European hyperbaric community. Workshop III addressed "Cost-Effectiveness of Hyperbaric Oxygen Therapy". This potentially most important section is the shortest at 23 pages and rather disappointing, incorporating very brief overviews of the current state of health economics in the UK and USA

with only Marroni and Oriani presenting papers which attempt to put figures to the cost effectiveness question, based upon Italian practice. Sadly, 6 of the 7 tables referred to in Oriani's presentation appear to have been omitted from the text.

The three sections of this book are likely to interest different audiences. There is some useful resource material in the first section for clinicians and wound healing researchers while those responsible for hyperbaric safety will find the second section a worthwhile addition to what is a very limited body of literature available on a vital subject.

This Workshop gathered together a range of eminent speakers with a great deal of experience in their respective fields. As usual, there are perspectives to be gained from the overviews presented in workshops such as this which cannot be gained from reading journals or textbooks. The transcripts of the question and answer sessions that followed each presentation enhance this. Nevertheless, the specialist appeal of this book is likely to limit its distribution to the libraries of major hyperbaric units and those whose survey of the literature must be complete.

Ian Millar

Key Words

Hyperbaric oxygen, medical conditions and problems, physiology, safety, treatment.

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HYPERBARIC FACILITY SAFETY:
A Practical Guide
Edited by Wilbur T Workman.
ISBN 0-941332-76-4. 1999. 754 pages.
Best Publishing Company, P.O.Box 30100, Flagstaff, Arizona 86003-0100, U.S.A.

Price from the publishers $US 128.00. Postage and packing extra. Credit card orders may be placed by phone on +1-520-527-1055 or faxed to +1-520-526-0370. E-mail <divebooks@bestpub.com>.

Tom Workman was one of the guest speakers at last year's Hyperbaric Technicians and Nurses Association. He has produced this large volume of useful information about how to organise and run a hyperbaric facility safely. In the process he has gathered 40 contributors, most of whom live and work in the USA but authors come from South Africa (2), Great Britain, Germany (3), Australia (2), Mexico and Japan. As with all multi-author books there are highs and lows. But the highs outweigh the rather dully written lows. The book is divided into seven sections. Section 1, General Considerations, covers chamber types, considerations of design and regulations. This last covers USA regulations in four chapters, European standards in three, Japanese, SW Pacific, South African and Latin American standards get a chapter each and there are three chapters on the international regulation of hyperbaric chambers. Most of the rest of this section is devoted to staffing and training in various regions. One thing which seems to be repeated again and again is the lack of government regulation, and sometimes government opposition, of minimum standards of safety and training. The world view outside the US is wide but unfortunately no contributors come from Russia or China, both of which have many hyperbaric facilities. All in all Section 1 gives a comprehensive view of the state of the hyperbaric chamber around the world.

Sections 2 (Hyperbaric Systems), 3 (Hyperbaric System Maintenance) and 4 (Fire in the Hyperbaric Environment) are full of clear, factual chapters written by people who know their job very well indeed and write from experience.

Section 5, Hyperbaric Mishap Analysis, shows clearly that careful design, well thought out procedures and detailed training are the cornerstones of avoiding mishaps. Some layouts have been accidents waiting to happen and they have happened. Not insisting on a complete change of clothing before entering the chamber has been responsible for at least three chamber fires and allowing children to take sparking toys into a chamber has resulted in two chamber fires. All were fatal. The Royal Adelaide Hospital based HIMS (hyperbaric incident monitoring study) is clearly described. This anonymous, voluntary reporting system is a replica of the system used in military aviation and in anaesthesia in Australia. It has no numerator but reflects the pattern of incidents and shows clearly where improvements in procedures are required.

Section 6, A Practical Approach to Hyperbaric Safety, is a summation of all that has gone before an a practical guide for every hyperbaricist on how to keep out of trouble, except perhaps with financial controllers who object to the costs of preventive maintenance. A useful chapter covers the evaluation of equipment and materials for use in an oxygen environment. Attention is drawn to the need for regular testing of the fire extinguishing system and to the problems that this causes. A number of these systems have failed to function when the need arose. The cause was almost always something blocking some of the piping or the nozzles. Unfortunately the system is designed to wet the chamber thoroughly and no one likes to take a working chamber out of action by filling it with water! The final section is potted biographies of all the contributors.

There is no doubt that this is an expensive book, and that better editing would have removed a number of puzzling typographical mistakes (such as connect for

cannot on page 340), but overall the collection of all this information between two covers will be extremely useful for anyone who wishes to run a hyperbaric facility safely. It is a practical guide and should be read, at least in part, by everyone who works in a hyperbaric unit.

John Knight

Key Words

Accidents, hyperbaric facilities, oxygen, safety.

DOCTOR AND PATIENT:
EXPLORING CLINICAL THINKING

Ken Cox
ISBN 0 86840 505.
1999. 1235 x 153 mm PB. 320 pages.
UNSW Press, University of New South Wales, Sydney, New South Wales 2052, Australia.
RRP $Aust 39.95

Ask anyone, they will tell you; Medicine is an Art, a Vocation and a Calling. It is a topic shrouded in mystery and concealed by layers of arcane language. It is a subject that is so complex and so difficult to master that only our most academically-gifted students, after many years of intensive training and examination by equally-gifted teachers, may eventually be deemed fit to practice the Ancient Skills of History Taking, Examination, Diagnosis and Treatment.

Of course, the reality is that the vast majority of each year's batch of eager new interns and RMOs are little better prepared for the actual Practice of Medicine than were the generations of their predecessors. I know that I was by no means alone when I found myself let loose in the wards with terrifyingly little understanding of how to actually *use* the astonishing amount of knowledge that I had painfully accumulated whilst an undergraduate. As Professor Cox notes, in one of the many succinct aphorisms that pepper this very readable book, "Medical school examinations concentrate on knowledge rather an *application* of that knowledge in the delivery of care".

As products of that system, most of us have probably had very little opportunity or encouragement to explore the thought processes involved in eliciting a history, performing an examination, deciding what further tests or advice might be helpful, working towards a clinical diagnosis, and ensuring that our patient is fully involved at each stage of the process. As time passes we (hopefully) get more efficient at these techniques. Specialisation, and sub-specialisation, helps by narrowing down the range of options we have to worry about. Over the years, trial and error helps us learn the cues, clues and critical issues that are important in our own area of clinical practice.

Professor Cox, a surgeon by trade, realised early in his career that much of the "mystery" of Medicine was, in reality, needless *confusion*. In 1976 he established the School of Medical Education at the University of New South Wales (where he also held the Chair of Surgery from 1969 to 1993). For the past several decades he has been engaged in the exploration of how doctors and patients actually think, and how discrepancies in their individual perceptions of illness can be understood and repaired.

In the course of his teaching he has always laid great emphasis upon the need for doctors to understand "where they are coming from". Running throughout this book are many examples of the ways in which a doctor's own background and personality can influence behaviour, interpretation and the outcome of the medical interaction. As Professor Cox methodically leads the reader through the processes of problem exploration, diagnostic formulation and treatment/management decisions, he relies upon examples rather than argument to emphasise his points.

It is a fitting tribute to Professor Cox's communication skills that he has presented his material in a way that is equally accessible and engrossing to readers from both a medical and a non-medical background. The fancy medical terms are still used (when necessary), but a couple of explanatory words are usually all that it takes to place the jargon in context and make it accessible to all. It provides an effortless example of how easily "our" language can be used to explain rather than to impress or confuse the listener.

Although this is a very readable book, it is certainly not light reading. There is much to pause and ponder. It is full of stories that trigger the odd personal embarrassing memory or two, and occasional insights that suddenly make other recollections fall into place. I found it a delightful and thought-provoking book that should be fascinating reading for anyone who has ever stopped to wonder exactly how we often get it right but occasionally get it so very wrong.

Sadly, undergraduate and post-graduate trainees (possibly those liable to gain most from this book) are also those most likely to put it aside for later reading. Although it contains invaluable information that will help them practice Medicine more safely and effectively, it fails to qualify on that most imperious of study demands, "Will there be any questions on this in the exams?" Of course the answer is "No". And thus one can be sure that we have not seen the last generation of medical graduates who spend the first few years of their professional lives in amassing a phenomenal volume of knowledge and the rest of their careers in discovering exactly what to do with it! Professor Cox's book convinced me that there is an alternative.

John Couper-Smartt

Key Words

Communication, general interest, medical conditions and problems, training.

IN-WATER RECOMPRESSION

48th Workshop of the Undersea and Hyperbaric Medical Society.

Edmond Kay and Merrill P Spencer, Chairmen and Editors. Undersea and Hyperbaric Medical Society, 10531 Metropolitan Avenue, Kensington, Maryland 20895, USA. Published 1999.

Price from the publishers $US 20.00. Postage and packing extra. Credit card orders may be placed by phone on +1-301-942-2980 or faxed to +1-301-942-7804. E-mail <uhms@uhms.org>.

This 48th Workshop of the Undersea and Hyperbaric Medical Society, was held on 1998/5/24 (to use the new international standard for dates) as a tribute to the late Dr Ed Beckman. Carl Edmonds opened the batting by describing the Australian underwater oxygen treatment for DCS, introduced in late 1960s to treat seriously affected divers in remote areas, many, many hours flying time away. Frank Farm from Hawaii presented statistics from Hawaii where many divers had three or four hour boat trips to shore so chose to treat themselves at sea using air, with a remarkably high recovery rate seeing that increasing paralysis seems to have been necessary for the diagnosis of DCS. Commander J M Chimiak, MC USN, whose first name escaped the editors, spoke on the USN and NOAA routines for in-water recompression. The USN considers that hyperbaric oxygen, using a closed circuit oxygen rebreather is the method of choice if in-water recompression (IWR) has to be carried out. However the USN Diving Manual states that "recompression in the water should be considered an option of last resort, to be used only when no recompression facility is on site and there is no prospect of reaching a recompression facility within 12 hours". Drs John Hardman and Leticia Smith presented evidence that immediate in-water recompression (IIWR) of neurological DCS has a high success rate, enough for them to "believe that safe IIWR protocols can now be used for the treatment of divers suffering from decompression sickness". Their definition, based on experimental work, of "immediate" was "within 30 minutes".

So far three speakers definitely for in-water recompression and one accepting that it might have a small place. The next speaker, Paul Webb, gave a wide ranging and very interesting discussion of heat loss and its effects. It is perfectly possible for well wet suited working divers to lose considerable amounts of heat, enough to interfere with mental function but which does not produce a low core temperature.

As with all workshops the discussion period after the morning's presentations was illuminating. Some clinicians wanted to know how many of the cases of IWR described had been supervised by a doctor. This in spite of mention of remote sites 12 or more hours away by air or 3 or 4 hours by boat from the shore. The cases described are very seldom seen by doctors because they are treated long before a doctor could reach them. This also applies to oil rig diving. Treatment is rapid, within 30 minutes or less, and very successful. But rapid treatment is never seen in hospital hyperbaric facilities, unless it is an inside attendant who is diagnosed promptly.

In the afternoon Ed Thalmann spoke about oxygen toxicity during in-water recompression. No figures were offered of the occurrence of oxygen toxicity during such treatment. His paper was, however, a good summary of how the USN recommends treating oxygen convulsions when using closed circuit oxygen rebreathers. His conclusions, printed in bold type, were "in-water recompression should never be attempted except as a treatment of last resort when it is certain that delaying recompression might put the diver in significant danger, and when the procedure can be done safely by trained individuals and will not delay evacuation to a recompression facility". The discussion on this paper made it quite clear that the never in the above statement should have been underlined for even more emphasis!

The next speaker was Richard Pyle who discussed technical diving practices and the need for these divers to be ready to undertake IWR as when problems arise in such a dive DCS is often of rapid onset and progressive. As readers of the Journal will remember Pyle and Youngblood published a large series (500 odd) of both air and oxygen IWR treatments with about 90% completely relieved of their symptoms. The discussion was largely devoted to Richard's and his buddy's experiences with DCS being treated with IWR.

The final speaker was Richard Moon, who was a guest speaker at the 1999 ASM. Richard is an advocate of doctors treating all cases of DCS in hospital chambers and has no place for recompression out of hospital. The final discussion was introduced by Dr Robert Overlock and is a pretty fair summary of the pros and cons. The ten pages of final discussion are well worth the price of this book. I cannot summarise it in a review, so readers will have to buy the book and guess which of the unidentified speakers is Alf Brubakk, the other 1999 guest speaker who is a believer in there being a place for immediate recompression. Not surprisingly, no consensus on the place of IWR was reached.

The only faults that can be found in this book are a few misprints of diving equipment names and the occasional omission of a word or two in a sentence which has the effect of giving the first sentence of a comment a different meaning from that derived from the following sentences.

John Knight

Key Words

Accidents, book review, decompression illness, immersion, meeting, oxygen, safety, thermal problems, treatment.

SPUMS ANNUAL SCIENTIFIC MEETING 1999

DECOMPRESSION ILLNESS IN THE ROYAL AUSTRALIAN NAVY 1961-1999

Robyn Walker

Key Words
Decompression illness, history, treatment, sequelae.

The 1960s

In 1961 the Royal Australian Navy (RAN) determined it had a requirement for an underwater medicine service. The Underwater Medicine Clinic was opened in February 1961 at HMAS RUSHCUTTER, a shore establishment where the divers were based. The RAN coerced an anaesthetist, Rex Gray, to accept a commission in the Navy and appointed him to the job. The requirement for an underwater medicine clinic was confirmed on his first day on the job when a diver conducting free ascent training suffered pulmonary barotrauma and was dead on arrival at the clinic. That was Rex Gray's introduction to underwater medicine.

The RAN further recognised there was a need for an underwater medicine research function, and laboratory facilities and civilian technical staff were provided. The School of Underwater Medicine (SUM) was officially established on 21 January 1963. SUM was relocated to HMAS PENGUIN from RUSHCUTTER in July 1968. Going back through the records, there are abundant press clippings relating the involvement of the RAN in making dramatic dashes across the country to rescue divers in distress. At that time, the RAN provided the major treatment facility for injured divers.

When SUM moved to HMAS PENGUIN, "the offices, library and laboratory were installed in a part of an old, ramshackle wooden building that is 200-300 yards away from the diving working area. The floors were bare, with gaping cracks, the walls and fibro roof were uninsulated and out of alignment." SUM remains in a similar building and no major structural modifications have occurred over the last 30 plus years!

In 1968, the Navy was still averaging one diving death a year. On average, one diver a fortnight was reported as losing consciousness during a dive on a rebreather set. Originally, diver error was considered the most likely cause of these incidents, until Carl Edmonds and his staff studied the equipment. Volunteers, including Carl, exercised to unconsciousness in the water while medical staff hovered above, retrieving the unconscious diver and collecting gas and blood gas samples for analysis. The answer was hypercarbia due to inadequate carbon dioxide absorbent systems. It would be difficult to obtain ethics committee approval for that type of experiment today!

The 1970s

It appears the first widely published work from the unit on decompression sickness was in 1976. Both the Singapore and Australian navies were interested in the types of diving accidents that were occurring. As it still is today, the distinction between Navy and civilian or recreational divers, was very marked. Navy divers had a disproportionate dominance of joint bends compared to recreational divers in whom cardiorespiratory and neurological symptoms were far more frequent. Jimmy How, Dawn West and Carl Edmonds reviewed a clinical series of 115 cases of DCS in civilian divers and, even at that time, commented that the classification between Type 1 and Type 2 decompression sickness (DCS) was not wholly satisfactory.[1] The Singapore Armed Forces recompression chamber treated 40 divers, while SUM treated 75. To be part of the study, the diver had to have had indisputable signs of decompression sickness or development of clinical symptoms during or after ascent, which were relieved or cured by recompression. They did not include cases of pulmonary barotrauma.

Various treatment tables were used. The air tables were still used widely, particularly in Singapore as well as the 18 m oxygen tables. However, Carl Edmonds liked to use what he called his "high oxygen pressure" tables, which was the maximum safe oxygen pressure, administered either in an RCC or underwater. He described this treatment as going to depth of relief on an appropriate PO_2 and staying on oxygen for the ascent. He did not give air breaks and followed up the treatment with 100% oxygen after surfacing. Basically these tables involved recompression to 9 or 12 m on 100% oxygen for periods of 2-3 hours. Carl believed there was a lesser risk of oxygen toxicity compared with the 18 m tables and he used a much slower rate of ascent, 12 minutes per metre. He did however qualify the use of his tables and he is quoted as saying, "You should only use these tables if you are a prudent diving physician who can predict outcome and final prognosis from the dive profile, the time sequence of events, and the clinical presentation. Non-experts should adhere to the strict guidelines detailed in the shallow oxygen tables."

The patients also received adjunctive treatment according to the severity of their presentation. Some received intravenous fluids, catheterisation was performed as necessary and a number of patients received steroids. Anti-epileptics and tranquillisers were administered to

neurological cases and electro-diagnostic and clinical monitoring was used as required.

Although all the divers were civilians, they ranged from recreational divers to abalone divers and locally employed divers, using either scuba or surface supply. The mean age was 32.4 years. The mean depth dived was 30 m and the mean duration of the dives was 120 minutes. The mean time of onset of symptoms was 33.1 minutes from the time of leaving the bottom. Eighty-seven (76%) exceeded any dive tables which were available. For 13 (11%) there was not enough data to say whether they were within the tables. There were only 15 (13%) who had dived in accordance with tables. The mean delay to definitive treatment was 50.9 hours.

Fifty-four divers (47%) presented with what was then known as Type 1 decompression sickness or joint pains. Upper limbs were the most affected, shoulders and elbows, followed by knees and hips. When multiple joints were affected, they tended to occur in neighbouring joints.

Sixty-one divers (53%) presented with Type 2 DCS namely cerebral, spinal, or combined cerebro-spinal, inner ear and cardio-respiratory disease. Spinal lesions tended to predominate in the cases from Singapore, where there were many fishermen divers with long deep dives, whereas in Australia, cerebral and cardio-respiratory symptoms seemed to be more common.

Many of these cases of Type 2 DCS had initially been treated with in-water air recompression. This was often aborted because of the difficulties encountered and the patients often surfaced far worse than at the commencement of the treatment. This procedure also often resulted in an unnecessary delay to definitive treatment. Spinal cord involvement was the most likely cause of persistent disability at that time.

Fifty-seven percent of patients (66) had symptoms within 10 minutes of completing their dive. Twelve divers (10%) developed symptoms during repetitive dives, on ascent or while decompressing. Thirty one (27%) developed symptoms immediately on surfacing and twenty three (20%) within the first 10 minutes. The longest duration between ascent and initial development of symptoms was 19 hours.

How, West and Edmonds used a simple grading system to assess the response to treatment. A complete cure was assessed as a grade 4 response; death was assessed as a grade 0 response, with partial responses in between. Ninety five divers (83%) had a complete or almost complete cure (grades 3 and 4). They did well with whatever treatment table was used. This included a mixture of air tables, shallow oxygen tables, surface oxygen, or Carl's high-pressure oxygen table. Two divers died and 14% (16 divers) showed no improvement.

Reviewing this sort of treatment is difficult because the severity and local conditions decided what treatment table was used. Mild Type 1 decompression sickness occurring in a patient remote from a recompression chamber, e.g. in Vanuatu, was likely to have been treated with 100% oxygen at surface pressure. In Singapore, they tended to use air tables, while in Australia, Carl Edmonds tended to use his own oxygen table. Looking at the graded responses to treatment, Carl's high-pressure oxygen table was assessed as having the best response and surface oxygen achieved better results than the deep air tables.

In the majority of cases, surface oxygen was administered, following completion of the recompression, for an hour on, an hour off, for up to 24 hours. A lot of the divers, even at that time, but particularly for spinal decompression sickness, were given a follow up hyperbaric treatment. The initial results indicated 60% (69 divers) achieved full recovery within 24 hours of their first treatment. A further 17% (20 divers) recovered within one month and 14% (16 divers) within one year. Only 7% (8 divers) were permanently affected and two divers (2%) died.

The authors compared the use of the shallow oxygen tables against the air tables. They thought that, certainly in military diving, the shallow oxygen tables had a dramatic effect and were seen to be the treatment of choice. This was not so for the civilian divers, because they were diving outside the tables, had more significant disease and presented later compared with the military cases. Certainly, the lack of any sort of decompression staging, inadequate equipment and equipment failure at depth contributed to the severity of the disease in the civilian divers and the lack of facilities for decompression played a role in the delays to treatment.

How et al. agreed that the shallow oxygen tables (US Tables 5 and 6) were much more attractive to the operators. Using them meant the staff did not have to spend days at the chamber as they did with the saturation air tables. They also felt that in-water recompression worked in remote areas when performed properly. Carl was certainly one of the main proponents of in-water oxygen therapy. Safe in-water oxygen therapy requires an adequate supply of oxygen, a full-face mask and a platform for the patient. The patient must be kept warm and be under the continuous observation of an underwater tender. Sheltered water is preferable.

The 1980s

The 1980s saw a change of personnel at SUM and can be described as the Des Gorman era. There was beginning to be some doubt as to whether the US Tables 5 and 6 were adequate. Some patients, particularly the paraplegics and the serious neurological presentations, did not seem to respond. The philosophy at that time at SUM

was to not surface the patient until there was full resolution of all symptoms. So they would start off with a Table 6 and, if there was no response, go to 30 m, then to 50 m and then proceed to an air saturation table. There are no published reviews of the treatment at that time, but after about 3-4 years of following this treatment philosophy, the return on that investment of time, staff and improvement in the patient was considered very poor. The desire to do air saturation tables soon passed. Instead, the philosophy changed to include a RN Table 54. This is a long air table, of approximately 39 hours. But so few experienced significant recovery with this table, that SUM changed to a short stay at 50 m, followed a RN Table 63, with a total time of only about five and a half hours.

At this time, SUM started to look at outcomes from treatment and neurological sequelae.[2] This review period went from 1984 to 1986. They studied 88 divers, one of whom had an air embolism and the rest were assessed as having had decompression sickness. All were treated in the RAN multi-place chamber and all were intravenously hydrated. Initially, they were treated at 2.8 bar on 100% oxygen (USN Table 6). If there was no response they were changed to an oxygen-nitrogen mixture and compressed to either 30 m or 50 m. Recurrent or persistent symptoms or signs were treated with daily hyperbaric oxygen (HBO). HBO was given at 9 m, 10m or 14 m for 2 hours, depending on who was running the treatment. They examined the patients clinically for neurological outcome at one week, one month and one year. The patients underwent a formal psychological assessment. The patients had 19 lead EEGs under conditions of rest, with photic stimulation and with hyperventilation, and underwent a CT head scan. When the EEGs and the CT head scans were done either a neurologist or a radiologist who was not aware of the patient's history evaluated the patients.

Before any treatment, a neurological deficit was detected in 68 of the 87 patients (78%) with DCS. At discharge 84 patients (96%) said that they were completely asymptomatic. Clinically no abnormality was detected. Twenty out of 61 (33%) had an abnormal psychological assessment on a battery of profiles, 22 (36%) were assessed as having abnormal EEGs and 8 (13%) had abnormal CT head scans. At follow up the numbers dropped off at a week, a month, and a year. After one week only 46 people (or half) presented for review. At discharge 96% were said to be completely normal, but a week later ten people (22% of those who attended) were detected, clinically, to have a neurological deficit. After one month, the numbers dropped again, but there was some recovery. While 10 were considered abnormal clinically at one week, by one month only 2 of those had persistent clinical signs.

Diver follow up is a problem with all our diving studies. There is a high dropout rate. The important thing was that the clinical morbidity at discharge was considerably lower than when the patients were followed up a week later. We may believe that we have good recovery at discharge, but if we do not follow up our patients we are not sure what is happening to them. After this work, there was certainly belief, in Australia and perhaps worldwide, that USN Tables 5 and 6 were not always effective.

Michael Loxton[3] undertook a study to determine the effectiveness of the Minimal Recompression Oxygen Tables, with the aim of trying to provide an accurate estimate of the failure rate and looking for predictors of poor outcome. There were 319 patients treated between January 1983 and December 1993. Fifty-six patients were excluded: insufficient information was recorded in 12 cases, some were later assessed as not having DCS and other cases had treatment on air or mixed gas tables. 263 patients were finally included in the study.

The mean age of the divers was 29. At that time, it was probably representative of the wider diving population that 83% (218) of the study group were male and 17% (45) were female. Most were treated on a USN Table 6, some with extensions. Others had a Table 5, and some were given a 9 m oxygen soak. A mean of 2.2 additional hyperbaric treatments was administered.

At the end of the first treatment, 153 divers (58%) still had residual symptoms. At the end of all treatments 76 divers (29%) still had residua. There continued to be an improvement, from one week to one month, to six months, to 12 months post treatment. However, even at 12 months, 24 divers (9%) had residual symptoms.

Patients who presented with neurological decompression illness were more likely to fail to respond fully. There was a significant number who had residual symptoms at the completion of all treatments. Thirty four percent of the neurological patients had residual symptoms, compared with 21% of the non-neurological, or the old Type 1, patients. This was a statistically significant change. When subdivided into sports divers, (who were the majority), naval divers, and professional divers the differences in poor outcome were not statistically different between the three groups. The proportion of Naval divers with Type 1 symptoms was higher, but not statistically different.

When looking at outcomes, Michael Loxton looked for associations between 17 variables and the presence of residual symptoms at the completion of all hyperbaric treatments. Seven variables were associated with a statistically significant increased risk of treatment failure. Those were:

the presence of neurological disease;
age over 35;
going to altitude after the dive;
failure to comply with DCIEM tables;
further diving after onset of symptoms;

treatment delay;
lack of first aid oxygen.

However when adjusted for confounders, the only variables that had significance with poor outcome were age over 35, treatment delay and further diving after onset of symptoms.

Seventy-six (29%) of 263 patients were left with residual symptoms. The shallow oxygen tables appeared to be more effective for non-neurological than for neurological DCS and neurological symptoms appeared to be more likely to persist after completion of all treatment. Certainly, he demonstrated a tendency for the residual symptoms to improve spontaneously. One of the conclusions was that the shallow oxygen tables appear adequate for the old Type 1, or non-neurological, symptoms but perhaps we should be seeking different treatments for neurological disease.

The 1990s

I believe that there are different subsets of patients. We see the patients who come in a week after diving with some mild paraesthesia and they just do not feel right. I treat them with Table 6 or an extended Table 6 and it seems to work quite well for them. Another group is patients who come in with serious, progressive, neurological disease and who, despite early surface oxygen, despite early recompression and despite aggressive fluids, do not seem to get better. That produces difficulty for us at the clinical level. The 22 year old who leaves the chamber paraplegic is a treatment failure and a significant burden for the community. My treatment philosophy is that the very acute patients, with progressive disease, who present within 24 hours of their injury and are not resolving at 18 m, are recompressed to 30 m. I use 50:50 heliox at that depth. Why? Why not? We do not know whether it is the right thing to do, but that is what I have elected to do. In the last 5 years, we have had a number of patients who have not done well at 18 m and who have resolved at 30 m. We have also used lignocaine for some of those.

There are divers who dive on open circuit, using trimix (helium, nitrogen and oxygen), to 78 m off Sydney. There was a group of 5 such divers, two of whom have died as a result of this activity. Two of the survivors presented a week apart after one of these deep dives. The first presented with shoulder pain and weakness in his arm. He deteriorated at 18 m, so I took him to 30 m on heliox. He responded very well and was asymptomatic after that treatment and at follow up. His buddy, who presented a week later after diving a very similar profile, had shoulder pain, arm pain, weakness and paraesthesia and was treated by one of our other physicians on an extended USN Table 6. He was very slow to respond and had to have a number of follow up treatments. He was still symptomatic at discharge. This is not statistical evidence, but anecdotally, it influences our decisions.

The Next Millennium

I am not sure what treatments we will be using, however we can only await the outcome of a double-blind randomised multi-centre trial assessing treatment outcome, to help guide our clinical decisions.

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TEN YEARS OF TREATING DIVERS

Chris Acott

Key Words
Accidents, asthma, decompression illness, flying after diving, incidents, sequelae, treatment.

Introduction

I am going to discuss of some of the clinical gems that I have gathered over the past 10 years. I think it was Carl Edmonds who said “How do you know when a diver is lying to you? You just have to watch his lips move.” That

is perfectly true. However, the only advantage of dive computers is that they do not lie.

Rapid ascents

Rapid ascents are good at providing work for diving doctors. They cause bubble disease. They cause morbidity and mortality. From my diving incident monitoring study (DIMS) one of the main causes of rapid ascents is that the diver does not understand his buoyancy jacket. BCs are badly designed ergonomically because the inflate and deflate buttons are next to each other. Often, in an emergency, divers push the wrong button and ascend instead of descend or vice-versa.

We treated one man who loved quick ascents. On most dive profiles he would get up to 8 or 9 m and go straight to the surface. A lot of this was due to the fact that he did not understand his BC. He was a novice diver, who had been sold a buoyancy jacket which floated him face down when he got to the surface. It was a type of BC designed for technical divers, with the buoyancy mainly in the back of the jacket. These jackets, which float the wearer face down, can be described as Darwinian for they occasionally eliminate those who use them. It was a totally inappropriate jacket for him to be using, but he liked novelties, so he bought the most expensive jacket in the shop. It was also unfortunate that he did not understand the inflate and deflate mechanisms. We advised him to retrain before going back to diving.

Denial

Of the divers with decompression illness (DCI) that I have seen, between 10% and 20% had been diving while symptomatic. The result of treatment is not very good when this occurs.

I have often wondered why people do this. I think denial has quite a lot to do with it. Could it be that during diving training they are not taught very much about decompression sickness? I do not think that is a big factor. I think there is ignorance, that they do not realise they have a problem. I have found that it does help to get an accurate history, if one can go through the dive with them step by step and try to work out where "things went wrong". This is particularly useful at follow up, when discussing whether they should give up diving.

Often a diver will come in complaining of minor symptoms, omitting the major symptoms. He will complain of something he associates with decompression sickness and other symptoms are just forgotten or ignored.

Everyone who treats divers talks about denial in divers, and why they do not present promptly for treatment. It is a major cause of delay to treatment. Do divers deny that they have symptoms? Or is it actually part of the disease, organic denial due to cerebral changes?

Recently our neuropsychologist presented me with some articles on organic denial. I am beginning to believe that the denial seen in the majority of divers is actually organic, due to cerebral changes. It is seen in people with right hemisphere strokes.

The reason that I started to think about this was a patient, who came in complaining of elbow pain and dragging his left leg. He actually said "Doc, you have got to fix my left elbow." He kept pointing to his left elbow as he walked. No mention was made of his noticeable limp. I said "Have you got a pain in your left elbow?" and he said "Yes". I said "Well, how long have you been dragging your leg like that?". His reply was "Since I got the pain in my elbow. You have got to fix my elbow". Then I thought that there had to be more to DCI denial than just emotion.

Treatment sequelae

At the medical review after DCI treatment, I always interview the partner. In this way one can find out a lot of things about how the diver has been behaving at home. Has he or she been grumpy? Does he or she go off the deep end a lot quicker than they used to? One can also estimate how the diver is progressing if one sees them at a week, at a month, six months and 12 months.

This story came from seeing a diver whose spouse insisted she be seen as well. She walked in and said "How do you think Joe's going". I said "I think he is doing well clinically. I have examined him. I cannot find anything wrong with him. He says he is fine and he is not having trouble with his memory." or something like that. She said "Well, that is fine Doctor, but can you tell me why he sleeps on the freezer in the garage every night?". One should always interview the spouse!

All our divers are seen by a neuropsychologist, and ours is very good. She understands decompression illness and is very interested in it. But she admits that there is a need for standardisation and validation of these tests. One does not really know how hospitalisation affects performance during these tests nor how any illness will affect the patient's performance. However they can be useful tools.

Returning to diving

After treatments when do we allow diving again? We consider several factors. These include the response to treatment and whether their dive "deserved" the disease they got.

Patients' questions after treatment

How will this affect my future diving? Will I get decompression sickness again? These are common questions. The answers depend on several factors. If we cannot find any problem with their diving we put it down to a chance occurrence and we tell them that they are at no greater risk of getting decompression illness again. But we add the rider, if you do get it, we cannot guarantee that you will get a satisfactory result following treatment.

Often divers ask "Why me?" I explain this by suggesting that he may be the only one admitting to symptoms, particularly if they were diving in a group.

I also explain to divers that DCI is similar to 'flu, it has something to do with the diver's immune system and that the diver was unlucky on that particular day. I use the analogy if one sits in a room with six people and someone is coughing, or has a respiratory tract infection, two or three people in that group will develop symptoms later, and two or three will not. The same applies to DCI, the more one dives, the greater one's chance of being injured. It is just a statistical chance.

Divers always say "I dived within my tables (or computer)". I always think, "Did they?" and look at their profiles. Usually they are denying what they have actually done. I like to look, if I can, at the profile on the dive computer and to compare it with the DCIEM tables.

Then, if all else fails, I tell them how the tables are derived. The majority of diving computers that we use today are derived from Haldane's concepts developed from his goat experiments. Some of the symptoms that Haldane recorded for serious decompression sickness in his goats were, "..the animal was obviously ill and apathetic." and "was not able to move nor be tempted with corn, of which goats are inordinately fond.". It is only a minor exaggeration to claim that if the diving tables or computers were based on the Haldane perfusion model then they are based on the desire of goats for corn.

Asthma and diving

Recently there has been a lot of discussion about asthmatics and diving.

Whether asthmatics are fit to dive keeps on coming up as does their chances of suffering barotrauma.

A factor that is often overlooked, is that the majority of asthmatic divers that I have seen, have had to be rescued on the surface because they became so short of breath that they could not swim back to the boat. Someone else had to jump into the water to rescue them, putting the rescuer at risk.

Are diabetics fit to dive?

I think that if a diabetic wants to dive, then one should dive as the British Sub-Aqua Club do it, with very strict controls. But if a diabetic diver develops decompression illness, he is going to get it from both ends. Hypoglycaemia will affect the outcome. If the diver is hyperglycaemic, to avoid hypoglycaemia during the dive, that too will adversely affect the outcome. We do know that hyperbaric air exposure causes hypoglycaemia in patients who are hyperglycaemic. Dr Orville Cunningham showed this in the 1920s. He used hyperbaric air was to treat diabetics. Hyperbaric oxygen frequently makes diabetics hypoglycaemic.

Flying after diving

When does one allow flying after treatment? This is very important, particularly for tourist divers. We advise 4-6 weeks, but when insurance companies are involved, that is significantly shortened. There are limited data available to us.

At the Diver Emergency Service, we are often rung by divers asking about when can they fly after diving. Frequently the caller dived within the last 24 hours. We reply "We always recommend waiting 24 hours before getting on an aeroplane." The question is repeated, again we reply "We advise you to wait 24 hours." This conversation goes on for about 5 minutes, and is concluded by "You know our number, and where you are going there is a recompression chamber, so you might like to give them a call if you develop symptoms on the flight." I cannot understand why they bother to ring just as they are about to board an aeroplane. Perhaps they expect some magical blessing from the Diver Emergency Service! We actually do get quite a number of calls from Alice Springs because of this! Somebody from the Alice Springs Hospital will ring up and say "I think I have a diver here with symptoms of decompression sickness, who has just flown in from Cairns."

Mask flooding

Mask flooding is a very dangerous event in novices. It is usually associated, particularly in training, with morbidity and maybe mortality. As John Bevan said last year, when diving happiness is a comfortable mask.

Safety stops

It is now built into our diving culture to have a safety stop. I think this is good and it has been shown to decrease bubble count. It is also good for stopping and pausing before you break the surface, because the last 4 m or 5 m of

the dive, where the volume change is largest, is the most dangerous part of the dive.

Unfortunately the safety stop is sometimes used inappropriately. By that I mean that if one has been diving between 10 m and 15 m for 30 to 45 minutes, there is probably no real need to do a safety stop at 5 m, unless to adjust buoyancy, particularly when a diver is low on air. We have treated a few people where this has happened. They were low on air, they signalled and started up. Unfortunately the diving instructor made everyone sit at 5 m for the 5 minute safety stop. The first diver to get low on air, usually a novice, often runs out of air, panics, does a breathhold ascent to the surface and ends up in our chamber with a cerebral arterial gas embolism.

Five minute neurological examinations

Another thing which is creeping into our culture is the on-site neurological examination. This has been published in some books for divers to perform a 5 minute neurological examination on a diver to see whether they have decompression sickness or not. One can probably give one patient with neurological disease to 20 doctors, and they will all find different signs. We have seen divers who had delayed treatment because they went to their dive leader who did a neurological examination and said "I cannot find anything wrong with you", and sent the diver away. I believe that on-site neurological examinations are a hindrance to the diver getting to the appropriate treatment. Some of these 5 minute neurological examinations have been abbreviated even further to what the authors consider to be a standard Romberg test.

Oxygen as first aid treatment

One hundred percent oxygen is the first aid treatment of choice, after securing the airway, breathing and circulation. We teach the Diving Medical Technicians (DMTs) on the courses that we run in Adelaide, DRABC (Diver Rescue, Airway, Breathing and Circulation), give 100% oxygen, and then to think about what is going on. Often when oxygen is used the patient will get better and occasionally they will not want to be evacuated for treatment. The use of surface oxygen has caused delay to evacuation, because a few hours after stopping the oxygen the diver became symptomatic again.

Robyn Walker alluded to someone having 15 hours of continuous oxygen before reaching treatment. That is rare in my experience. Usually I find that divers want to give an air break as soon as the oxygen is started. They will give 20 minutes on oxygen and then a five minute, or longer, air break. We advise giving oxygen for long periods of time, 4 to 5 hours, without air breaks, depending on how long it is going to take to reach the chamber.

Often the data on the use of oxygen does not show the percentage of oxygen given. Neither does it show the interval from the dive to development of symptoms, nor how long it took before the diver was given oxygen, nor how long the oxygen was given for, nor how many air breaks were given, nor the total length of time on oxygen before recompression. This is data that is seldom recorded.

What percentage oxygen to use?

Is it better to give a lower inspired oxygen over a longer period than a higher oxygen for a shorter period? In the 1970s and 1980s, a lot of divers were reported to have received "100% oxygen". But with the equipment they were using, the divers were probably only receiving 50% or 60% oxygen. However, they seemed to lose their symptoms anyway.

Audience participation

Vanessa Haller, Victoria

In the early 80s I used to work in the hyperbaric chamber in Hobart. We mainly dealt with abalone divers. Whether denial is organic or not, we noticed that when we questioned them on the surface after treatment their answers were very different to what to what they said when they were at 18 m (60 ft) on oxygen. For one thing they were a lot more truthful about their dive profiles.

Chris Acott

I agree that one can get two conflicting stories. I have noticed many divers under pressure in the chamber say "I feel like a cloud has been lifted. I can think clearly now". I have often found when trying to talk to divers about when the symptoms developed, that they did nt really know where they had been a few hours before.

Unidentified speaker

If a person develops DCI, is treated and recovers, is he or she likely to be worse affected or more difficult to treat, if DCI recurs ?

Chris Acott

We have only had a couple of patients like that in our chamber. Talking to other diving physicians, they have found in the second case of decompression sickness the outcome has not been good. But there are a lot of compounding factors. Just because a diver develops DCI it does not mean that it will happen again. If the diving profile did not deserve the disease one would have to consider a PFO or some anatomical defect. In that situation, we would advise the diver not to dive again.

Unidentified speaker

But there is no evidence that a second DCI incident would be worse?

Chris Acott

No. We do not really know what is actually happening when we treat patients. What we are arguing about is that perhaps recovery might be neuronal recruitment. If so, divers are knocking off their neuronal reserves with each attack of DCI.

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UNCERTAINTIES IN PREDICTING DECOMPRESSION ILLNESS

David Doolette

Key Words

Bubbles, decompression illness, physiology, research.

Introduction

Decompression tables present a list of ostensibly safe schedules. Divers may expect that dives conducted according to such schedules will be free from decompression illness (DCI) and dives outside the limits will result in DCI. This belief is embodied in Haldane's statement, subsequent to the publication of his decompression tables, "that compressed-air illness has now practically disappeared except in isolated cases where from one cause or another the regulations have not been carried out".[1] The basis for this misconception might be the classification of diving outcome into DCI or no DCI. Using this classification, a particular dive either will or will not result in DCI for an individual. However, the outcome of an identical dive profile may differ for another individual, or the same individual on another occasion. The categorical assertion that decompression schedules distinguish safe (zero risk of DCI) from unsafe dives for the entire population is not only untrue but also impossible. Many commonly used decompression tables have a reasonably low risk of DCI, but any assumption of safety obscures the fact that there will be exceptional incidents of DCI.

Despite his later unequivocal statement, Haldane's original work with goats showed typical biological variability in individual animal susceptibility to DCI.[2] Figure 1 shows some of Haldane's goat data plotted in the form of a dose–response curve. This curve illustrates a low, but finite risk of DCI, following a trivial diving exposure (in this case low exposure pressure) with the risk increasing with the exposure. The exposure where risk rises most rapidly defines the most common limiting exposure for the population. Haldane's (and all subsequent) assertion of safety is based on defining the limiting exposure from a point towards the left of this curve. However, theoretically, there is no point on such dose–response curves where risk is zero.

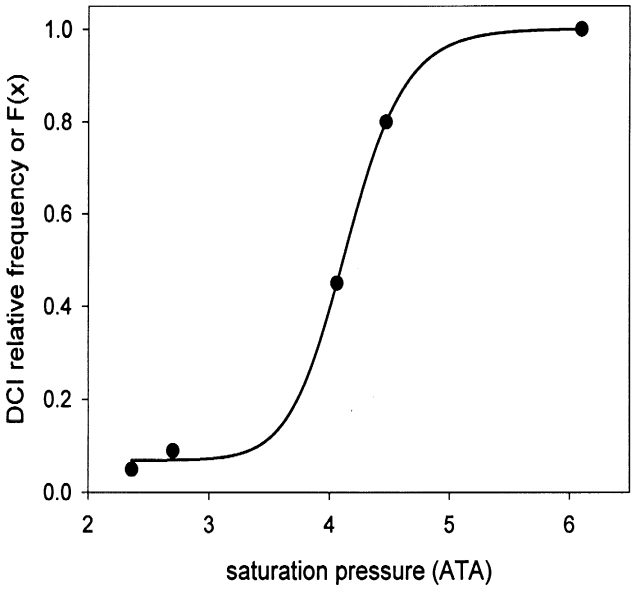

Figure 1. Dose–response curve (cumulative distribution function) for DCI constructed from data tabulated in Haldane's experimental studies with goats.[2] Groups of 4 to 23 goats were exposed to the pressure indicated on the x-axis for 4 hours (3 hours at 4.47 ATA) and decompressed to 1 ATA over 2 to 10 minutes (31 minutes from 6.1 ATA). The circles show the proportion (relative frequency) of goats experiencing any symptoms of DCI. The line is a sigmoid curve, F(x), fitted to the original data.

This paper examines two aspects of uncertainty involved in the prediction of DCI. Firstly, DCI is the result of complex processes that are only superficially evaluated in the decompression theory that underlies decompression tables. Secondly, the main aim is to illustrate that sensitivity to DCI will be normally distributed in a population of divers.

The normal distribution

The sigmoid dose-response curve in figure 1 is derived from an underlying bell-shaped distribution of sensitivity to DCI (see Figure 2). Many biological phenomena conform to a particular bell-shaped distribution called the "normal distribution".

Figure 3 that shows computer simulations of 3 different dice experiments. The distribution of values found

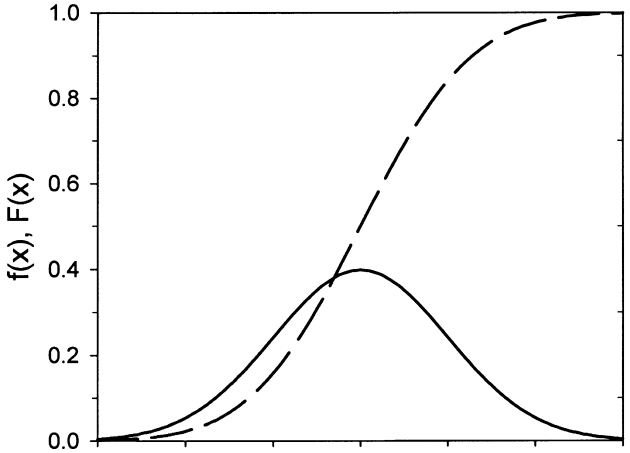

Figure 2. The normal distribution of possible values of a variable X. The x-axis gives the possible values of X. The solid line shows the normal probability density function, f(x) and the dashed line shows the cumulative density function, F(x).

for a sample of 1,000 rolls of one die is shown in figure 3a. If the die is fair, each value from 1 to 6 is equally likely (rectangular or uniform probability distribution function) and this is reflected in the frequency distribution. However the sum of two dice rolled 1,000 times, shown in figure 3b, shows more results in the centre because there is only one way to get a sum of 2 or of 12 but six ways to get a sum of 7. Overlaid on this frequency distribution is the predicted normal probability density function (normal distribution). Figure 3c shows the frequency distribution of the sum of 100 dice rolled simultaneously and repeated 1,000 times. The levels of observed sums are on the x-axis (intervals of 5) and the bars represent the frequencies of those sums (y-axis). Sums near 350 are common and larger or smaller levels are increasingly less common. The frequency distribution is clearly normally distributed, fitting the bell shaped curve of the predicted normal probability density function. This is a (idealised) mathematical model of the frequency distribution. Whereas the probability density is an abstract concept, this continuous function can be seen as describing the *relative* frequency for *every* level on the x-axis (even though only integer levels are possible in the case of dice sums).

The probability (numerically equivalent to the relative frequency) of an individual event in a population can be estimated from the probability density function of a sample. For example, in the dice experiment of figure 3c, only sums between 295 and 417 occurred in this sample of 1,000 rolls, although sums between 100 and 600 are possible. Using the normal probability density function the probability of rolling any number, say 590 (which would be very low), can be estimated. The probability of any range of levels is estimated from the area under the probability density function (integral) over that range. The cumulative distribution function (or probability integral) gives the probability of all levels less than or equal to each value on the x-axis. For a bell-shaped curves the cumulative

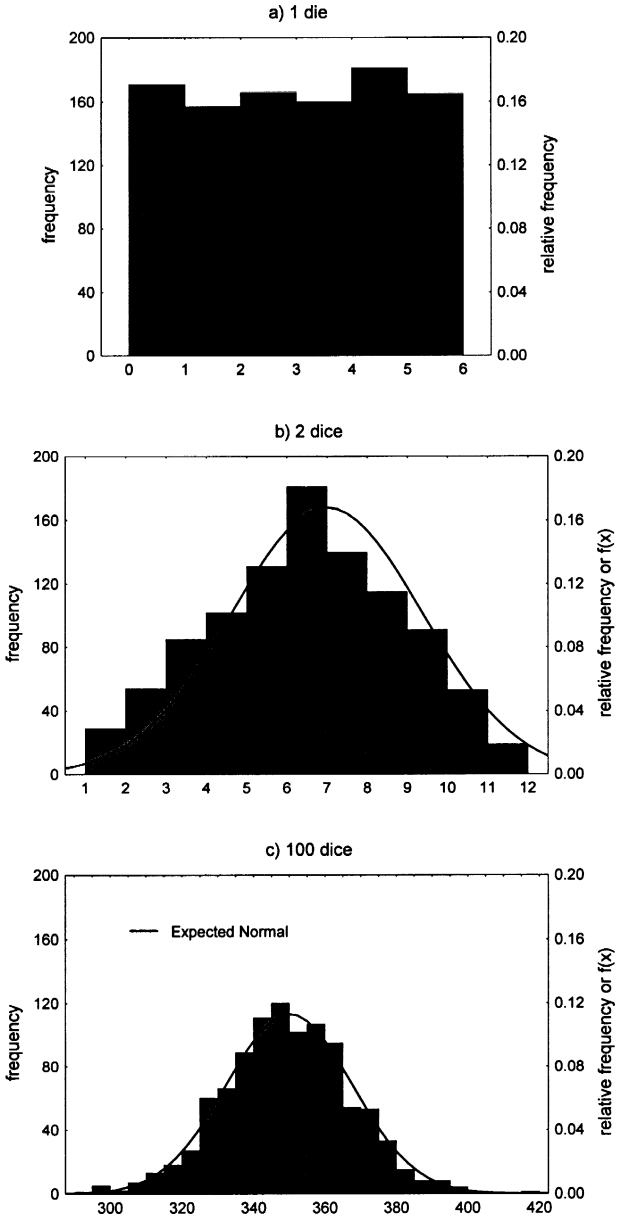

Figure 3. Frequency histograms of a computer simulation of dice rolling experiments illustrating the central limit theorum. The histograms are of the frequency and relative frequency (y-axis) of the sum of the face values (x-axis). The simulations are for 1,000 rolls of 1 (3a), 2 (3b) or 100 (3c) dice. Overlaid on histograms 3b and 3c is the predicted normal probability distribution function (solid line), f(x).

distribution function is sigmoid (see figure 2). For the dice experiment the cumulative distribution function at 590 would estimate the probability of rolling 590 or less (which would be very high).

The normal probability density function describes a family of symmetrical bell-shaped curves that differ only in height and width. The height and width of the curve is determined by a single parameter, the standard deviation.

A quite different experiment could result in a frequency distribution with a similar appearance. The risk of DCI for an upward excursion from saturation is a function of the decompression and the time spent at reduced pressure.

If a group of subjects were decompressed, a frequency histogram of the number of individuals first displaying symptoms (y-axis) during various time intervals (x-axis) might look like the histogram in figure 3c and could be described using a normal probability density function. In other words the sensitivity to DCI in this sample would be normally distributed. If the cumulative number of individuals having displayed symptoms at each time interval was plotted against the same x-axis the result could be described using cumulative distribution function. Such a cumulative distribution function is recognisable as a dose–response curve where the value at any time would estimate the probability of DCI for an individual exposed to that pressure for that duration.

Although the results of many experimental designs naturally present as sigmoid dose–response curves (cumulative distribution function) the underlying bell-shaped probability distribution is not obvious. A common experimental design is to expose groups of subjects to different levels of decompression stress (e.g. exposure pressure, exposure time, extent of decompression) and record the number of individuals from each group displaying symptoms of DCI. A plot of the relative frequency of symptoms in each group (y-axis) against decompression stress (x-axis) would give a sigmoid dose–response curve as is shown in Figure 1 using some of Haldane's original data.

An important aspect of probability density functions is the belief that a relatively few models (of which the normal probability density function is an important one) fit many real world situations, a notion supported by empirical observation. Many biological phenomena appear to be normally distributed which can be in part explained by the central limit theorem.

Central Limit theorem

A simplified explanation of this theorem states that, given certain conditions, if a variable (S_n) is the result of a sum of a large number of other variables ($S_n=X_1+X_2+\ldots+X_n$), a sample of variable S_n will be normally distributed.

This is true regardless of how the underlying variable (X_1, X_2,…X_n) are distributed. This is illustrated in figure 3. Normal distribution is the assumed model for biological phenomena because any measured variable (e.g. sensitivity to DCI) is the result of many underlying genetic and environmental factors.

A biological response as complicated as DCI is the result of many contributing factors, ranging from the well quantified to the unknown. By virtue of the central limit theorem alone, it is reasonable to predict that sensitivity to DCI should be normally distributed in a population and therefore outcome after decompression is uncertain. To illustrate that this is not just a statistical "black-box" approach, we will now examine experimental findings of aspects of the cascade of events that lead to DCI to see if they conform to this prediction of a normal distribution.

Events leading to DCI

For any dive, the sequence of events leading to DCI can be grouped broadly into:

1 uptake and elimination of inert gas;
2 bubble formation upon decompression; and
3 pathophysiological response to bubbles.

Decompression schedules determine the presence or absence of DCI following any defined dive profile (pressure/time/breathing gas history) using mathematical models incorporating some or all of these mechanisms. In typical decompression models, the input (dive profile) and output (DCI) are measured but the intervening mechanisms are modelled using latent variables (unobserved theoretical constructs). In other words, the models are not based on actual measurements of tissue gas uptake or bubble formation. Haldane's schedules, and later derivations, calculate uptake and elimination of inert gas (latent variable) in the context of a threshold for symptoms of DCI, with only the implicit assumption that bubbles do not form within safe schedules.[2] Later models have incorporated bubble formation as a latent variable with varying degrees of sophistication.[3] Most models do not incorporate pathophysiological responses.

Uptake and elimination of inert gas

Mathematical models of inert gas kinetics come in varying degrees of sophistication. Distributed models account for diffusion of gas between capillaries and tissue units of specified geometry. The membrane-limited diffusion compartmental model ignores tissue geometry and confines diffusion to a membrane between well-stirred blood and tissue compartments. The simplest model is the single perfusion limited compartment, which ignores tissue geometry and diffusion, where gas uptake into a well-stirred compartment, consisting of tissue and blood, is limited only by delivery of gas in the arterial blood. Each simplification results in a more easily handled set of equations. The difference between arterial blood and a single perfusion limited compartment gas content declines exponentially with a rate constant, defined by blood flow, compartment volume and solubility of gas in blood and tissue. Many decompression models incorporate several theoretical well-

stirred single perfusion limited compartments that are not identified with any particular body tissue. It is unfortunate that a misleading interchange of the terms compartment and tissue is found in decompression literature. As this is a theoretical treatment, actual inert gas content of any identified critical tissues is never known. In fact, perfusion limited models have been shown to fit poorly to actual gas exchange data.[4]

Nevertheless, even the most sophisticated deterministic model will not account for the distribution of inert gas uptake across individuals. Figure 4 shows the frequency distribution of concentration of inert gas tracer (nitrous oxide) in the brains of 9 sheep after 15 minutes inhalation of that gas under similar conditions. A distribution with a wide dispersion and some central tendency is identifiable even in this small sample. The predicted normal probability density function is overlaid on the histogram. Statistical analysis (Shapiro-Wilks W test) indicates that this sample is normally distributed.

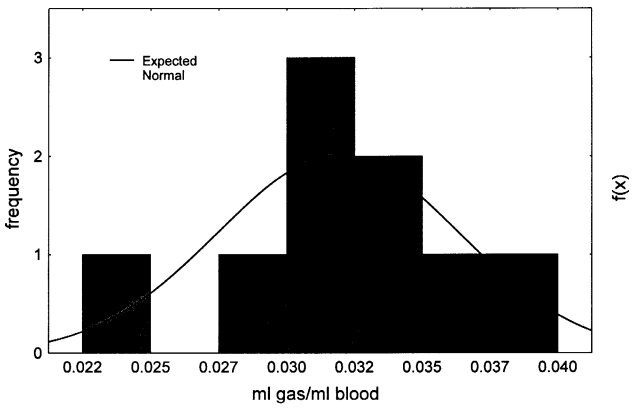

Figure 4. Frequency distribution of inert gas (nitrous oxide) concentration in sheep brain. Nine sheep breathed 10% nitrous oxide for 15 minutes and nitrous oxide content of brain effluent blood was assayed by headspace gas chromatography. Values of brain effluent inert gas concentrations are on the x-axis and the frequency of those values on the y-axis. The solid line is the predicted normal probability distribution function, f(x).

Bubble formation

Bubbles form in tissues if ambient pressure is reduced below total tissue dissolved gas tension (concentration/solubility). Bubble formation is important for two reasons, both dependent on the total amount of gas that separates into bubbles. Firstly, bubbles probably cause DCI. Secondly, elimination of gas from tissue containing free (undissolved) gas (bubbles) will be slower than predicted by many decompression models. The peak volume of free gas that separates from tissue depends on the number of bubbles.[5] The number of bubbles that form for any given decompression is difficult to predict. If bubble inception can result from homogenous nucleation, the rate of bubble number formation will be critically dependent on the surface tension at the site of bubble formation, which is unknown. If, as is widely believed, bubbles nucleation is heterogeneous, the number (and distribution) of these pre-existing nuclei must also be known.

It is difficult to find data on number of nuclei except in the engineering cavitation literature. Figure 5 shows some of the data of Crump of bubble formation from nuclei in a venturi nozzle.[6,7] Replotting the original data as a frequency distribution reveals that, under the controlled experimental conditions, the number of bubbles formed follows a normal distribution (Shapiro-Wilks W test).

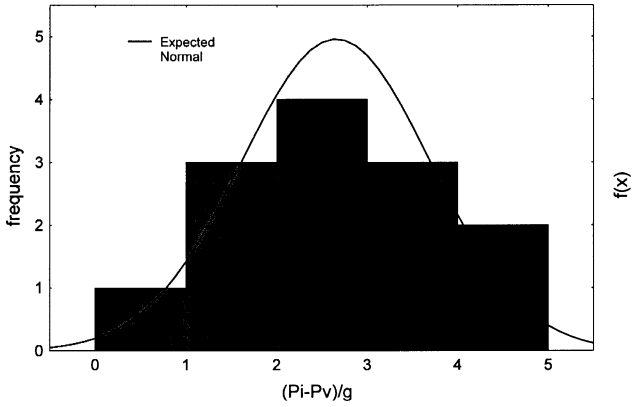

Figure 5. Frequency distribution of bubble nuclei, formed at a venturi nozzzle, in water. The histogram is constructed from data of Crump[7] shown in figure 3.2 of Knapp et al.[6] The solid line is the predicted normal probability distribution function, f(x).

Pathophysiology

DCI is a multi-causal disease and one postulated mechanism is complement activation at the blood-bubble interface. The extent of such activation will depend on the surface area of the gas phase, which will be a function of both total free gas volume and bubble number. Figure 6 shows data on the activation of C5A in blood samples with introduction of a gas stream.[8] The data for 36 such measurements (6 blood samples from each of 6 human subjects) is replotted in the form of a frequency histogram. The data is normally distributed (Shapiro-Wilks W test).

Predicting the distribution of DCI

If the processes that combine to cause DCI are each normally distributed, it is possible to predict the distribution of DCI itself. Although only a portion of all contributing factors, the uptake and elimination of inert gas, bubble formation upon decompression and the pathophysiological response to bubbles serve as examples. For any defined dive profile, the most likely level of response at each of these steps is determined by the input

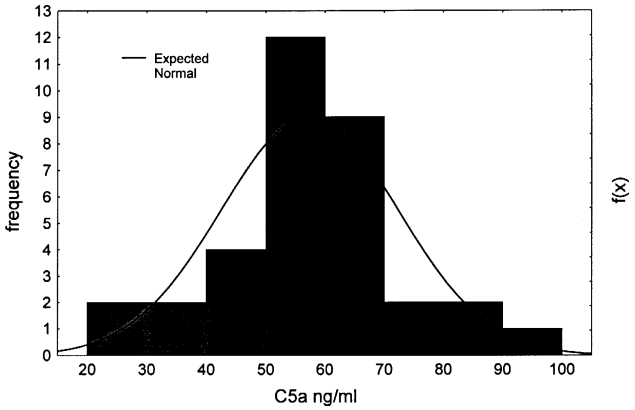

Figure 6. Frequency distribution of bubble-activated C5a in human serum. The histogram is constructed from data shown in figure 4b of Bergh et al. The solid line is the predicted normal probability distribution function, f(x).

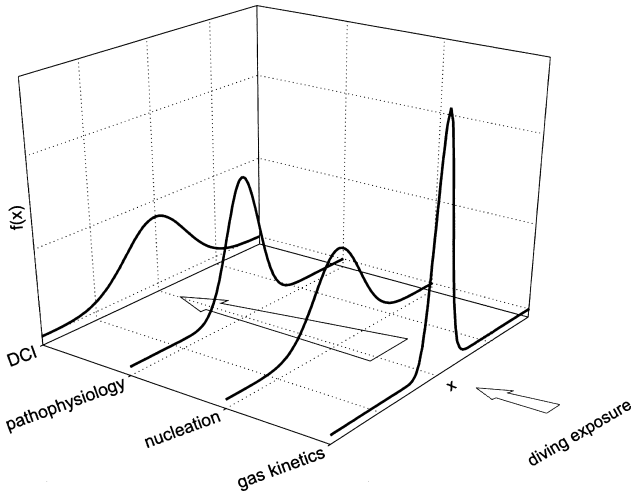

Figure 7. A linear (sequential) combination of independent normal variables has a normal distribution. The steps involved in the production of DCI are normally distributed. A process of multiplication connects the steps. As a result, DCI has a normal distribution that can be predicted from the distributions of the preceding steps.

from the preceding step. However, the input from the preceding step will not influence the shape of the distribution or determine the exact outcome of the subsequent step. Thus DCI (X_{DCI}) is the result of a linear combination of statistically independent variables (X_{GAS}, X_{BUB}, X_{PATH}). Any linear combination of independent normal variables will have a predictable normal distribution.

The standard deviation (which determines the shape) of the resulting normal probability density function can be calculated from the standard deviation of the underlying variables. The formula for this calculation depends on the nature of the linear relationship of the variables. In the current example it is reasonable to assume that a process of multiplication connects each of the events leading to DCI.

$$X_{DCI} = X_{GAS} \times X_{BUB} \times X_{PATH}$$

The coefficient of variation (CV = standard deviation/mean) allows comparison between distributions of variables with different units of measurement. For the relationship above, the coefficient of variation of DCI can be computed from the individual coefficients of variation at each step according to the following formula:[9]

$$(CV_{DCI})^2 \cong (CV_{GAS})^2 + (CV_{BUB})^2 + (CV_{PATH})^2$$

From the distributions shown in figures 4–6, CV_{GAS} = 0.14, CV_{BUB} = 0.39, and CV_{PATH} = 0.26. Using the formula above results in CV_{DCI} = 0.49. The dispersion in each intervening step combines and produces a greater dispersion in the resulting distribution. This relationship is illustrated in figure 7.

Discussion

All mathematical models are a necessary simplification of reality, but such simplification can result in inaccuracy. Decompression models present a theoretical framework for organising decompression experience, not an accurate description of the physiological and pathophysiological pathways to DCI. For instance, Haldane's experimental observations were the "safe" decompression of goats from assumed (but probably incomplete) saturation; all other aspects of his model were purely theoretical.[2] Tabulated decompression schedules may be considerably altered from the underlying model output as a result of field testing, and at the other extreme schedules generated from desktop or diver carried computer are purely model based. In the latter case the accuracy of the model is critical.

Since the sensitivity to DCI is normally distributed in a population of individuals we cannot predict a schedule that separates dives always free of DCI from those always resulting in DCI (square cumulative distribution function). Even the most sophisticated or accurate deterministic model will fail occasionally. Probabilistic decompression models attempt to address this by assigning a probability of DCI to a defined dive profile (based on mechanistic models).[10,11] Setting limits far to the left of the true sigmoid cumulative distribution function separates dives with very low risk of DCI from all others. Such conservative limits result in uselessly short bottom times. Practical schedules are a compromise between useful bottom time and acceptable risk of DCI.

The normal distribution of sensitivity to DCI does not imply that attempts to improve prediction of DCI should be abandoned. If more is learnt about the nature of the intervening processes and incorporated into more accurate models we can make better predictions of the most likely

outcome (without influencing the shape of the distribution of X_{DCI}). Such knowledge may also allow tailoring of schedules to specific populations so that the intervening normal distributions of processes would be more compact as would the final distribution of sensitivity to DCI (for that population).

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THE NATURAL PROGRESSION OF DECOMPRESSION ILLNESS AND DEVELOPMENT OF RECOMPRESSION PROCEDURES

Richard Moon

Key Words

Decompression illness, history, treatment.

Beginnings

Whereas diving with an open bell, and hence exposure to compressed gas, dates back to at least the 16th century, when decompression illness was first observed is not known. However, when the vacuum pump was invented in the 17th century, there was a tremendous fascination with the effects of vacuum on living things. The first example of decompression illness (DCI) and subsequent recompression was described by Robert Boyle in 1670, when he described the effects on animals of decompression in a bell jar. An excerpt from one of his papers is reproduced below:

> "We took a viper and including her in the greatest sort of small receivers, we emptied the glass very carefully, and the viper moved up and down, as if it were to seek for air, and after a while, foamed a little at the mouth, and left off that foam sticking to the inside of the glass: her body swelled not considerably, and her neck less, till a pretty while after we had left pumping; but afterwards the body and neck grew prodigiously tumid and a blister appeared on the back…The jaws remained mightily opened, and somewhat distorted…the air being readmitted after 23 hours in all, the viper's mouth was presently closed, though soon after it was opened again, and continued long so; and scorching or pinching the tail made a motion in the whole body, that argued some life".

This is probably the first written account of decompression sickness (DCS) in animals (and the partial effectiveness of recompression).[1]

Compressed Air Work and Diving

The observations of Robert Boyle remained a laboratory curiosity, with little practical relevance until the development of diving and compressed air work in the 19th century. In 1854 Pol and Watelle first described DCI in miners working in compressed air at Avaleresse-la-Naville in France.[2] A compressed air environment (caisson) in which men performed the excavation, was utilised to keep water and mud out of the working environment. At that site there were 64 men employed, of whom 16 are known to have suffered accidents, and there were two deaths.

After observing bends in compressed air workers during the construction of the bridge over the Seine at Argenteuil in 1861, Foley had recommended construction of a portable recompression chamber capable of sustaining a pressure of 2.5 atmospheres (described by Bert).[3] Presaging an era of questionable clinical use of hyperbaric air therapy, he suggested that this device could be used for treatment of conditions other than bends, such as croup, asthma, snakebite, smallpox and rabies.

The first American major construction job in which compressed air was used was during construction of the bridge (now named the Eads Bridge) over the Mississippi River at St. Louis. Both bridge piers and one of the abutments required compressed air work. There were around 600 men employed in compressed air work in this project. During the East and West Piers there were 91 reported cases of bends, of which 30 were classified as serious; two were crippled for life, and there were 13 deaths. Two thirds of those at the East Pier were attacked immediately on coming out, either on the stairs or as soon as the man had climbed to the top. Some of the severe cases were observed to improve spontaneously, as described in a volume published in 1881 by CM Woodward, Professor of Mathematics and Applied Mechanics at Washington University:[4]

> "Pressure 48 lb. GL (age 30) had worked 5 months; was in the compressed air for two hours. Twenty five minutes after coming out he had epigastric pain, paresis of both legs, and retention of urine; also shooting pains in the legs and back. In three and a half months he could walk by the aid of sticks.
>
> "Pressure 50 lb. LB (age 24) had not worked in the compressed air before. Half an hour after his first shift of two hours, he was attacked with paresis of legs and epigastric pain. He was better in a few days, and was warned by Dr Jaminet not to work in compressed air again. In spite of his advice, he worked one more shift of two hours. He afterwards suffered from paraplegia, and paralysis of the bladder and rectum. Cystitis afterwards developed, from which he recovered in two and a half months.".

Climbing the progressively longer staircase from the level of the work site to the surface immediately after decompression appeared to be detrimental, and at the East Abutment an elevator was installed. At that site there were 28 cases; 27 completely recovered and there was one death, the particulars of whom were described by Woodward:

> "The comparative immunity enjoyed thus far at the abutment seems to have made the men reckless, and the doctor complains that they would not obey orders as to lying down after coming up from the air-chamber, and as to not drinking water for thirty minutes after coming up. (No reason is given for this last rule)
>
> "Some half-dozen light cases, easily disposed of, occurred previously to April 14 [1871], when the pressure was 49 pounds. On that day a man was taken, who died two weeks later. This was the only death by compressed air at the East Abutment; and it would appear from the report of the doctor and of Superintendent McComas that the man brought his fate upon himself. He had failed to bring his dinner, so went home to eat it, contrary to orders. Then, on the way back, he 'filled himself' with beer. Moreover, on coming up from his second watch, he left the works before his hour of rest was up……On reaching home in the afternoon, the man was taken sick with vomiting. His dinner had evidently been eaten with great haste, and was still undigested. In a few minutes general paralysis supervened. The history of his case up to his death shows that the man's blood was in a bad state. He had worked in the air-chamber over three months."

Medical support for the St. Louis Bridge construction was provided by Eads' personal physician, Dr Alphonse Jaminet, who had a personal experience with bends after spending 2 hours at a pressure of 45 psig (approximately 31 msw). Decompression time was 3 minutes , after which he complained of epigastric pain. Since the lock was at the level of the river bed, he then had to ascend a staircase nearly 100 feet high, which he accomplished with difficulty. Despite leg weakness he was able to climb into his buggy, drive home and stagger into his office, where he became paralysed. It was reported that he was unable to speak for a time. Twelve hours later he began moving his legs. Notwithstanding the woefully inadequate decompression schedule (by today's standards) and the severity of his manifestations, he recovered completely.

Snell, describing the experience in digging the Blackwall Tunnel under the Thames River in 1896, noted that pain resolved after days to weeks; none longer than 5-6 weeks.[5]

> "Paralysis usually passes off in from one to a few weeks, unless accompanied by bladder troubles. The fatal cases have usually died from cystitis and bed-sores…..When the bladder is involved some chronic trouble with micturition may remain; and impotence of a more or less lasting character may result from such an illness".

In the largest series of decompression illness in the medical literature, Dr. Frederick Keays, Medical Director of the Pennsylvania East River Tunnels, reported over 3,000 cases of DCS and 20 deaths in tunnelling work in New York. Manifestations are listed in Table 1.[6]

Decompression sickness in divers was first described in the medical literature by a French Navy physician, Leroy

TABLE 1

DECOMPRESSION SICKNESS IN NEW YORK CITY TUNNEL WORKERS

Manifestation		Number	%
Pain		3,278	88.78%
Pain with local manifestations		9	0.26%
Pain and prostration		47	1.26%
Brain symptoms (hemiplegia)		4	0.11%
Spinal cord		80	2.16%
Sensory	36		
Motor	34		
Sensory and motor	10		
Vertigo, "staggers"		197	5.33%
Dyspnea, sense of constriction of chest ("chokes")		60	1.62%
Partial or complete unconsciousness with collapse		17	0.46%
TOTAL		**3,692**	**99.98%**

Compiled from Keays [6]

de Méricourt in 1868, based upon observations made in 1867 on Mediterranean sponge divers, who shortly before had changed from breath hold diving to using the Denayrouze diving apparatus.[7,8] Additional details of bends in this population were reported by Dr. Alphonse Gal, and reprinted by Bert.[3] Forty years later, the disease was described in the English medical literature by Blick, who observed the disease in the pearl divers of Broome and reported 200 cases, of whom 140 lived.[9] Eleven of these cases died, 8 from septicemia due to cystitis and decubitus ulcers and 3 from meningitis. The prognosis of the remainder was surprisingly benign:

> "The rest, after a longer or shorter time, recovered, most of them completely, about 10% being permanently affected with slight paresis, generally of the anterior muscles of the legs.
>
> "I have had patients who have been twice, thrice, or even oftener paralysed, and who have more or less completely recovered.
>
> "The treatment after the establishment of paralysis is that of all organic nervous disease-one can only wait on Nature's efforts, though in this disease Nature is kinder than usual….I have been often astonished at the way apparently hopeless paraplegics have recovered in the course of many months.".

Zografidi published further observations detailing the clinical course and autopsy findings of sponge divers.[10] Fig. 1 depicts a 23 year old sponge diver who dived to 70 meters for 25 minutes and made a rapid ascent. He quickly developed pain, followed by paralysis, and was treated with purging, intestinal antiseptic, application of ice to the head and spine, ergotamine, quinine, quinquina, kola and oxygen. He continued to deteriorate, and on the 8th day of the illness he developed decubitus ulcers in the regions of the sacrum, ribs, scapulae and elbows, which was followed by sepsis, complicated by encephalopathy. He died on day 36. The photograph in Fig. 1 was taken two days prior to death .

Green and Leitch reported spontaneous recovery in 8 of 187 cases of serious DCS in a series collected by the Royal Navy between 1965 and 1984 .[11]

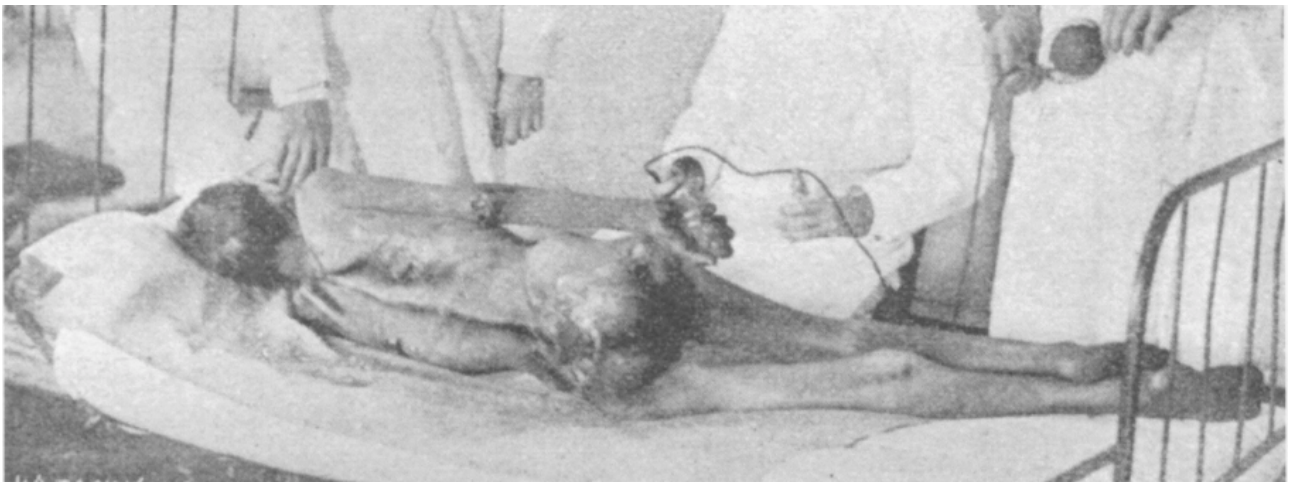

Figure 1. A 23 year old sponge diver who dived to 70 m for 25 minutes and made a rapid ascent. The photograph, showing decubitus ulcers on his buttocks, elbows and back, was taken on day 34 of his illness, two days before his death [9].

Development of Recompression Tables for Compressed Air Work and Diving

In 1872 Dr. Andrew Smith, Surgeon to the New York Bridge Company and the man responsible for the welfare of the caisson workers at the Brooklyn Bridge, observed 110 cases severe enough to require treatment, which often consisted of ergot, whiskey or ginger.[12] Atropine was also used, most likely ineffectually, for spasms. Nausea and vomiting were treated with calomel (mercury chloride). The more mildly affected were reported to have prescribed their own treatment at the local saloon. The impression of the workers and engineers at the time was that most of the afflicted men recovered, irrespective of Smith's treatments. In fact, Smith was in favour of recompression treatment, and conceived a design for a medical lock specifically for this purpose. Unfortunately for the bridge engineer, it was never built. After John Roebling, the bridge designer, died of tetanus following a crush injury to his toes, his son, Washington, took over as Chief Engineer. After exiting the caisson on two occasions he suffered lower limb paresis due to DCS. Unfortunately he was not as lucky as Jaminet, and after the second attack, though eventually able to walk, remained impaired for the rest of his life.[12]

Recompression treatment, although noted anecdotally as early as the 1840s to be effective, was not implemented for several years. Pol and Wattelle had suggested recompression as a treatment,[2] although the idea was not well accepted, perhaps because for an injured man it was counter-intuitive to re-enter the environment that was responsible for his illness. Recompression treatment for bends was not, in fact, systematically utilised until the end of the 19th century. Ernest Moir, an engineer who in 1889 assumed responsibility for construction of the first tunnel under the Hudson River between Manhattan and New Jersey, should be credited with the installation of the first recompression chamber.[13] Injured men were recompressed using air to two thirds of the pressure at which they had been working, then slowly decompressed to atmospheric pressure at 1 psi per minute or slower, requiring 25-30 minutes. Moir recognised that recompression was less successful after a delay, a fact well known to 20th century diving doctors. Before instituting this treatment, Moir reported that approximately 25% of the workforce per year had been dying from DCS. Afterward, he reported that of 120 men at work, only two died during a 15 month period. This positive experience was confirmed by Snell in England in 1896.[5]

Keays used a recompression schedule that was slightly different from the one used by Moir. Keays used a recompression pressure equal to the one in which the man had been working.[6] In his algorithm, as soon as pressure equalled tunnel pressure, decompression was initiated, with the decompression time in minutes at least as great as twice the number of pounds of pressure. In the event of recurrences, the procedure was repeated up to two or three times. For joint pain, Faradism (use of electrical stimulation to cause muscle contraction), hot compresses and hot baths were sometimes used to supplement recompression . Keays' results for joint pain in one knee are shown in Fig. 2. Additional details of the history of hyperbaric medicine of this era can be found eloquently detailed in volumes by McCullough[12] and Phillips.[14]

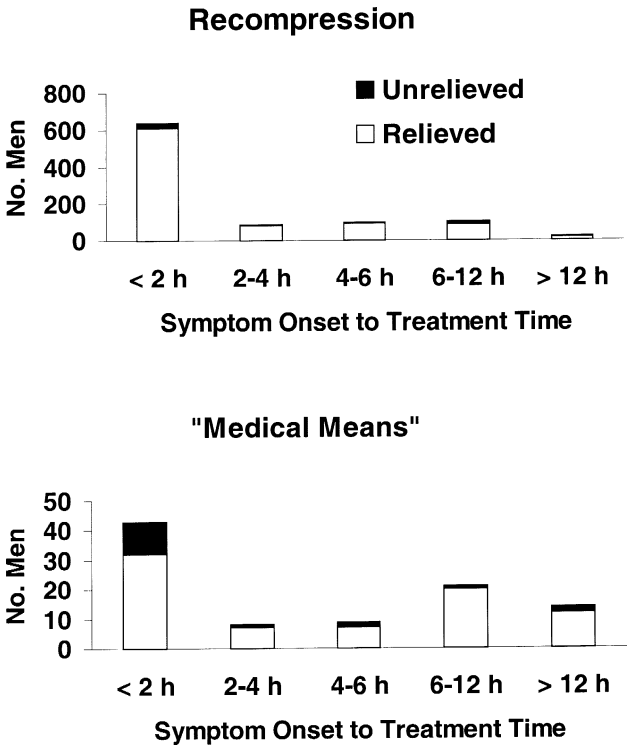

Figure 2. Keays' data comparing recompression with non-recompression therapy ("medical means") for 1,050 compressed air workers with joint pain in one knee.

Recompression therapy for divers took longer to catch on. In the Royal Navy, even as recently as 1907, although oxygen administration at the surface was recommended for the treatment of bends, and in-water recompression practiced, recompression chambers were not routinely available for diving work.[15] The US Navy (USN) Diving Handbook in 1905, containing a meagre 44 pages, did not even mention DCI or its treatment.[16] It was not until the 1924 edition of the USN Diving Manual that a standard therapy was proposed (Table 2).

The development of tables since the beginning of the 20th century has been concisely and accurately detailed in a recent *SPUMS Journal* article by Dr Chris Acott.[17] There are few reports in the medical literature pertaining to recompression treatment of DCS until the 1930s. Even at that time, treatment was largely empirical. Reporting on the experience during construction of the New York-Queens Midtown Tunnel in 1938 wrote that the recommended treatment pressure was equal to that to which the worker was originally exposed, although sometimes, in

TABLE 2

USN DIVING MANUAL (1924 EDITION) INSTRUCTIONS FOR TREATING DCS

Compress to 45 psi (101.5 fsw).
If substantial relief not achieved, compress to 60 psi (135.3 fsw).
Stay at depth of relief until symptoms relieved.

Decompress at the following rates:

60-45 psi	1 psi/min
45-30 psi	1 psi/3 min
30-15 psi	1 psi/5 min
15-0 psi	1 psi/10 min

order to achieve relief of symptoms, it was necessary to treat at 5, 10 or 15 psi higher.[18] After recompressing an injured man, stated Thorne, "The consensus favors waiting twenty to thirty minutes before starting the decompression process". One can infer that there was not unanimity of opinion on this issue. He went on to report that, despite the waiting period, symptoms were apt to recur, requiring one or more additional recompressions.

Oxygen administration, in part because it was not readily available in sufficient quantities until the mid 20th century, was not routinely used until much later. In the 1870s, Paul Bert, working in France, first noted that when 100% oxygen was administered to animals with DCS, some of the signs would resolve.[3] He suggested that oxygen administration caused resolution of gas within the veins and the right heart, but that recompression (with air) was necessary to resolve bubbles that had migrated into the central nervous system. Bert did not put the two modalities, oxygen and pressure, together, and it was Nathan Zuntz, a Professor of physiology in Berlin, who in 1897 suggested simultaneously using both to treat DCS, although he did not have facilities to try it.[19] The initial results of what would today be considered abbreviated oxygen therapy, were somewhat disappointing. Keays reported that oxygen had been given to several severe cases during decompression, but afforded no appreciable benefit.[6]

In 1939 Yarbrough and Behnke reported methods by which injured divers could be treated using oxygen under pressure, but these were not initially adopted.[20] Although the authors were USN investigators, 30 years would elapse before the USN introduced oxygen recompression as a routine. Instead, in the 1943 USN Diving Manual a new table was introduced, in which the diver was compressed using air to the depth of relief (up to 300 fsw), remained there for 30 minutes, then decompressed according to a schedule requiring up to 387 minutes of decompression time. In 1944 four tables were issued (long and short air tables, long and short O_2 tables).[17] The short tables had a maximum recompression depth of 100 fsw, while the long

TABLE 3

EXPERIENCE WITH LONG AND SHORT AIR AND O_2 TABLES IN THE USN IN 1944

Table	Cases treated	Successful treatments	Recurrences
Short O_2 Table	6	6	0
Short Air Table	9	7	2
Long O_2 Table	10	5	5
Long Air Table	5	3	2

(Compiled from Van Der Aue[21])

tables had a maximum depth of 165 fsw. In the O_2 tables 100% O_2 was administered at depths of 60 fsw and shallower, but only for a total of 95 minutes. Only 30 minutes were spent breathing O_2 at 60 fsw (compared with the current standard of 60 minutes or more using USN Treatment Table 6), and a total of 95 minutes of O_2 in all. In 1947 Van Der Aue and colleagues reported the 1944 experience with these four tables, shown in Table 3.[21]

USN Tables 1 (Short O_2 Table), 1A (modified Short Air Table), 2 (modified Long O_2 Table), 2A (modified Long Air Table), 3 (modified Table 2A; not tested) and 4 (for difficult cases) were then promulgated, and used exclusively between 1945 and 1965. Several bends occurred in tenders during use of these therapeutic tables, and the failure rate for the injured divers was nearly 30%.[22]

USN Tables 5 and 6, in which divers were administered 100% O_2 at 18 msw and 9 msw continuously, except for short air breaks to reduce O_2 toxicity, were then developed, tested and adopted for field use. Workman's 1968 analysis revealed that these oxygen tables had a high degree of success.[23] Experience since that time has confirmed the initial observations (see Table 4), and these treatment tables remain in use today.[24]

It was believed that tables different from the ones used for DCS would be required for the treatment of arterial gas embolism (AGE). Studies to determine the appropriate treatment pressure for AGE were performed by investigators in the US Navy in the 1960s. Intracarotid air injection was performed in anaesthetised dogs. Using a skull window technique, Waite observed that all visible bubbles disappeared at a compression depth of 100 fsw, suggesting that this should be the appropriate compression depth for AGE in divers.[25] It was believed, however, that fleet diving officers would not accept a table with only 100 fsw as the maximum depth.

Thus USN Table 6A, specifically designed for treatment of arterial gas embolism, incorporated a 30 minute period of air breathing at 165 fsw, followed by O_2 administration according to USN Table 6.

TABLE 4

SINGLE RECOMPRESSION SUCCESS RATE OF USN OXYGEN TREATMENT TABLES

Source	Number of cases	Complete relief %	Substantial relief %	Comments
Workman [23]	150	85	95.3%	after 2nd treatment
Erde and Edmonds [47]	106	81		
Davis [48]	145	98		Altitude DCS
Bayne [49]	50	98		
Pearson and Leitch [50]	28	67	83	
Kizer [51]	157	58	83	Long delays
Yap [52]	58	50	84	Mean delay 48 hours
Gray [53]	812	81	94	
Green [54]	208	96		All pain only, USN Table 5
Ball [55]	14	93 (mild cases)		
	11	36 (moderate cases)		Many cases with long delays
	24	8 (severe cases)		
TOTAL	**1763**	**81%**		

(Compiled from Thalmann[24])

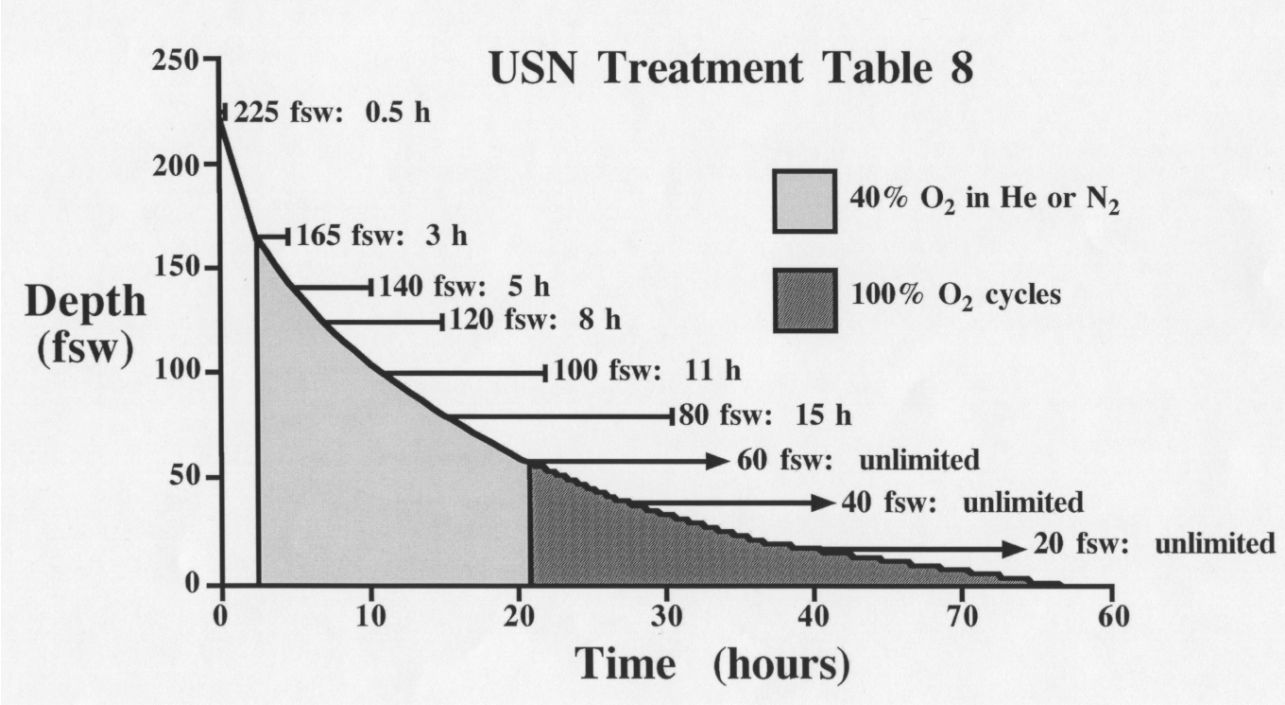

Figure 3. USN Treatment Table 8. Designed for treatment of deep "blowups", in which there has been more than 60 minutes of missed decompression stop time. It can be used in other situations, for example to compress to a higher pressure than 6 ATA (50 msw,165 fsw), or to stop decompression between 18 m (60 ft) and 50 m (165 ft). Maximum times at each depth are shown; times at 18 m, 12 m and 6 m (60, 40 and 20 ft) are unlimited. Decompression occurs in increments of 0.6 m (2 ft). When deeper than 50 m (165 ft, 6 ATA), to reduce narcosis, a 16-21% O_2 in helium can be administered. Four treatment cycles, each consisting of 25 minutes of "treatment gas" followed by 5 minutes of chamber air can be administered deeper than 18 m (60 ft). Treatment gas used deeper than 18 m (60 ft) is 40% O_2 in either He or N_2; at 18 m (60 ft) or shallower treatment gas is 100% O_2. USN Treatment Table 7 guidelines are used for O_2 administration at 18 m (60 ft) or shallower. Further details can be found in the US Navy Diving Manual [27]. Figure reproduced from Moon et al [29], with permission.

Developments since the 1960s have included "specialty" tables designed to treat bends under exceptional circumstances, such as saturation treatment (e.g. Miller saturation table,[26] USN Treatment Table 7[27]) after deep dives or after rapid ascent to the surface with omitted decompression ("blowups"). USN Treatment Table 8 is a relatively new table designed for the latter purpose (see Fig 3).[27] Other developments have included modification of standard USN treatment tables to allow more intensive treatment. For example, the Catalina modification of USN Table 6 allows up to eight 20 minute periods of 100% oxygen breathing at 18 msw.[28,29] Others have modified USN Table 6A to allow longer than the usual 30 minutes at 50 msw, and breathing enriched oxygen mixtures.[30] Shorter tables have been designed to permit treatment of DCI while continuously breathing 100% oxygen in a monoplace chamber without an excessive risk of oxygen toxicity.[31-34]

Altitude Decompression Illness

In France in 1875 three men, Crocé-Spinelli, Sivel and Tissandier, attempted to reach an altitude record in the hydrogen filled balloon *Zenith*. The balloon ascended to an altitude of about 8,000 m and then began descending. Desiring to go higher, the balloonists dumped all of the available ballast, and the balloon resumed its ascent. After landing, Crocé-Spinelli and Sivel were dead. In Tissandier's written account (which is reproduced by Bert)[3] related that at one point in the flight he was unable to move his arms. One might speculate that this effect might have been due to neurological DCS rather than the hypoxia to which it has traditionally been ascribed.

The possibility of altitude DCS was first considered by Henderson in 1917.[35] He speculated that the disease would not occur until an altitude of 20,000 feet had been reached, an impossible feat for aircraft of that era. Juxta-articular pain, which was probably due to bends, was described in a paper by Barcroft and colleagues in 1931.[36] In a study at 30,000 feet in an altitude chamber, one of the authors (Margaria) developed knee and muscle pain.[36] Paralysis was first described, at an altitude of 35,000 feet, in 1938.[37] Altitude bends were experienced during operational flights in World War II, since few aircraft were pressurised, and limited or no oxygen pre-breathing was performed. Of 215 B-17 bomber aircrew during World War II who answered a questionnaire, 39 reported 90 instances of bends[38]. When pressurised high altitude reconnaissance aircraft were first introduced, two of three Spitfire pilots who engaged and shot them down at 42,000 ft suffered severe DCS.[39]

Treatment Tables for Altitude Decompression Illness

Bends were systematically studied in flight using a B-24 bomber, using pressure bags in which air crew with bends were placed. Symptoms were treated by pressurising the bags to 3.5 psig (see Fig 4).[40] The resulting "descent" from 35,000 feet to around 21,000 feet was successful at resolving even severe symptoms such as "chokes". Despite oxygen pre-breathing altitude DCS still exists today. In an anonymous survey, three quarters of 416 active duty and retired U-2 pilots admitted to having experienced in-flight bends at some time during their career.[41] Manifestations of altitude DCI are somewhat different from bends associated with diving.[42] Of 447 cases observed during altitude exposure in a hypobaric chamber, 83.2% had musculoskeletal involvement (70% knee pain, which is less common in diving than arm pain), 2.7% had chokes, 2.2% skin manifestations, 10.8% paraesthesias, and 0.5% more severe neurological features.[43] The low prevalence of neurological features may be due to the use of oxygen pre-breathing prior to ascent and early recompression in this setting. It is also possible that individuals in the training and operational environments are more likely to report frank neurological involvement than other forms of DCS.

Minimal recompression was observed in the World War II era to be effective in most cases of altitude bends, with only a minority of victims reporting symptoms after

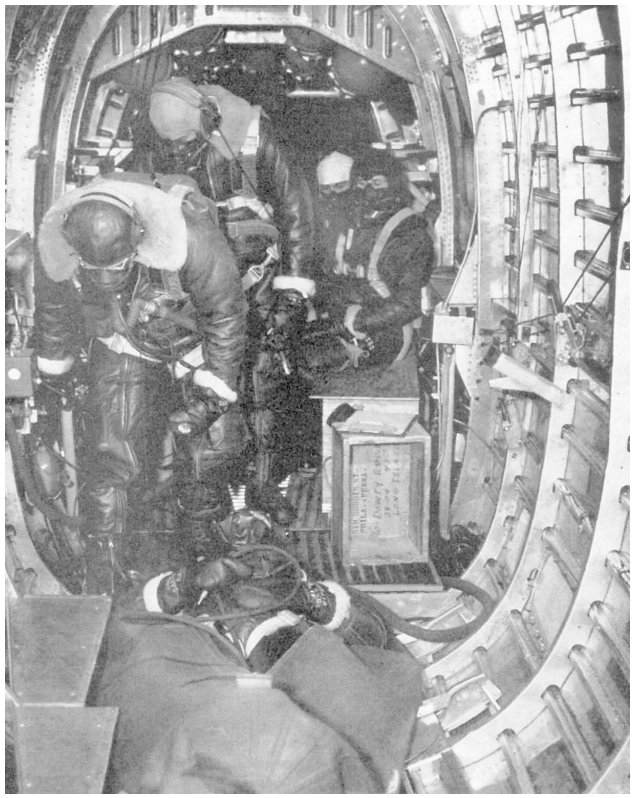

Figure 4: Recompression bag developed in World War II to treat altitude DCS in air crew. Inflatable to a pressure of 3.5 psi, it was a forerunner of the bags currently used to treat altitude sickness (photograph from Lawrence and colleagues [40]).

return to ground level.[39] However, in some cases the symptoms and signs persisted, as exemplified by the following case, initially reported in 1943, and published by Ferris:[44]

> "This man developed vasodepressor syncope during descent, recovered consciousness briefly on reaching ground level, and then lost consciousness again. When examined in the hospital, he was unconscious and the arterial pressure was 108/76 mmHg and the pulse rate was 80 per minute. The patient remained stuporous for two days, during which time he developed several convulsive seizures beginning in the right arm and becoming generalised, and examination revealed a right hemiparesis. As consciousness returned, the patient complained of impaired vision, which then cleared in the next few days. Lumbar puncture on the third day revealed a cerebrospinal fluid pressure of 190 mm H_2O".

The modern, more meticulous approach to treatment of altitude bends includes a period of 100% oxygen breathing at ground level, followed by recompression with oxygen if symptoms persist.[45,46]

As the Space Shuttle cabin pressure is 1 ATA and space suit pressure is only one third ATA, DCS could also occur in space during extravehicular activity. Although it has been rarely reported during space operations, ground based chamber simulations of the decompression profiles in use by Space Shuttle astronauts has produced both pain only and neurological bends. There is also the possibility of catastrophic decompression in the event of suit rupture. A recompression chamber was initially eliminated from the Space Station currently under construction because of space and weight considerations. If recompression is required for an injured astronaut it will be by breathing 100% oxygen in a pressurised space suit, although installation of a pressure chamber has received reconsideration.

Summary

The initial observations in the 19th century of DCS in compressed air workers and divers revealed a disease that could cause severe neurological injury and death, but would often surprisingly resolve spontaneously. The standard of care for this illness today includes recompression with oxygen. For diving injuries occurring in scuba divers or air crew there are presently no convincing data indicating the superiority of any table compared with USN Table 6 or its equivalent. Recompression is a highly effective treatment in most cases if applied shortly after the onset of symptoms.

Therefore there is (fortunately) little opportunity to monitor the natural course of this illness. However, in keeping with the 19th century experience, it is frequently observed that, when severe instances of DCI do not respond to recompression therapy, the prognosis several weeks or months later is often good. Unlike traumatic spinal cord injury, in which improvements are usually small, the cord is often tolerant to injury of similar severity due to gas bubbles.

The obvious combinations of treatment pressure, time and breathing gas composition have been examined, at least anecdotally. Further advances in the treatment of decompression illness are unlikely to occur through new table development, but rather mechanisms by which more prompt recompression can be administered and new adjunctive resuscitative measures.

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TESTING JS HALDANE'S DECOMPRESSION MODEL

Chris Acott

Key Words
Diving theory, history, physiology, research, tables.

Last year I presented a paper on J S Haldane summarising his work.[1] As the majority of dive computers are based on the decompression algorithm that J S Haldane designed, I present here the testing procedures, for goats and humans, used to support his conclusions. Quotations from Haldane's report[2] are printed in *italics* in this paper.

Haldane's assumptions

1 That for bubble formation, the tissue pressure must be greater than the environmental pressure.

2 That tissues can hold gas in supersaturation, and only if the decompression rate is correct this supersaturation can be tolerated without risk.

3 That there were no symptoms of decompression sickness (DCI) without bubbles. We now know that is not correct.

4 From Sir Leonard Hill's work Haldane assumed that carbon dioxide had no influence on decompression risk. We now know this is probably incorrect.

5 That tissue perfusion is the limiting factor in gas uptake. Haldane adopted a mathematical simplification, in the form of an exponential, to describe uptake of inert gas. Hill considered that the limiting factor was gas diffusion.

6 That gas elimination is the mirror image of gas uptake. We now know that is incorrect.

Half times

Using these assumptions and the published work of other people Haldane divided the body into arbitrary half time tissues. A half time is half the time it takes to saturate a tissue with a specified gas. A short half time denotes a tissue with a good blood supply and rapid saturation. A long, or slow, half time is a tissue which takes up the gas slower because of a lesser blood supply. Haldane calculated these half lives to represent what happened in the body. Moir's data gave him his 20 minute half time. Haldane used Hill and Greenwood's[3] data of nitrogen excretion to provide his 5 minute half time. That is based on the nitrogen content of urine. Hill and Greenwood considered that as the kidneys are very well perfused in life they represented what we now call a fast tissue. They experimented on themselves in a chamber under pressure where they passed urine every 5 minutes or so. The urine was passed out of the chamber and the nitrogen content measured. JS Haldane's experiments on goats gave a 75 minute half life. From his mathematical calculations he showed that, if the body had an equal perfusion, then this would represent a 10 minute half time. I have not been able to find the origin of his 40 minute half time.

Funding for Haldane's research was both private and from the Admiralty. At first Haldane started by using small animals. Later he used a large chamber, donated by Ludwig Mond, based at the Lister Institute in London. Haldane worked with Boycott, who was Professor of Physiology at Oxford University, and Damant, who was a Lieutenant in the Royal Navy (RN) and Inspector of Diving. When the tables were developed, Damant and Mr Catto, Gunner, RN, were the divers who tested the tables in London and in the water in Scotland. Damant was also involved with the second RN Deep Diving Unit in the 1930s. In the 1930s Damant modified the Haldane tables so that RN divers could dive to 300 feet on air.

The experiments

Haldane's team used goats because they were the largest animals which could be conveniently dealt with. They were also relatively cheap. Experience had shown that in smaller animals symptoms of were not easily detected. Haldane used 5 to 8 goats for each experimental dive. He realised that the goats had to be fit. The pressures that the goats, and humans, were exposed to were recorded in pounds per square inch of gauge pressure usually referred to as *lbs*. Sometimes they were quoted as *atmospheres absolute*. Haldane used *15 lb* as the conversion for one atmosphere. In this paper pressures are given as absolute pressure in bar and as sea water depths in metres {m} and feet {ft} with the original pressures in brackets [*and in italics*].

There were four series of goat experiments. In Series 1, 24 goats were exposed to 6 bar {50 m, 165 ft} [*75 lbs (6 atmospheres absolute)*] for 12, 15 and 30 minutes and then decompressed, either in stages or at a uniform rate. Actually there were 164 experiments on 34 animals but the results were inconclusive, because of poor planning, so only those experiments which showed the difference between staged and uniform decompression were retained for use. Series 2 was 20 goats exposed to 6 bar {50 m, 165 ft} for 15, 30, 60, 120 and 240 minutes and then decompressed either in stages or at a uniform rate. Series 3 had 15 goats exposed to 4 bar [*45 lbs (4 atmospheres absolute)*] {30 m, 100 ft} for 15, 30, 45, 60, 90, 120, 240 and 480 minutes. Most of the series 4 goats had been compressed in series 3. Series 4 exposed a total of 26 goats of a range of pressures; 6 bar {50 m, 165 ft} for 0.5, 3, 6, 10, 15, 30, 30, 60, 120, and 180 minutes; 4.4 bar (*51 lbs*) {34 m, 112 ft} for 180 minutes and then decompressed to the surface (1 bar) in 4 minutes (2 goats died, six developed bends and two had no symptoms); 4 bar {30 m, 100 ft} for 120 minutes then decompressed to 1.4 bar (*6 lbs*) {4 m, 12 ft} in six minutes (one animal died, one became paraplegic and the third developed bends); 3.6 bar {26 m, 86 ft} for 120 minutes then decompressed to 1.4 bar (*6 lbs*) {4 m, 12 ft} in 6.5 minutes (one goat had no symptoms and the other 3 developed bends and dyspnoea); 3 bar (*30 lbs*) (20 m or 66 ft) for 60 minutes with uniform rate decompression in 10 minutes gave 4 bent goats and 16 without symptoms; 2.7 bar (*25 lbs*) (17 m or 56 ft) for 240 minutes with decompression in 2 minutes produced 2 bent goats and 21 undamaged; and 2.3 bar (*20 lbs*) (13 m or 44 ft) for 240 minutes with decompression in 2 minutes bent one goat out of 22.

The chamber was not ventilated until decompression was started unless the exposure was over 4 hours when CO_2 levels tended to rise above 2% of atmospheric. There was no temperature control in the chamber. At the end of the dive, the goats were allowed to run free, but were closely observed for about 30 minutes, and then observed frequently after that. They had one pressure exposure per day, and after that they were allowed to roam free for a week before being used again in another experiment. Table X of Haldane et al.'s paper (reproduced in Table 1) records 57 different experiments involving 675 goat dives. After making allowances for goats used in both series 3 and 4 of the experiments, each goat must have done about 10 dives. It was a magnificent and marathon effort.

Decompression symptoms

Haldane' list of symptoms of decompression sickness in goats are quite similar to those in human beings. The words in italics are taken directly from the published work.[2]

1 Bends. The commonest symptom which we have observed consists of the exhibition of signs indicating that the animal feels uneasy in one or more of its legs. The limb, most commonly a fore-leg, is held up prominently in the air, and the animal is evidently loth to bear weight upon it. .. "Bends in parts of the body other than the limbs are very difficult to identify in animals; we have however occasionally noted symptoms which might be bends in the trunk, though we are not prepared to definitely identify them as such.

2 Temporary paralysis may be of two kinds. ... The animal. while showing no signs of general illness, or in other instances having already had bends, exhibits foot-drop or a more extensive palsy in one or more hind- or fore-limb. The paralysis does not usually come on till about 15 minutes after decompression, rapidly becomes more marked for a few minutes after the first signs are noted, and then soon begins to mend, so that there is marked improvement in about half an hour, and by next day the animal is found quite well. showed some improvement within 30 minutes, and 24 hours later they had made a spontaneous improvement, and were found to be otherwise well. This form of paralysis chiefly involves the hind legs.

3 Pain. In some cases the animals have shown signs of acute pain by urgent bleating and continuous restlessness. .. In other instances animals showing only severe bends bleat in a most distressing manner and are evidently in acute pain; at the same time they may gnaw at some part of their body (such as the testicles) as if localising the origin of the pain. In animals which have recovered, we have not had any instance where these signs persisted for more than 10 or 15 minutes.

4 Permanent paralysis. The onset is usually immediately after decompression, the condition is complete from the first and for at leas several days there are no signs of improvement. In a few cases the first paralysis has passed off (to all appearances completely) in two or three hours and the animal has been found next morning to be again paralysed. This second paralysis is permanent. A similar

history has often been noted in human cases.....In the most severe cases the animals have been killed; others have however begun to mend and have lived for some months with a slight spastic paralysis of the hind legs. .. In some there has been retention of urine, one animal had to be killed on account of acute distension of the stomach which came on some 20 hours after the onset of the paraplegia.

5 A fair number of cases have occurred where the animal has been obviously ill, but in which it has been impossible to identify any definite local symptoms or any definite dyspnoea. The goat may lie down, refuse to move or be tempted with corn (of which goats are inordinately fond) sometimes lying extended on the side, sometimes hurriedly rising, walking a few steps and then lying down again.

6 Dyspnoea is usually the precursor of death and only a minority of goats survived after showing clear dyspnoea. ....The delay in the onset of first symptoms is often most striking; the animal may appear quite normal for as long as 10 or 15 minutes, dyspnoea then appears, the goat falls down helpless and in another 15 minutes is dead.

Results

Table 1 (page 48) shows that these were deep diving goats. Thirty seven of the experiments were at *75 lb* {6 bar, 50 m, 165 ft}. There was one experiment at *51 lb* {4.4 bar, 34 m, 112 ft} where the exposure was 180 minutes and the decompression took 4 minutes. Not surprisingly the 10 goats provided 2 bends, one bad bend, one was obviously ill, two developed paraplegia and two died. Sixteen experiments were at *45 lb* {4 bar, 30 m, 100 ft}.

Only three experiments were at less than 30 lbs gauge pressure (3 bar), equivalent to 20 m or 66 ft. These were one at 20 m for 60 minutes with 10 minute uniform decompression which produced bends, as defined above, in 4 out of 19 goats (21%). Another, with 23 goats at 25 lb {2.7 bar, 17 m, 56 ft} for 240 minutes with a 2 minute decompression, gave 9% bends. The third, with 22 goats at 20 lb {2.3 bar, 13 m, 43 ft} for 240 minutes with a 2 minute decompression produced 5% bends. Over all these experiments on 64 goats resulted in 7 cases of bends (11%).

Haldane's conclusions about goats from his experiments included:

The variation in the individual susceptibility of different goats is very marked. ... The complete explanation of this individual variation in susceptibility probably requires a knowledge of the details of caisson disease far beyond that which we at present possess. Data exist, however, on which the influence of several factors may be discussed.

It would appear that there is no clear difference between the sexes in liability to decompression symptoms in general. The experiments suggested however that under certain circumstances there might be a marked difference in the susceptibility to death. ...in Series II are shown 1 death in 7 males and 4 deaths in 11 females. All these last four animals were to some degree advanced in pregnancy, and their mortality is very probably to be associated with this condition, which, in the goat, is accompanied by a marked increase in the subcutaneous and intra-abdominal fat.

The only conclusion to be drawn is that these figures do not indicate that either sex or weight was a determining factor in the incidence of decompression symptoms.

Conclusions. Of the four factors considered in detail, it appears therefore that age, sex and blood volume were without appreciable influence. Pregnancy and a low rate of expiratory exchange seem to favour the occurrence of symptoms.

Pathology of caisson disease

When discussing the pathology of caisson disease in goats Haldane concluded that:

There is not much doubt that some of our animals which showed no symptoms must have had bubbles present in the blood.

In our own animals attempts were made to see bubbles in the retinal vessels during life. Though an excellent view of the fundus may easily be obtained, no bubbles were ever seen in animals with severe dyspnoea, so the method cannot be taken as giving any indication of the absence of bubbles in the blood. Some of these animals died and plenty of free gas was found in the retinal vessels post-mortem.

.. goats were killed before the expiration of the appropriate period for the development of bends. The experience of similar experiments indicates that they might have shown symptoms if they had not been killed. Yet three of four had no bubbles in the blood.

Synovial fluid is almost always full of bubbles; exposure for 15 minutes at 75 lbs.[6.1 atmospheres absolute or 28 fathoms = 168 feet] (51 m) is sufficient to cause their presence, while decompression in 100 minutes uniformly is not enough to prevent their formation. In animals which have died within 3 hours of decompression, we have found them in every case.

Solid organs. Fat commonly shows bubbles, often in extreme abundance. They are more numerous in the abdominal than in the subcutaneous fat; the latter is much

TABLE 1

Pressure lbs positive	Compression minutes	Actual exposure minutes	Decompression minutes	No. of goats	No symptoms		Bends					Temporary paralysis	Various indefinite	Paraplegia	Dyspnoea	Total severe symptoms	Death
					Number	Percent	?	Slight	"Bends"	Bad	Total						
75	6	0.5	60 un	8	7	87			1		1					0	0
		1	1	6	6	100					0					0	0
		3	1	5	4	80					0		1			1	0
		3	10 un	2	2	100					0					0	0
		6	1	6	6	l00					0					0	0
		10	1	7	6	86					0			1		1	0
		15	1	6	2	33	1		1		2	1				1	1
		15	10 un	7	2	29			3		3		1			1	1
		15	31 st	34	29	85		2	2	1	5				0	0	
		15	31 un	36	19	53	2	3	8		13		1	2		3	1
		15	90 un	12	9	75			3		3					*	*
		30	31 st	23	12	52			7	1	8	3				3	0
		30	31 un	6	1	17			3	1	4	1				1	0
		30	68 st	14	14	100					0					0	0
		30	68 uu	14	7	50			7		7					0	0
		60	31 st	22	15	68			3	1	4	1	1		1	3	0
		120	31 st	9	0	0				4	3	7			1	1	1
		120	70 st	14	9	64			4		4				1	1	0
		120	70 un	13	4	31		1	6		7	1		1		2	0
		120	92 st	19	15	79			3		3	1				1	0
		120	100 un	19	10	53			1	2	3	2	1		2	5	1
		180	134 st	14	12	86			2		2					0	0
		180	134 un	10	5	50	1	1	3		5					0	0
		240	31 st	8	2	25			3	1	4	1				1	1
		240	31 un	4	0	0				2	2	1				1	1
51	6	180	4	10	2	20			2	1	3		1		2	3	2
45	6	15	2	15	14	93			1		1					0	0
		30	2	15	12	80			3		3					0	0
		45	30 st	14	14	100					0					0	0
		60	1	13	10	77			4		4					0	0
		60	10 un	13	7	54			4	1	5			1		1	0
		60	30 st	13	9	69		1	3		4					0	0
		60	52 st	13	10	77		2	1		3					0	0
		60	30 st	8	5	62			1	1	2		1			1	0
		120	1	10	4	40			1	1	2	1		3		4	0
		120	10 un	12	6	50			4		4	1			1	2	0
		120	30 st	13	12	92			1		1					0	0
		120	57 st	15	13	87			2		2					0	0
		240	10 un	11	6	55			4		4	1				1	0
		240	30 st	13	11	85			2		2					0	0
		240	62 st	15	9	60			6		6					0	0
		480	10 un	11	6	55			3		3	2				2	0
30	6	60	10 un	19	15	79			4		4					0	0
25		240	2	23	21	91			2		2					0	0
20		240	2	22	21	95			1		1					0	0
75	39	1	7 un	8	7	87					0			1		1	0
		10	10 un	4	3	75					0					0	1
		12	45 st	4	1	25			3		3					0	0
		12	45 un	6	4	67			2		2					0	0
		15	45 st	6	4	67			2		2					0	0
		15	45 un	12	4	33			8		8					0	0
		30	45 st	11	7	64			3	1	4					0	0
		30	45 un	12	5	42			5	1	6				1	1	0
		30	10 un	4	0	0					0					0	4
		30	10 un#	4	4	100					0					0	0
		30	10 un##	4	0	0			2		2			1		1	1
		60	75 un	4	2	50			1		1					0	1

* These two entries were left blank in the original publication. # Recompressed at once to 15 lbs for 32 minutes. ## Recompressed to 15 lbs for 37 minutes 18 minutes after decompression. un = uniform decompression: st =decompression by stages.

more vascular. Other solid organs for the most part show no bubbles outside the blood vessels.

Among the solid organs, bubbles outside the vessels are found most frequently in the central nervous system.

Duration of bubbles. It is difficult to say how long bubbles may remain the vessels and tissues after their first formation in animals which survive. [Zografidi (Revue de Médecine, 1907, p 159) records the finding of numerous bubbles in the peripheral vessels, but not the heart, of a diver who was paralysed and died 33 days after decompression!] The question is much complicated by the fact that we have reason to believe bubbles may continue to form for a long, and quite unknown, time after decompression.

..bubbles have been found in the blood of one animal which died two days after decompression (and that in an animal which had shown no dyspnoea) and in the joints up to 26 hours. In the spinal cord bubbles may persist much longer: in two cases we have found them 15 days after the last exposure to pressure and in 27 days after the last occurrence of symptoms.

Haldane's tables

The Haldanian staged decompression tables were tested at 7 different profiles, in the chamber, with Lieutenant Damant and Mr G V Catto, Gunner RN, as the subjects. The profiles are shown in Table 2, which has been constructed from Appendix 1.[2] The pressures ranged from 26-53 m {86-175 ft}. The compression times were much longer than most of the goat dives. It is not clear whether this was a result of inadequate compressors or was deliberate. Neither man developed symptoms.

Then Damant and Catto did 20 test deep dives from HMS SPANKER off Rothsay, Isle of Bute, in Scotland. These dives are shown in Table 3, constructed from Appendix 2 (page 50).[2] These were without incidents of decompression sickness. But on one of them the diver, Mr Catto, became entangled and the planned bottom time of 12 minutes was extended to 31.5 minutes, but by extending the decompression time to one and a half hours the diver was recovered without symptoms. These dives were done using hand pumps with a crew of six for each pump. Pumps in those days usually had a handle on a wheel on each side so that two men could work at once. At depth they had to maintain 24 revolutions a minute to supply the diver with adequate air. The men had to be relieved every five minutes.

Nowadays a total of 27 bends-free dives would not be enough to establish the safety of a new dive table.

Haldane's tables, published as Tables 1 and 2 in Appendix 4 of the report, are reproduced here as Tables 4 (page 51) and 5 (page 52).[2] Haldane's Table 1, headed *Stoppages during the ascent of a diver after ordinary limits of time from surface*, was limited to 33 minutes of staged decompression. It went down to 34 fathoms (or 204 feet = 61.3 m) where it allowed 12 minutes with 32 minutes of decompression with six stops starting at *60 ft* (18 m).

Haldane's Table 2, headed *Stoppages during the ascent of a diver after delay beyond the ordinary limits of time from surface for longer times at depth*, allowed over an hour at 34 fathoms (or 204 feet = 61.3 m) with a decompression time of 238 minutes. and gave a greater risk of decompression sickness. The original published table has an arithmetical error in the last three feet-measured entries. They are two feet deeper than the fathom depths

TABLE 2

EXPERIMENTS CARRIED IN THE CHAMBER AT THE LISTER INSTITUTE 1906

Subjects Lieutenant Damant and Mr A.Y.Catto, Gunner, R.N.

Date	Start of dive	S I h.min	Pressure	Depth m	Depth feet	Compression time	Time at depth	Bottom time	Deco time	Stops
25th July	NR		39 lb	26	86	17	60	77	24	2
26th July	1037		50 lb	33	110	24	27	51	34	4
26th July	1503	3.01	55 lb	36	121	28	19	47	31	4
27th July	1029	18.08	60 lb	40	132	30.5	20	50.5	37.5	5
27th July	1537	3.40	67 lb	45	147	36	18	54	36	5
30th July	1057	18.50	74 lb	49	163	39	15	54	42	6
31st July	1100	22.27	80 lb	53	176	44	12	56	51	7

NR = Not recorded.

TABLE 3

DEEP DIVING EXPERIMENTS CARRIED OUT OFF ROTHSAY, ISLE OF BUTE, FROM HMS SPANKER, 1906

Diver	Date	Time of dive	Depth			Bottom	Decompression time	
			Fathoms	Feet	m	time	Total	Stops
Damant	21st August	p.m.	15	90	27	62	18.5	2
Catto	21st August	p.m.	15	90	27	61.5	17.5	2
Catto	22nd August	a.m.	23	138	42	22	35.5	5
Damant	22nd August	p.m.	23	138	42	22.5	32.5	4
Damant	23rd August		25	150	45.5	20.75	37.75	5
Catto	23rd August		25	150	45.5	21.25	37.75	5
Catto	24th August	a.m.	27	162	49	18.5	55.5	5
Damant	24th August	p.m.	27	162	49	17.75	44.5	6
Catto	25th August		29	174	53	17.5	46	6
Damant	25th August		29	174	53	15	48.5	6
Catto	27th August	1123.25	30	180	54.5	13.5	45.75	6
Damant	27th August	1415.75	30	180	54.5	13.25	44.5	6
Damant	28th August	1018.5	30	180	54.5	16	47	NR
Catto*	28th August	1417	30	180	54.5	31.5	90	9
Catto	30th August	a.m.	30	180	54.5	15.75	46.25	6
Damant	30th August	p.m.	30	180	54.5	14.3	46.25	6
Damant	31st August	1108.25	36	216	65	8	48.5	7
Catto	31st August	1412.75	35	210	63	7.75	50.25	7
Catto**	3rd September	1426		142	43	19	38	NR
Damant**	3rd September	1503.5		139	42	70#	39	NR

*1431.75 *Diver called up , but could not come up as he was foul, until* 1448.5
** This was a working dive raising a 56 lb. weight *by meas of an arrangement of rope and pulleys. The heavy rope and blocks used caused great friction and resistance.* The height that the weight was raised was observed on deck.
#This bottom time is improbable given the ascent time and the notation *No ill-effects*. The record shows Damant left the surface at 3.03, arrived on the bottom at 3.05 and took the first sample at 4.00. The likely explanation is that he left the surface at 3.53 arrived on the bottom at 3.55 and took the first sample after 5 minutes. This would give a bottom time of 19 minutes, the same as Mr Catto's dive.
NR = Not recorded.

The Haldane tables have now been modified so often that they now bear very little resemblance to the originals. But the assumptions and theory used to produce them have endured. At present the air tables produced by the Canadian Defence and Civil Institute of Environmental Medicine (DCIEM) are considered to be the most conservative available. They have been developed from the results of thousands of closely monitored dives undertaken in Canada, the USA and the UK. Table 6 (pages 53 and 54) compares some of the profiles in Haldane's Tables I and II with similar profiles in the DCIEM tables. In table 6 the top line of each pair of entries is taken from the Haldane Tables and printed in ***bold italics***. The lower line in from the DCIEM tables. Times that are taken from Haldane's Table II, for *the ascent of a diver after delay beyond the ordinary limits of time from surface* or below the limiting line, which should only be exceeded under unusual circumstances, in the DCIEM tables are marked*.

Why blame the diver

Why are divers blamed for developing decompression sickness? There is a widespread attitude that a diver must have done something wrong to develop decompression problems, although we know that both bubble formation and decompression illness are random events.

I think Haldane probably started it by stating "*so the compressed air illness has now practically disappeared, except in isolated cases, where from one quarter or another the regulations have not been carried out*".[4] He had so much faith in his tables which had vastly reduced the incidence of decompression problems.

In 1922 Haldane recommended recompression treatment, but had little to do with experiments in

TABLE 4

[Table 1 of Appendix IV (page 442)]

STOPPAGES DURING THE ASCENT OF A DIVER AFTER ORDINARY LIMITS OF TIME FROM SURFACE.

Depth Feet	Depth Fathoms	Pressure Pounds per square inch	Time from surface to beginning of ascent	Approximate time to first stop	Stoppages in minutes at different depths* 60 ft.	50 ft.	40 ft.	30 ft.	20 ft.	10 ft.	Total time for ascent in mins.
0-36	0-6	0-16	No limit	-	-	-	-	-	-	-	0-1
36-42	6-7	16-18.5	Over 3 hours	1	-	-	-	-	-	5	6
42-48	7-8	18.5-21	Up to 1 hour	-	-	-	-	-	-	-	1.5
			1-3 hours	1.5	-	-	-	-	-	5	6.5
			Over 3 hours	1.5	-	-	-	-	-	10	11.5
48-54	8-9	21-24	Up to 0.5 hour	-	-	-	-	-	-	-	2
			0.5-1.5 hours	2	-	-	-	-	-	5	7
			1.5-3 hours	2	-	-	-	-	-	10	12
			Over 3 hours	2	-	-	-	-	-	20	22
54-60	9-10	24-26.5	Up to 20 mins.	-	-	-	-	-	-	-	2
			20-45 mins.	2	-	-	-	-	-	5	7
			0.75-1.5 hours	2	-	-	-	-	-	10	12
			1.5-3 hours	2	-	-	-	-	5	15	22
			Over 3 hours	2	-	-	-	-	10	20	32
60-66	10-11	26.5-29.5	Up to 0.25 hour	2	-	-	-	-	-	-	2
			0.25-0.5 hour	2	-	-	-	-	-	5	7
			0.5-1 hour	2	-	-	-	-	3	10	15
			1-2 hours	2	-	-	-	-	5	15	22
			2-3 hours	2	-	-	-	-	10	20	32
66-72	11-12	29.5-32	Up to 0.25 hour	2	-	-	-	-	-	2	4
			0.25-0.5 hour	2	-	-	-	-	3	5	10
			0.5-1 hour	2	-	-	-	-	5	12	19
			1-2 hours	2	-	-	-	-	10	20	32
72-78	12-13	32-34.5	Up to 20 mins.	2	-	-	-	-	-	5	7
			20-45 mins.	2	-	-	-	-	5	10	17
			0.75-1.5 hours	2	-	-	-	-	10	20	32
78-84	13-14	34.5-37	Up to 20 mins.	2	-	-	-	-	-	5	7
			20-45 mins.	2	-	-	-	-	5	15	22
			0.75-1.25 hours	2	-	-	-	-	10	20	32
84-90	14-15	37-40	Up to 10 mins.	2	-	-	-	-	-	3	5
			10-20 mins.	2	-	-	-	-	3	5	10
			20-40 mins.	2	-	-	-	-	5	15	22
			40-60 mins.	2	-	-	-	3	10	15	30
90-96	15-16	40-42.5	Up to 10 mins.	3	-	-	-	-	-	3	6
			10-20 mins.	2	-	-	-	-	3	5	10
			20-35 mins.	2	-	-	-	-	5	15	22
			35-55 mins.	2	-	-	-	3	10	15	30
96-108	16-18	42.5-48	Up to 15 mins.	3	-	-	-	-	3	5	11
			15-30 mins.	3	-	-	-	3	7	10	23
			30-40 mins.	3	-	-	-	5	10	15	33
108-120	18-20	48-53.5	Up to 15 mins.	3	-	-	-	2	3	7	15
			15-25 mins.	3	-	-	-	5	5	10	23
			25-35 mins.	3	-	-	-	5	10	15	33
120-132	20-22	53.5-59	Up to 15 mins.	3	-	-	-	2	5	7	17
			15-30 mins.	3	-	-	-	5	10	15	33
132-144	22-24	59-64.5	Up to 12 mins.	3	-	-	-	3	5	5	16
			12-25 mins.	3			2	5	10	12	32
144-156	24-26	64.5-70	Up to 10 mins.	3	-	-	-	3	5	5	16
			10-20 mins.	3	-	-	2	5	10	12	32
156-168	26-28	70-75	Up to 10 mins.	3	-	-	2	3	5	5	18
			10-16 mins.	3	-	2	3	5	7	10	30
168-180	28-30	75-80.5	Up to 9 mins.	3	-	-	2	3	5	5	18
			9-14 mins.	3	-	2	3	5	7	10	30
180-192	30-32	80.5-86	Up to 13 mins.	3	-	2	3	5	7	10	30
192-204	32-34	86-91.5	Up to 12 mins.	3	2	2	3	5	7	10	32

* During each stoppage the diver should continue to move his arms and legs.

TABLE 5

[Table II of Appendix IV (page 443)]

STOPPAGES DURING THE ASCENT OF A DIVER AFTER DELAY BEYOND THE ORDINARY LIMITS OF TIME FROM SURFACE

Depth Feet	Depth Fathoms	Pressure Pounds per square inch	Time fom surface to beginning of ascent	Approximate time to first stop	Stoppages in minutes at different depths 80ft	70ft	60ft	50ft	40ft	30ft	20ft	10ft	Total time for ascent mins.
60-66	10-11	26.5-29.5	Over 3 hours	2	-	-	-	-	-	-	10	30	42
66-72	11-12	29.5-32	2-3 hours	2	-	-	-	-	-	-	10	30	42
			Over 3 hours	2	-	-	-	-	-	-	20	30	52
72-78	12-13	32-34.5	1.5-2.5 hours	2	-	-	-	-	-	-	20	25	47
			Over 2.5 hours	2	-	-	-	-	-	-	30	30	62
78-84	13-14	34.5-37	1.25-2 hours	2	-	-	-	-	-	-	15	30	47
			2-3 hours	2	-	-	-	-	-	5	30	30	67
			Over 3 hours	2	-	-	-	-	-	10	30	35	77
84-90	14-15	37-40	1-1.5 hours	2	-	-	-	-	-	5	15	25	47
			1.5-2.5 hours	2	-	-	-	-	-	5	30	35	72
			Over 2.5 hours	2	-	-	-	-	-	20	35	35	92
90-96	15-16	40-42.5	1-1.5 hours	2	-	-	-	-	-	5	15	30	52
			1.5-2.5 hours	2	-	-	-	-	-	10	30	35	77
			Over 2.5 hours	2	-	-	-	-	-	30	35	35	102
96-108	16-18	42.5-48	40-60 minutes	2	-	-	-	-	-	10	15	20	47
			1-2 hours	2	-	-	-	-	5	15	25	35	82
			Over 2 hours	2	-	-	-	-	15	30	35	40	122
108-120	18-20	48-53.5	35-60 minutes	2	-	-	-	-	5	10	15	25	57
			1-2 hours	2	-	-	-	-	10	20	30	35	97
			Over 2 hours	2	-	-	-	-	30	35	35	40	142
120-132	20-22	53.5-59	0.5-0.75 hours	3	-	-	-	-	5	10	15	20	53
			0.75-1.5 hours	3	-	-	-	5	10	20	30	30	98
			Over 1.5 hours	3	-	-	-	15	30	35	40	40	163
132-144	22-24	59-64.5	25-45 minutes	3	-	-	-	3	5	10	15	25	61
			0.75-1.5 hours	3	-	-	-	10	10	20	30	35	108
			Over 1.5 hours	3	-	-	-	30	30	35	40	40	178
144-156	24-26	64.5-70	20-35 minutes	3	-	-	-	3	5	10	15	20	56
			35-60 minutes	3	-	-	-	7	10	15	30	30	95
			Over 1 hour	3	-	-	20	25	30	35	40	40	193
156-168	26-28	70-75	16-30 minutes	3	-		-	3	5	10	15	20	56
			0.5-1 hour	3	-	-	3	10	10	15	30	30	101
			Over 1 hour	3	-	5	25	25	30	35	40	40	203
168-182* (168-180)	28-30	75-80.5	14-20 minutes	3	-	-	-	3	3	7	10	15	41
			20-30 minutes	3	-	-	2	2	3	10	15	25	60
			0.5-1 hour	3	-	3	3	7	10	20	30	35	111
			Over 1 hour	3	-	15	25	30	30	35	40	40	218
182-194* (180-192)	30-32	80.5-86	13-20 minutes	3	-	-	-	3	3	7	15	15	46
			20-30 minutes	3	-	-	3	3	5	10	15	25	64
			0.5-1 hour	3	-	3	5	10	12	20	30	35	118
			Over 1 hour	3	5	20	25	30	30	35	40	40	228
194-206* (192-204)	32-34	86-91.5	12-20 minutes	3	-	-	3	3	5	7	10	20	51
			20-30 minutes	3	-	3	3	3	5	10	20	20	67
			0.5-1 hour	3	3	3	5	10	15	20	30	35	124
			Over 1 hour	3	15	20	25	30	30	35	40	40	238

* These original figures are incorrect. The correct figures are bracketed below.

TABLE 6
COMPARISON OF DECOMPRESSION REQUIREMENTS *HALDANE 1908* AND DCIEM 1999
*** Depth and time taken from Haldane's Table II or time is below the limiting line in DCIEM tables**

Depth		Bottom	Time to	Stop times (minutes) at different depths (feet)								Total
feet	m	time	1st stop	80	70	60	50	40	30	20	10	time
50	***15***	***60***	***2***								***5***	***7***
50	15	60										1
50	***15***	***180***	***2***								***10***	***12***
50	15	*180									43	43
50	***15***	***280***	***2***								***20***	***22***
50	15	*280									97	97
60	***18***	***45***	***2***								***5***	***7***
60	18	45										1
60	***18***	***90***	***2***								***10***	***12***
60	18	90									19	19
60	***18***	****180***	***2***							***5***	***15***	***22***
60	18	*180								5	77	82
70	***21***	***15***	***2***								***2***	***4***
70	21	15										1
70	***21***	***30***	***2***							***3***	***5***	***10***
70	21	30										1
70	***21***	***60***	***2***							***5***	***12***	***19***
70	21	60								2	11	13
70	***21***	***120***	***2***							***10***	***20***	***32***
70	21	*120								8	56	64
80	***24***	***20***	***2***								***5***	***7***
80	24	20										2
80	***24***	***45***	***2***							***5***	***15***	***22***
80	24	45								4	12	16
80	***24***	***75***	***2***							***10***	***20***	***32***
80	24	75								9	35	44
80	***24***	***120***	***2***							***15***	***30***	***47***
80	24	*120							3	20	76	99
90	***27***	***10***	***2***								***3***	***5***
90	27	10										2
90	***27***	***20***	***2***							***3***	***5***	***10***
90	27	20										2
90	***27***	***40***	***2***							***5***	***15***	***22***
90	27	40								6	11	17
90	***27***	***60***	***2***						***3***	***10***	***15***	***30***
90	27	60							2	9	32	43
90	***27***	****90***	***2***						***5***	***15***	***25***	***47***
90	27	*90							6	15	62	83
100	***30***	***15***	***3***							***3***	***5***	***11***
100	30	15										2
100	***30***	***30***	***3***						***3***	***7***	***7***	***23***
100	30	30								6	10	16
100	***30***	***40***	***3***						***5***	***10***	***15***	***33***
100	30	40								9	18	27

TABLE 6 (Continued)
COMPARISON OF DECOMPRESSION REQUIREMENTS *HALDANE 1908* AND DCIEM 1999
*** Depth and time taken from Haldane's Table II or time is below the limiting line in DCIEM tables**

Depth		Bottom	Time to	Stop times (minutes) at different depths (feet)								Total
feet	m	time	1st stop	80	70	60	50	40	30	20	10	time
100	***30***	****60***	***2***						***10***	***15***	***20***	***47***
100	30	*60							6	9	43	58
100	***30***	***110***	***2***					***5***	***15***	***25***	***35***	***82***
100	30	*110						4	9	39	112	164
110	***33***	***35***	***3***						***5***	***10***	***15***	***33***
110	33	35							4	7	19	30
110	***33***	****110***	***2***					***10***	***20***	***30***	***35***	***97***
110	33	*110						8	16	50	136	210
120	***36***	***30***	***3***						***5***	***10***	***15***	***33***
120	36	30							5	7	17	29
120	***36***	****60***	***2***					***5***	***10***	***15***	***25***	***57***
120	36	*60						6	8	19	61	94
130	***39***	***30***	***2***						***5***	***10***	***15***	***33***
130	39	30							7	8	23	38
130	***39***	****90***	***3***				***5***	***10***	***20***	***30***	***30***	***98***
130	39	*90					7	7	22	54	144	234
140	***42***	***25***	***3***					***2***	***5***	***10***	***12***	***32***
140	42	25							7	8	19	34
140	***42***	****90***	***3***				***10***	***10***	***20***	***30***	***35***	***108***
140	42	*90				4	6	8	30	68	157	273
150	***45***	***20***	***3***					***2***	***5***	***10***	***12***	***32***
150	45	20							6	8	11	24
150	***45***	****90***	***3***				***5***	***10***	***20***	***30***	***30***	***98***
150	45	*90					7	7	22	54	144	234
160	***48***	***15***	***3***				***2***	***3***	***5***	***7***	***10***	***30***
160	48	15							7	7	10	23
160	***48***	****60***	***3***			***3***	***10***	***10***	***15***	***30***	***30***	***101***
160	48	*60				6	6	7	18	45	129	211
170	***51***	***14***	***3***				***2***	***3***	***5***	***7***	***10***	***30***
170	51	14							6	7	10	23
170	***51***	****60***	***3***		***3***	***3***	***7***	***10***	***20***	***30***	***35***	***111***
170	51	*60			4	5	6	8	23	54	144	244
180	***54***	***13***	***3***				***2***	***3***	***5***	***7***	***10***	***30***
180	54	13							8	7	11	26
180	***54***	****60***	***3***		***3***	***3***	***7***	***10***	***20***	***30***	***35***	***111***
180	54	*60		3	3	5	7	9	29	65	155	276
190	***57***	***13***	***3***				***2***	***3***	***5***	***7***	***10***	***30***
190	57	*13					4	5	6	9	31	55
190	***57***	****55***	***3***		***3***	***5***	***10***	***12***	***20***	***30***	***35***	***118***
190	57	*55		4	4	5	6	10	28	65	154	276
200	***60***	***12***	***3***			***2***	***2***	***3***	***5***	***7***	***10***	***32***
200	60	*12						6	5	8	18	37
200	***60***	****50***	***3***	***3***	***3***	***5***	***10***	***15***	***20***	***30***	***35***	***124***
200	60	*50		5	4	5	6	10	27	62	153	272

recompression treatment of decompression sickness. He said "*the trouble, however, about the use of recompression chambers is that it is very often very difficult to get the patient out without the symptoms recurring, and that decompression in these sorts of situations may take up to many days*".[5] Also he recommended underwater recompression if no chamber was available, but he qualified that by saying that "*The trouble, however, is to get the man up again safely*".[6]

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ARTICLES OF INTEREST REPRINTED FROM OTHER JOURNALS

MECHANISMS OF EMBOLISM

R W (Bill) Hamilton

Key Words

Air embolism, decompression illness, medical conditions and problems, physiology, pulmonary barotrauma.

Decompression Illness: Mechanisms of Disease II is a continuation of a course on decompression diseases carried on for many years along with the UHMS Annual Meeting under the direction of **David Elliott**, whose position was ably assumed by **James Francis** last year in Seattle. James said about the first session last year that he could relieve David but not replace him, but he is well on his way to establishing a commendable course pattern of his own. Last year began with a description of decompression illness (DCI) from the point of view of how it comes about, considering the formation, growth, and behaviour of bubbles, and some of their overall consequences.

This year's course got out the magnifying glass and focused on embolism, or embolic phenomena. The subject was tackled by a strong group, including some regulars, some new faces, and a couple of old friends who had not been among us as often recently as we would like. The main target of gas embolism is the brain, and effects there were examined in detail, but the course began with a careful look at the lung, inasmuch as pulmonary barotrauma is the source of most cerebral gas embolism in divers.

The lung was elegantly characterised by **David Denison** as many "densely branching and closely intertwined tubular trees" contained in a "flimsy bag". The continuing and rather magnificent function of this organ, described at one point as an invagination, was covered with particular attention to the surface tension effects on the alveoli. Lung function is assessed by three main categories of test: blowing in a bag, breathing inside a box (a body plethysmograph), and use of a tracer (CO) to measure diffusion. This broncho-alveolar tree ruptures at a differential pressure of as little as 70 mmHg (10 kPa or about 1 msw). Most such overpressures are caused by behaviour rather than disease.

Tom Neuman looked at the clinical features of pulmonary barotrauma, beginning with the frustration that accompanies having neither a clear clinical definition nor a reliable incidence of the condition. He cites studies showing radiologically detectable gas in otherwise asymptomatic submarine-escape trainees at about a 1% level, suggesting it may be more common than we think. Gas can take many routes once it escapes the lung and, fortunately, many of these have benign consequences. When bubbles get into the blood, there can be serious or even fatal results.

Eric Kindwall asked if the lung ruptures in compression chambers, then answered "almost never". He reports a couple of cases, one in a tracheotomy patient with

a lung tumour and thick mucus secretions. Another patient in a monoplace chamber had a seizure and was decompressed immediately, with the end result of a fatal cardiac arrest. Eric knows of no cases where there have been such problems during the clonic phase. Another couple of cases were also categorised as "deserved" (or better, "expected"). In both tunnel work and clinical hyperbaric therapy, the risk of burst lung is "vanishingly small".

David Denison came back to cover lung rupture in other circumstances, converging mainly on pneumothorax, which may be spontaneous. These occur commonly in clinical practice, but systemic arterial gas embolism is much less common than in divers. Heavy expiratory efforts can bring on surgical emphysema, for example, in such persons as mothers giving birth, musicians playing the trumpet, and divers skip breathing with deep breaths. "Lung function tests can be bad for your health." He recommends against taking really deep breaths under water. Obstructive lung disease seems not to predispose to pulmonary barotrauma, but restrictive lung disease does.

Ian Calder reported from his perspective on "material which the Great Reaper provides". He has seen some 200 diving-associated deaths, 16% of which had lung damage as a major contributor. He points out that special care is necessary to make the diagnosis properly, including post-mortem, pre-autopsy radiography. Interestingly, he finds little evidence of pulmonary barotrauma in areas of fibrosis.

Our Director, **James Francis,** looked into possible mechanisms for lung rupture. With **Peter Benton**, he took another look at the data from the British Navy's submarine escape tower. Out of 115,000 ascents (1975–97) there were 10 cases of pulmonary barotrauma, 6 diagnosed by x-ray detectable gas and 4 by symptoms. All 10 had a FVC lower than normal. For a few years, they disqualified as submariners those who had an FEV_1/FVC ratio < 75%, but later dropped that policy. He confirms what David Denison says about restriction rather than obstruction being guilty, i.e., that a stiffer lung is more susceptible. Aviators, who endure a greater Boyle's Law change than divers, rarely have lung barotrauma. The diver's lungs are filled with blood and have a negative 20 cm static lung loading, and are therefore stiffer. Further, because of blood pooling in the thorax, the diver's vasculature is more open to catch bubbles.

Discussion after these talks served for the most part to reinforce what had been said with additional examples. Maximum inspiration clearly is a risky activity in the water. An interesting point was made about scarring of the lung tissue. Calder says it makes the lung tissue stronger, but Denison points out that the tissue near the scarring is stretched so may fail; thus they were actually in agreement.

Simon Mitchell presented **Des Gorman**'s paper on the distribution of bubbles to the central nervous system. Autoregulation plays a big role in circulation through cerebral vessels, and this depends strongly on the endothelium and its biochemistry. Perhaps 80% of bubbles going to the cerebral circulation end up back at the right heart chamber. Almost all bubbles cleared by recompression do so in the first doubling of pressure.

Pulmonary specialist **Andy Veale** from Auckland (in what he characterised as "the asthma capital of the world"), New Zealand, took a sort of whole-body approach to risk management related to pulmonary barotrauma, sharing some gems of wisdom. For example, many beginning diving students (who have a lot of embolisms) do not really want to be there; he said the instructor might be better able to detect the embolism or its cause than the doctor. He considers diver-related factors more important than medical factors in setting people up for pulmonary barotrauma. In addition to various risk factors, he gave us some hints on management of cases of this type.

Tom Neuman described some other places where embolic bubbles may go in addition to the nervous system. These include muscles, heart, liver, and the vascular endothelium. Muscle is not particularly susceptible, but bubbles can invoke a rise in "tissue damage" enzymes. The heart itself is not very much at risk, nor is the liver. Outside of the nervous system, the vascular endothelium is the organ most injured by arterial gas embolism.

Arthur Dick, whom many will recall from his "diving doctor" days but who is now a neurologist, showed what various effects could occur from expected bubble distribution in the brain. He showed, for example, how a "watershed" area where circulations meet from different directions is more vulnerable to low blood pressure. Embolic occlusion leads to a central core of irreversible damage, and a surrounding area of relative hypoperfusion; cells there may die due to triggering of apoptosis (programmed cell death). Among sources of bubbles is the patent foramen ovale; some of these are being closed prophylactically. He mentions post-surgical neurological deficits that are probably due to small embolisms; devastating cerebral injury follows when larger volumes of air are introduced.

Drew Dutka, another neurologist, followed up with details on "post ischaemic hypoperfusion" and its mechanisms. He pointed out that gaseous and solid emboli behave differently, in part because of their immediate and widespread distribution and consequent disruption of the blood-brain barrier. Therefore, oedema and inflammation may play a greater role in gas embolism. Despite the possibility that hyperbaric oxygen could support injury by free radicals, the record shows that embolism patients treated with HBO_2 do much, much better than those who are not.

It should come as no surprise that **Simon Mitchell** said a few words about the anti-arrhythmic agent lidocaine (others mentioned it also) as adjunctive therapy for embolism. He mentioned an impressive list of things that lidocaine does as reasons why it should be helpful, supported by an extensive bibliography. Case reports and pre-post comparisons on heart surgery patients show distinct benefits, but it is difficult to get such data on divers. Other drugs with some of the same actions can also be beneficial.

This course was extremely well put together, with the various topics presented from various perspectives, adding up to an extremely good coverage of this important topic. As notable as the presenters was the fact that discussions included many others with a great deal of expertise to share on the subject.

The Director handed out individual preprints or abstracts of the author's topics that were extremely helpful. However, there was no written program listing the presenting authors and their topics, and this would have been helpful. Unfortunately no proceedings will be published; to do so is not consistent with this type of course.

Too often a patient experiencing cerebral arterial gas embolism with its dramatic and often devastating neurological symptoms is diagnosed as suffering from "pulmonary barotrauma". Pulmonary barotrauma is a likely cause, but that is a lung problem and not a brain problem, so is really not the disease. As far as this reporter could tell, not once in this entire course was this mistake made out loud. The two were kept in their proper places.

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WHEN DIVE TABLES ARE FOR SISSIES

Gordon Wright

Key Words
Incidents, environment, low air, tables.

Experienced divers might be able to teach newcomers the right way to dive, but, too often, their own diving practices expose novices to danger. Gordon Wright has seen the results from the point of view of both victim and rescuer.

The trouble with divers is that they don't practise what they preach. How many times when I was being trained did I hear: "Plan the dive and dive the plan", "Aim to surface with 50 bar", "Check tides and currents before diving"? And what about all that emphasis on decompression tables and buddy checks?

I have now dived with a number of training agencies and been disappointed by them all. Why is what I find in practice so different from what I had been led to expect?

My third-ever dive was at the narrow entrance to a large sea loch. It was a falling tide, the loch was emptying, and there were standing waves and overfalls below the dive site. The Diving Officer, Training Officer and dive leaders said it would be OK. The current tugged at my legs as I followed my dive leader in and, when we submerged, we immediately started drifting downstream faster than I could swim. I was really exerting myself. We pressed ourselves onto the bottom and I hung onto boulders and kelp in an effort to gain some anchorage, while trying to get my breathing back under control. We tried to move back upstream towards where we had entered, moving a couple of metres at a time by finning furiously and pulling along the bottom, then resting. My demand valve was pulled from my mouth by the battering of the kelp, but I got it back in place and was relieved to see the dive leader indicating that we should work towards the shore.

I emerged, almost exhausted, 12 minutes after entering the water. Four other divers had experienced the same problems, so diving was cancelled.

On my fifth dive I dutifully followed my dive leader, counting down my air supply until, at 20 m, I showed him that I had 50 bar remaining. On we swam. I reached 25 bar, still at 18 m. I was getting worried and my breathing rate was increasing. At 10 bar I made for the surface. We were a long way out from the beach, so we pulled ourselves along an offshore reef until my air ran out.

On my sixth dive I surfaced with less than 20 bar.

On my 10th dive we descended to 30 m and swam parallel to the shore, stirring up thick silt. We had no buddy line or physical contact, so I stayed very close. The dive leader turned round and swam back into the silt, immediately disappearing. I swam after him, but he was gone. It was black, so dark that I could not see my own fins with my torch. I was alone, disoriented and afraid. I shone my torch around and waited. Nothing, just blackness, and a need to control my breathing. I could not tell which way was up. It was horrible. I put a bit of air into my stab (buoyancy compensator) and waited, a bit more, then a bit more, until I felt I was moving. I could not see my bubbles or judge my rate of ascent. When I came into the light I found I was rising too fast. I surfaced and swam ashore.

My 20th dive was at a site where the chart indicated strong currents. The instructor reassured us that it was high tide and would be no problem. On our second dive we were practising assisted lifts in midwater when the instructor unexpectedly indicated that we should surface. We came up to find that we were a long way offshore and moving fast. He shouted that we should swim for a rocky point as fast as we could to avoid being swept out into the bay, where we would drift for miles. I finned as powerfully and steadily as I could. My buddy became tired, so the instructor took her in tow. We just made it, scrambling onto the rock as we struggled for breath.

So much for my diving experiences. I have spent several years on search and rescue helicopters. This is another potentially high-risk business and it has given me a careful approach to preparation and planning, to risk assessment and control.

It has also exposed me to the consequences of diving misfortune. I have dealt with divers bent, swept away and drowned. I have rushed them to hyperbaric chambers, searched hopelessly for them while they drift away down in the dark and delivered them dead to the waiting ambulance.

These experiences have not put me off diving, but have reinforced my safety consciousness. So I ask myself why the incidents described above happened to me. And I conclude that it is largely down to experienced divers paying mere lip-service to basic dive-planning and safety procedures.

I believe that the link between the training program and actual diving practice is broken. Divers do not plan properly, and so lead novices like me into situations in which they are exposed to unacceptable risk.

I perceive an attitude problem. Dive tables are for sissies, after all, we have computers now. Is it unmacho to do a proper buddy check on a dive boat? If you have a buddy, what is the problem about checking his or her equipment and having the favour returned?

Familiarity breeds a casual and sloppy approach, lack of attention to detail, overconfidence and bravado. As a consequence, a moderate risk becomes unacceptably high, because these experienced divers simply do not follow the rules they teach.

So I have learned to trust nobody, except my own, inexperienced buddy. And I take everything I hear from the experienced with a pinch of salt.

As I have seen only too often, completing 2,000 dives does not protect you from getting bent if you ignore the tables, any more than it stops you being swept away if you ignore the tides.

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PANIC STRUCK

Susy Worzencraft

Key Words
Equipment, incidents, panic, rescue.

Here is the tale of a too-fat woman in a too-tight suit, diving after a too-long interval.

Terror engulfed me at 21 m. I remember looking at my computer, overwhelmed by the weight of water above me. I started to gasp, sucking greedily at my regulator as my starved lungs craved air, unable to expand against the squeeze of my suit. One second my brain was telling me to calm down, and the next my thoughts were paralysed by a primitive urge to race upwards to safety. And then guilt set in: “How could you spoil your buddy’s dive?” I thought. What a strange consideration. I was hyperventilating and thought I would die in the grey murk at the bottom of this pit.

I grabbed at my buddy and stuck out my thumb. “Up!”

As I began to ascend, I was convinced that there really was something wrong. Usually, I use that slow languid ascent time for meditation, to watch the depth gauge gradually count backwards, back to sound and light and away from this parallel world. Right now, I dared not look at my

computer. I focused on my buddy, using her as my depth gauge, trying to keep alongside her. As I went up, my breathing gradually eased, and with it my mind. The ascent seemed endless but at least I felt I had regained some control.

Ah! Head above water. Inflate the jacket. Remove the reg. Oh, that first breath! But it was not enough, and again I was struggling to breathe as the neck seal gripped my throat and the jacket compressed my chest.

The shore was sunlit and crowded with brightly coloured stick people and their toy cars. I finned towards them but they were receding. Now I was under water, heading the wrong way. My buddy offered to tow and I was on my back, tank and weight belt pulling my chest even tighter. I was panicking. This was really serious.

Suddenly I am two people. One frantically tearing at the base of my neck seal to relieve the pressure, intent only on this violent need for air. The other one, more detached, sees my buddy signalling for help, shouting again and again for the rescue boat.

"This is not a practice," she yells at the distant and static sightseers. Will they never get going? My other self decides to leave her to deal with my rescue, she is coping much better than I am. I'll concentrate on breathing for the time being.

I let the frenzied me carry on getting air into my starving lungs while eyes and ears work separately, hearing and then seeing the little orange RIB roar towards us.

Dangling against the rubber side, a voice asked me for my weight belt. I was hauled aboard and laid face down like a deadweight, by now feeling mildly foolish.

On the way back my rescuers tried taking my suit off over my head. "We have to cut it off," said a voice after several attempts had failed. Oh, no, not my dear shabby faded old suit, I thought, my companion on so many memorable dives. The body on the floorboards leapt into life at this threat, jumped to her knees and neatly demonstrated the knack of extricating a head from a neck seal. All were astonished at this sudden recovery. Back at the jetty I was offered an arm for support, but refused. Apart from a still-heaving chest and a severe fright, I was back to normal.

We beat our way through the crowds at the top of the steps, who seemed quite disappointed to see the unpretty sight of a purple-faced woman walking unaided, rather than a stretchered body being sombrely carried away. I was tempted to apologise for being alive, but decided to reserve my words for thanking my rescuers.

In retrospect, it had to happen. A fat woman in a too-tight suit, diving after a too-long interval. I'd never

experienced anything like it before. It kept me awake for many nights afterwards surging back and making me gasp over again. I had discovered my mortality.

Why did the rescuers leave my buddy alone in the middle of the lake? She had to make her own way back and without her this tale would have been a tragedy. Fortunately, she helped by one thoughtful woman diver on shore, after all the rest had gone to the steps for a bit of entertainment.

I agree with the need for speed in an emergency, and have nothing but gratitude for the rescue service, but if my buddy had not been so competent and experienced, this could have turned into a very nasty incident.

I went diving soon after and was very nervous. Subsequent dives have almost, but not quite, restored my confidence. I had bought some very expensive equipment, so had to carry on diving but I did not invest in a new, larger suit. Instead of the pork pie and doughnut diet, I am now on bananas and yoghurt.

My buddy is still willing to dive with me. I cannot thank her enough for everything.

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GLEANINGS FROM MEDICAL JOURNALS

PULMONARY BAROTRAUMA

Spirometric indices and the risk of pulmonary barotrauma in submarine escape training
Benton PJ, Francis TJR and Pethybridge RJ.
Undersea Hyper Med 1999; 26 (4): 213-217

Abstract

Between 1975 and 1997, a total of 115,090 ascents, from depths of between 9 and 28 m, have been made by trainees at the submarine escape training tank HMS DOLPHIN. During this 22-year period, 53 incidents have occurred in which, after an ascent, the trainee required hospital or recompression therapy or both.

Scrutiny of the incident records revealed unequivocal evidence of pulmonary barotrauma in six incidents with an additional four in which, despite a negligible gas burden, a confident diagnosis of acute neurologic decompression illness with short latency could be made. No causative mechanism other than arterial gas embolism following pulmonary barotrauma can be implicated in these four cases despite the absence of clinical or radiographic evidence of lung injury.

In all 10 cases the forced vital capacity (FVC) of the trainees was less than the predicted value for their age and height, revealing a statistically significant ($P < 0.01$) association between values of FVC below predicted and pulmonary barotrauma. The median FEV_1 for the 10 cases was also significantly ($P < 0.05$) less than the predicted value after allowing for age and height. No such association was found for the FEV_1:FVC ratio. FVC would thus seem to be the measure of lung function most closely associated with increased risk of pulmonary barotrauma. Possible reasons for this finding are discussed. It is concluded that although the association between low FVC and pulmonary barotrauma is statistically significant, it is insufficiently specific for low FVC to serve as an exclusion criterion for submarine escape training.

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Key Words
Air embolism, decompression illness, emergency ascent, pulmonary barotrauma.