The Editor's Offering

The big news for the September 1998 issue is that the SPUMS home page is up and walking. We cannot really say running, as it is still partly under construction.

But by typing in "http://www.spums.org.au" readers will be able to find the objectives of the society, the current office holders, down loadable application forms for membership and the diving doctors list, the contents of the last Journal, next year's conference arrangements and a list of the SPUMS policies. Still to come is the index for the Journal 1971-1997.

Also to come is being listed by the common search engines. When that has been achieved all one will have to remember of the home page address is "spums" ! As it is the down loadable application form has produced at least one new member.

This issue is accompanied by the Initial Management of Diving Accidents Supplement. The workshop on the Initial Management of Diving Accidents held during the 1997 Annual Scientific Meeting (ASM) at Waitangi in New Zealand produced so many papers that the ideal solution would have been to devote a Journal to them. But editors have to cope with authors and sometimes papers get held up in the processes between speaking and the text being ready for printing. The papers were not all ready by November 1997 when the SPUMS policy on The Initial Management of Diving Accidents was available. It was decided that the policy should be published in the December 1997 Journal but not all the other papers were ready to use. Now, more than a year after the Waitangi meeting, it is possible to produce the supplement. The SPUMS policy has been reprinted so that the Supplement can be used as an independent source of useful information.

The original papers in this issue are very clinically orientated, a study of the effects of hyperbaric exposure on the uptake of intramuscular midazolam, often used to control fits, and a study of the sharpened Romberg test, that struggle to remain upright when standing with both feet in line, arms crossed and eyes closed. This study confirms that there is a learning curve with this test and shows that most subjects learn quickly. Being unable to last more than 30 seconds is strongly correlated with decompression illness (DCI) among New Zealanders who have symptoms suggestive of DCI.

Of interest are Chris Acott's paper on the development of the short oxygen tables and Richard Moon's contribution on adjuvant therapy for decompression illness (DCI). These include oxygen, fluids as first steps and various drugs further down the treatment trail. It appears that fluids are seldom pushed sufficiently, or even given, to overcome the inevitable dehydration of any diver from immersion, let alone that due to the effects of DCI. Very few diving accident victims are going to need intravenous fluids and even fewer are going to be lucky enough to be in a boat with the equipment and a person able to put up a drip. It makes sense to offer frequent drinks, low in glucose, to all conscious divers with DCI who do not have injuries which will require an anaesthetic. Dr Moon provides a list of commonly available fluids and their electrolyte compositions and his recommended, easily made up, tipple with the appropriate electrolyte and carbohydrate concentrations which is mix one part orange or apple juice with two parts water and add 1 teaspoonful of salt to each litre of the mixture.

The papers from the 1997 ASM also include the transcript of the panel discussion attempting to find a consensus on the recompression treatment of DCI. A long and interesting discussion was recorded. About the only conclusion that could be agreed was that recreational divers, who normally present long after symptoms appeared, have such a varied series of signs and symptoms that designing trials to test treatments is fiendishly difficult. Among the problems is the vast range of signs and symptoms make assessing the results of treatment accurately. The New Zealand scoring system, described in the June Journal,[1] is a giant step forward from the very basic, usual system (cured, unchanged and worse) which can lose vast improvements in the very coarse filter.

Mike Davis discusses the theoretical aspects of oxygen therapy equipment, ending with the recommendation that an independent assessment of equipment performance to identify those systems and designs most suited to diving operations would provide a valuable SPUMS diploma thesis.

Bob Halstead contributes a piece, which has appeared in *Dive Log*, about current practices in the diving world. He wonders whether divers ask the right questions when laying down rules for diving practice. We also reprint a piece from *Diver* about the perils of starting a new season's diving. Different environments but similar problems all over the world. And on page 180 we introduce you to two more divers types.

1 Mitchell S, Holley A and Gorman D. A new system for scoring severity and measuring recovery in decompression illness. *SPUMS J* 1998; 28 (3): 85-94.

ORIGINAL PAPERS

HYPERBARIC OXYGEN DOES NOT DELAY THE ABSORPTION OF INTRAMUSCULAR MIDAZOLAM

Gregory Emerson and Peter Hackett

Abstract

AIM: To determine if hyperbaric oxygen at a pressure of 2.8 atmospheres absolute (ATA) (2.8 bar or 18 msw) delays the time to the peak blood level of midazolam given by intramuscular injection.

METHOD: Twenty volunteers were given 0.05 mg/kg of midazolam by intramuscular injection while breathing 100% oxygen at 2.8 bar. Blood was collected every five minutes to determine when the peak blood level occurred. This was then compared with the results when the same group was given the same dose at one atmosphere, breathing air.

RESULTS: In 65% of the subjects, peak blood levels occurred earlier while at 2.8 bar than at 1 bar. In only 15% did peak blood levels occur significantly later at 2.8 bar than at 1 bar. Mean time to peak blood level was 33 minutes at 2.8 bar and 41 minutes at 1 bar.

CONCLUSION: Hyperbaric oxygen at 2.8 bar does not delay the absorption of intramuscular midazolam.

Key words

Drugs, hyperbaric oxygen, treatment.

Introduction

Medications are not given by intramuscular (IM) injection in hyperbaric chambers because it is presumed that vasoconstriction secondary to hyperoxia would delay absorption. Previous studies have confirmed that vasoconstriction does occur under hyperbaric conditions. The effect of this on drug absorption from IM sites is theoretical and has not been tested.

This study was to determine if hyperbaric oxygen at 2.8 atmospheres absolute (ATA or bar) delays the absorption of IM midazolam.

This has clinical applications as IM midazolam is now frequently the first line pharmacological treatment for convulsions. Convulsions in the hyperbaric environment are not infrequent and can be difficult to manage.

Method

Approval was gained from the Fremantle Hospital Ethics Committee. Informed consent was obtained from the 20 volunteers and all were given written information on the aims of the study and potential side effects. The volunteers had to be fit for hyperbaric exposure and over the age of 18. Contraindications to participation included intercurrent illness, not being within 25% of ideal body weight, on other medications and pregnancy.

A 16 gauge intravenous (IV) cannula was placed in an antecubital vein and a baseline 3 ml blood sample taken. Blood was collected in 4 ml lithium heparin plastic tubes. The subject was then compressed to 2.8 bar and 100% oxygen was commenced using a head hood. One minute after commencing oxygen, 0.05 mg/kg midazolam was given by IM injection into the lateral aspect of the right thigh. This dose was chosen to ensure quantifiable serum levels while avoiding deep or prolonged sedation in the subjects. A 5 mg/ml preparation of midazolam was used. Doses ranged between 3 and 5 mg. Further sample collection began ten minutes after the injection and continued every 5 minutes for 60 minutes. After each blood sample was drawn a 3 ml saline flush was given to avoid cannula occlusion. Immediately before taking the next sample 3 ml of blood was withdrawn from the cannula to avoid saline dilution.

The volunteer and an attendant were compressed to 2.8 bar (18 m) for 60 minutes, with 5 minute air breaks after 25 and 55 minutes. Decompression followed the 1992 DCIEM "In Water Oxygen Decompression" tables. Ascent from 18 m to 9 m was over 15 minutes, followed by 5

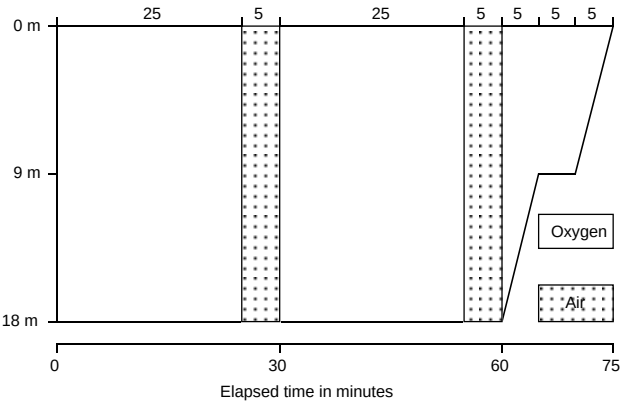

Figure 1. The dive profile used. The numbers across the top are minutes for each segment of the profile. Dotted areas are air breaks. The participants breathed oxygen except when having air breaks. The inside attendant breathed air except during the ascent from 9 m.

minutes at 9 m and then ascent to the surface over a further 15 minutes (Figure 1). The attendant breathed oxygen from 9 m to the surface.

After at least three days the procedure was repeated on the same volunteers who acted as their own control group. This three day break was used to avoid residual midazolam contaminating the base line sample. Days were chosen where the volunteers had experienced a similar level of physical activity to the original sampling. This should have avoided any significant alteration in drug absorption due to variations in baseline muscle blood flow. At no stage was sampling done on volunteers after heavy exercise. Sampling was identical for the control group except that the subjects were kept at 1 bar and were breathing air. All samples were taken by the author.

On completion of each sampling period the subjects were observed for 1 hour and then allowed home under supervision. All were advised not to drive or operate heavy machinery within 6 hours of the injection.

Samples were analysed by high performance liquid chromatography after extraction from alkaline solution into diisopropylether which was taken to dryness. Quantification was effected by comparison with standard additions of midazolam and internal standard blank serum with final analysis by absorption at a wavelength of 254 nm. The correlation coefficient over the range 10 to 100 µg/l was 0.999, with coefficient of variation at 10 and 100 µg/l being 8.4% and 3.3% respectively.

Statistical analysis was performed using the paired T test. Univariate analysis was undertaken to describe the results in each of the two testing periods.

Results

Twenty volunteers were studied. There were 13 men and 7 women. Mean age was 31 years (range 22-46). All volunteers had previous exposure to either diving or hyperbaric chambers. The results are displayed in Table 1.

In 65% (13/20) of the subjects, peak blood levels occurred earlier at 2.8 bar than at 1 bar. In 25% (5/20), peak blood levels were later at 2.8 ATA than at 1 ATA. In 10% (2/20) peak blood levels occurred at the same time at 2.8 and 1 bar. The mean time to peak blood level at 2.8 bar was 33 minutes (95% CI: 28-38). Mean time to peak blood level at 1 bar was 41 minutes (95% CI 35-47). This difference in mean peak times is not statistically significant ($t = -2.0$, $df = 19$, $p=0.060$). In all but 4 of the subjects, an earlier peak time corresponded to a higher peak level.

If the assumption is made that a difference of up to five minutes in the time of achieving peak blood level at the two pressures is not clinically relevant, then 65% (13/20) had their peak earlier at 2.8 bar, 20% (4/20)peaked at the same time in both pressures and only 15% (3/20) peaked later at 2.8 bar than at 1 bar.

TABLE 1

Time to peak level data

Subject	**2.8 bar**		**1 bar**	
	Time minutes	Level µg/ml	Time minutes	Level µg/ml
1	30	32	40	51
2	35	31	55	18
3	35	43	45	41
4	35	54	50	47
5	20	51	30	19
6	30	54	45	38
7	45	47	55	39
8	25	44	55	56
9	10	38	45	26
10	35	59	50	35
11	15	70	60	29
12	35	82	50	60
13	30	58	45	34
14	35	50	35	26
15	30	50	30	52
16	35	18	15	28
17	35	51	30	36
18	35	52	15	73
19	35	34	25	51
20	55	36	50	23

Time to peak blood level of greater than or equal to 45 minutes has previously been used to define delayed absorption.[1] In this study 90% of the 2.8 bar group had peaked by 45 minutes as compared to 60% in the 1 bar group.

No side effects occurred in any of the subjects.

Discussion

Midazolam, 8-chloro-6-(2-fluorophenyl)-1-methyl-4H-imidazo benzodiazepine, is used for premedication, sedation and anaesthetic induction. It is also becoming increasingly used as an anticonvulsant after it was shown to be effective in status epilepticus.[1,2] It has a number of advantages over diazepam because of its unique physiochemical properties. At a pH of 4, the diazepine ring of midazolam opens producing a highly water soluble compound. Consequently it is available without the need for organic solvents such as propylene glycol which can cause venous irritation and cardiac arrhythmias. At a pH of greater than 4, the ring closes, resulting in increased lipophilicity and, consequently, its fast onset of action.[3] Unlike diazepam it is rapidly absorbed following IM

injection with a bioavailability of 90%.[4] Onset of sedation is rapid and previous studies have shown mean peak blood levels at 20-25 minutes but with considerable individual variation.[4,5] Central nervous system effects of midazolam follow the blood levels closely.[6] It is metabolised by the cytochrome P450 system to several metabolites including the active alpha-hydroxymidazolam. The elimination half-life of 1.5-3 hours is short compared with more than 20 hours for diazepam.[7] Alpha hydroxymidazolam has an elimination half-life of 1 hour.[8]

Hyperbaric medicine staff should be familiar with the use of midazolam because there are multiple causes of potential convulsions in the hyperbaric chamber. Common causes include cerebral oxygen toxicity, pre-existing epilepsy and cerebral irritation from decompression illness or carbon monoxide poisoning. Convulsions from cerebral oxygen toxicity are generally self limiting once the oxygen is ceased. Convulsions secondary to the other causes, however, can be prolonged requiring treatment along standard emergency guidelines.

Intravenous midazolam or diazepam are the first line pharmacological agents in the treatment of convulsions. However not all patients in a hyperbaric chamber will have an intravenous cannula in place. Midazolam given IM is now the preferred first line treatment for convulsions in many Australian emergency departments. Its use enables the insertion of an intravenous line for ongoing treatment under easier conditions. Intravenous lines are difficult to insert in a convulsing patient and there will also be a time delay if a doctor is required to lock into the chamber to place the line. Intramuscular midazolam has been shown to be effective in stopping prolonged seizures in children and adults. In the study by McDonagh et al. only 5% failed to respond.[2] Seizures were terminated in 1 minute 53 seconds on average (range 15 seconds to 6 minutes 7 seconds). Epileptiform activity has also been shown to either disappear or be significantly reduced after IM administration.[9] Midazolam is more effective than IM diazepam and as effective as IV diazepam 5 minutes after injection.[10,11] Intramuscular diazepam is absorbed both slowly and erratically and the peak blood concentration does not occur until 60 minutes.[12] Rectal absorption of midazolam has been shown to be poor and irregular.[13]

Medications are not usually given by the intramuscular route in hyperbaric chambers because hyperoxia results in vasoconstriction. This has been shown in skeletal muscle,[14] the brain,[15] retina,[16] and abdominal organs.[17] The vasoconstriction has been presumed to delay the absorption of the intramuscular medication. Potential problems of delay in absorption include failure of the drug to reach a therapeutic concentration and also the release of a bolus of drug once hyperbaric exposure ends and vasoconstriction ceases. Although vasoconstriction has definitely been shown to occur, the effects of this on drug absorption from the intramuscular site have never been reported. This study suggests that absorption of intramuscular midazolam is significantly delayed in only 15% at 2.8 bar compared to 1 bar and that the mean time to peak blood level is shorter at 2.8 bar. Although statistical significance has not been reached due to a lack of power (0.15), the hypothesis that hyperbaric oxygen delays the absorption of intramuscular midazolam is not supported.

Intramuscular midazolam could therefore be a safe and effective treatment for patients with prolonged convulsions in hyperbaric chambers. Because of the unique pharmacology of midazolam this recommendation cannot be extended to all medications under pressure. However, the situation of a prolonged convulsion in a patient under pressure would be the main circumstance where it would be useful to be able to give IM medications. In the small percentage of patients whose convulsion does not respond to IM midazolam after 3 minutes then IV access can be obtained and treatment with IV midazolam instituted as would occur under normal emergency medicine practice.

One of the limitations of the study is that, although vasoconstriction occurs, no studies have addressed when it begins. Measurements in most of the studies begin at 15 minutes and have confirmed vasoconstriction at that stage. Theoretically a considerable amount of midazolam may have been absorbed before vasoconstriction occurred. However, vasoconstriction is a reflex precipitated by hyperoxia and would be expected to occur immediately the tissues become hyperoxic. Additionally, one study has found that in patients with traumatic cerebral oedema treated with hyperbaric oxygen, intracranial pressure (ICP) decreased as soon as treatment pressure was reached.[18] Reduction in ICP is attributed to vasoconstriction of cerebral arteries.

The dose of midazolam used was subtherapeutic but this should not have affected the time to peak blood level. Increasing the dose increases the peak blood level but the time to the peak is determined by the volume of distribution and the rate of drug absorption from muscle. Volume of distribution of midazolam is neither age nor sex related but obesity increases it.[19] In this study all subjects were within 25% of their ideal body weight. The rate of drug absorption from muscle is determined by blood flow to the site and the physiochemical properties of the drug. Variation in blood flow to the site was minimised by controlling for physical activity prior to testing, testing at the same time of day and using the same site for injection.

Conclusion

Hyperbaric oxygen at 2.8 ATA does not delay the absorption of IM midazolam. This suggests that IM midazolam may be a safe and effective treatment for prolonged convulsions in patients in hyperbaric chambers who do not have intravenous access already established. There is a need for further studies in a clinical setting.

Acknowledgment

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The above paper formed the thesis submitted for the Diploma of Diving and Hyperbaric Medicine, which was awarded to Dr Emerson in July 1998.

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SHARPENING THE SHARPENED ROMBERG

C-T Lee

Key Words

Decompression illness, investigations, treatment.

Abstract

The Sharpened Romberg Test (SRT) is a test of balance commonly used in Diving Medicine. Interpretation of an abnormal test can be confounded by

several factors. This study was conducted to further evaluate the usefulness of the SRT.

In the first part of the study, naval and civilian volunteers in a Naval Base were recruited as subjects. The SRT scores were recorded in two separate trials; once in the morning (4 attempts) and once in the evening (4 attempts) to evaluate the effect of practice on the SRT.

In the second part of the study immediate pre- and post-dive scores in a group of divers were measured to evaluate: (1) the effect of decompression; (2) the effect of the normal post-dive fatigue'; and (3) the vestibular effect of swaying after a boat ride. Comparisons were also made between the distributions of the SRTs of the normal subjects and those of a retrospective group of DCI patients treated at the Slark Hyperbaric Unit, Royal New Zealand Navy Hospital (RNZNH), Auckland.

The SRT was found to have an early learning effect. Second attempts were significantly better than the first ($p<0.001$) within the same trial. However this learning effect plateaued by the third and fourth attempts. No difference was found between trials (morning and evening).

There was a post-dive decline in the scores of the first attempts only ($p<0.05$). Subsequent second to fourth attempts were not affected by diving. The practice effect is only evident between the first and second attempts within the same trial but not between trials. The pre- and post-dive data showed that the SRT was not affected by decompression, post-dive fatigue or the vestibular sensation of swaying that is commonly experienced after a boat ride.

Comparison of the distributions between controls and DCI patients showed a bimodal pattern. Fifty-four percent (54%) of the DCI patients had 'normal' scores (60 seconds), while 14% had scores between 16-35 seconds and 32% scored less than 15 seconds. In contrast, 95% of the control groups had 'normal' scores while 5% scored between 16-35 seconds. Therefore, accepting a score of less than 40 seconds as being "abnormal" will give the SRT a sensitivity of 46%, specificity of 95% and predictive value of 82%.

Introduction

Decompression Illness (DCI) is a multi-system pathological entity with a myriad of presentations.[1,2] Initially DCI was first described in caisson workers and then in divers, aviators and astronauts. Limb pain was the predominant symptom in these groups of patients.[3-7] Over the past three decades published reports of DCI have mainly been from the recreational diving population.[8-13] This is due to the increasing popularity of the sport worldwide. Neurological involvement, especially those referring to the spinal cord and vestibular system, appears to be more common in this group of divers.[2,14-16] Animal studies have shown that, in the spinal cord, bubbles and haemorrhage were seen predominantly in white matter and tended to be most conspicuous in the lateral and dorsal columns.[17,18]

Manifestations of neurological DCI range from mild, subjective symptoms to the dramatic presentations of unconsciousness, paraplegia or quadriplegia. In practice, divers commonly present with subjective complaints, often with little or no objective evidence of neurological abnormalities.[9] It appears that the clinical neurological examination lacks the accuracy to detect the diffuse and multilevel pathology seen in decompression illness. Therefore, the diagnosis of DCI requires a high index of suspicion, and a history of recent diving or exposure to raised environmental pressure.[19]

The usefulness of the Sharpened Romberg Test (SRT) as a clinical marker of DCI was recently highlighted, especially in cases where the disease process was in question.[20,21] Almost 49% of the 35 cases with DCI in that series were found to have grossly abnormal SRT scores with seventy percent (70%) of these achieving a 'normal' score after completion of hyperbaric treatments. Therefore, in this series at least, the SRT score was useful as a quantifiable sign in 50% of the cases.

The Sharpened Romberg Test

The classical Romberg Test as described by Moritz Romberg (1795-1873) is routinely used in neurology to assess proprioceptive loss. It is, however, not sensitive to vestibular or cerebellar impairment.[22,23] Barbey described the first modification of this test in 1944[24] and Fregly, in the late 1960s, employed this "sharpened" Romberg Test (SRT) together with his ataxia test battery as measurements of vestibular impairment at the US Naval Aerospace Medical Institute.[25,26] Also known as "Tandem Romberg"[27] or "Modified Romberg",[28] the SRT has also been employed in several ataxia test batteries in gerontology and toxicology.[29-32] Dr Carl Edmonds introduced its use to Australian diving medicine in 1974 as an alternative to the classical Romberg Test, as it is more sensitive to proprioceptive and vestibular impairment. Since then the SRT has found wide acceptance in the routine assessment of diving patients.[33-35]

Variations in the SRT

The Sharpened Romberg Test, as originally described by Fregly, involved the subject "standing on the floor with eyes closed and with arms folded against chest, feet aligned in strict tandem heel-to-toe position, and body very nearly, if not completely, erect for a period of 60 seconds. A maximum of four trials were administered."[23,36]

Several variants of the SRT have since been described. Some involved the subject having to stand in the usual tandem heel-to-toe fashion but with arms strictly by the side.[28,29,37] Others allowed the subject in this position to freely move his arms in order to regain posture.[30,31] The SRT has also been performed with subjects standing on narrow wooden rails in order to lower the 'ceiling effect'.[38] One investigator proposed that the SRT should be performed with the head tilted.[33]

Factors affecting the SRT

Although the SRT is a sensitive test of proprioception, its specificity in DCI is not clearly defined. Being a test of static postural equilibrium, the SRT is affected by several factors other than dorsal column or vestibular diseases.

AGE AND GENDER

Studies have confirmed that the SRT performance worsens with advancing age. [24-26,29,31] Decline in performance generally begins between the age of 30-40 years in males and as early as 30 years in females.[25,39] The reasons for these gender differences are unknown.[23] The number of females tested was generally small[25] and in selected groups[29] and therefore the finding should be interpreted with caution, especially as one study failed to demonstrate a difference.[28]

LEARNING EFFECTS

Like many tests of performance, SRT scores can improve with subsequent attempts due to a learning or practice effect. Thomley et al. had 18 subjects practise on the SRT twice a day for five consecutive days.[40] Both learning and ceiling effects were reported but the tests were stable over trials. Other studies have shown similar results.[28,25] Briggs et al. found that the majority of their subjects obtained the maximum balance times (60 seconds) in the first trial.[29] A minimum of three trials appeared to provide a good indicator of balance capabilities. The most consistent and sensitive means of measuring the SRT is to record the best score out of 4 attempts.[20,30,31]

FOOTWEAR

No difference was found between wearing shoes or being barefooted.[29] However, shoes with soft soles (such as tennis/basketball shoes) are generally not to be worn because soft surface conditions (which would include foam mats on the floor or thick carpets) distort proprioceptive input and hence would not suitable.[23,26,37]

DOMINANCE

Some investigators required the subjects to perform the SRT with the dominant leg behind. However, in one study no effect of dominance was found.[29]

ACTIVITY LEVEL

In a study that employed self-reported questionnaires, a significant effect was found between activity level and balance performance (including the SRT).[31]

The SRT in diving medicine

Maintenance of postural equilibrium is a dynamic process in which visual, vestibular and somatosensory (proprioceptive, cutaneous and joint) information are integrated with muscular and skeletal responses to maintain the body's position over the base support. The Romberg test assesses the vestibular and somatosensory contribution to balance by eliminating the visual input. The Sharpened Romberg Test (SRT), by having the subject stand heel-to-toe, makes further demands on the vestibular and somatosensory systems by narrowing the base support. It is generally more difficult to perform and is therefore more sensitive to processes that interfere with these systems.

In the context of diving medicine, the SRT appears to be a useful quantifiable sign. In the study by Fitzgerald, the substantial improvements (70%) in the SRT post-treatment scores indicate that DCI causes a deterioration in the SRT.[20] However, other factors which affect the balance system could also contribute to this deterioration of the SRT score. These are summarised below.

a Divers conducting their dives from a boat out in open sea frequently experience persistent vestibular symptoms, described as a sensation of swaying motion, on returning to land. This might adversely affect the SRT performance of a diver presenting for assessment.

b Improvements in the SRT score seen in divers being assessed in sequence (pre-, during and post-treatment) could be due to a learning effect rather than an indication of the actual resolution of the disease being treated.

c Decompression per se (which is known to produce asymptomatic bubbles) or feelings of fatigue after diving could, in theory, affect the SRT.

d Improvements in the SRT score during and after recompression treatment could be due to an effect of hyperoxia rather than a resolution of disease.

e Alcohol consumption is common during most dive trips, and could confound the SRT score.

The aim of this study was to further define the usefulness of the SRT in diving medicine by testing the following hypotheses :

1 The SRT is resistant to the effect on the vestibular system caused by rocking motion of a boat.

2 Scuba diving and decompression per se has no effect on the SRT

3 The recommended protocol used for scoring the SRT is not affected by practice

4 The normal feeling of fatigue post-dive does not affect the SRT score.

Attempts were also made to determine the SRT score or test method which could distinguish between the normal (non-DCI) and the DCI patients.

Methods

This study was conducted in three parts. The first part involved the prospective review of SRT scores in a group of volunteers from Naval and civilian personnel at the Naval Base in Auckland. This group consisted of both divers and non-divers. The second part involved the pre- and post-dive evaluation of the SRT scores in a group of divers. Finally, the SRT scores of the patients with DCI treated at the Slark Hyperbaric Unit (SHU), Auckland, between May 1996 to April 1997 were reviewed.

In part one of the study, the subjects were "captive volunteers" actively recruited by the author. Each subject received an explanatory letter and gave written consent for participation. Divers were entered into the study only if they had not dived for the past seven days and had no history of decompression illness. Exclusion criteria were the same as those in the study by Fitzgerald.[20] A subgroup of 47 participated in 2 separate tests: once in the morning (4 attempts) and once in the evening (4 attempts).

The second part of the study was conducted at the dive site. Divers attending a conference were briefed during registration and participation forms distributed. Baseline SRT scores for divers going for their dives were measured before the commencement of the diving activities. The post-dive SRT scores were recorded for the same individuals within 24 hours after their day of diving. All dives involved a boat ride to the dive location in open sea for the day. Sea conditions were mild to moderate for those dives. Participants were instructed not to consume alcohol for at least 12 hours prior to the tests.

Comparisons were also made between the scores of the control population and a retrospective group of DCI patients treated at SHU between May 1996 and April 1997.

The Sharpened Romberg Test in this study was done with subjects barefoot or wearing flat shoes standing on a flat surface. They stood heel-to-toe with their arms folded across the chest and eyes closed. The test procedure was similar to that proposed by Fregly[29] except that the best score of the 4 attempts was used. Timings were stopped once the subjects lost balance, opened their eyes, moved their feet to regain posture or when the required 60 seconds was attained. The test was discontinued when the score of 60 seconds was obtained on any one attempt. If the subject scored less than 60 seconds, the number of seconds attained was recorded and further attempts made until a score of 60 was attained or up to a maximum of four attempts had been made. Attempts scoring less than 5 seconds were considered as false starts and not recorded.

The data collected were entered into Microsoft Excel version 5.0 and analysed using SPSS for Windows. Distribution scores for balance tests are generally skewed. Statistical tests of significance for age were performed using T-Test while those for SRT scores were analysed using Mann-Whitney U Test and Wilcoxon Signed Rank Test for independent and paired samples respectively. An alpha level of 0.05 was set as the criterion for all tests of statistical significance.

Results

Sharpened Romberg Test data were obtained from 102 subjects. One subject with a history of lower limb pathology was excluded from the study. Forty eight of the subjects were divers with no known history of DCI and 53 were non-divers. Forty-seven subjects had two separate measurements of their SRT trials.

Table 1 summarises the age distribution of the study population. Divers in the under 40 age group were generally older than the non-divers. The age distributions of those in the 40 and over group were the same. A comparison of the SRT scores between the divers and the non-divers showed no significant difference (Table 2). This is despite the divers in the over 40 group having an older mean age .

Each subject was allowed 4 attempts per trial to attain a score of 60 seconds. From the study sample of 101 subjects it was found that 71% attained the required 60 seconds at the first attempt, 89% by the second, 93% by the third and 95% by the fourth attempts (Figure 1). A

TABLE 1

AGE DISTRIBUTION OF 101 CONTROL SUBJECTS

Age group	Subjects	Number	Mean Age + SD	t-test
< 40 yrs	Divers	29	30.34 + 7.44	$p < 0.05$
	Non-divers	40	22.45 + 6.68	
≥ 40 yrs	Divers	19	48.26 + 7.76	Not significant
	Non-divers	13	47.85 + 6.65	

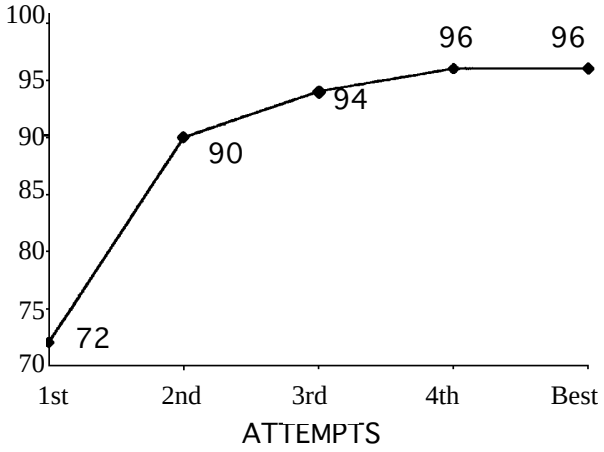

Figure 1. The number of controls scoring the maximum (60 seconds) in each of the four attempts during the trial.

TABLE 2

SIGNIFICANCE OF SHARPENED ROMBERG TEST SCORE DIFFERENCES

Ages	**Subjects**	**Attempts**	
		First	**Best**
< 40 yrs	Divers	*Not	*Not
	Non-divers	significant	significant
≥ 40 yrs	Divers	*Not	*Not
	Non-divers	significant	significant

*Mann-Whitney U Test

significant difference ($p < 0.001$; Wilcoxon signed ranked sum test) was found between the scores of the first and second attempts. Comparison of the scores between the second, third and fourth attempts showed no significant differences ($p>0.05$).

Figure 2 shows the subgroup (N=47) who had their SRT scores recorded on two separate occasions. No significant difference was found when scores of Trial A (first) and B (second) were compared ($p>0.05$; Wilcoxon Signed Rank Test).

Among the group of divers who had their pre- and post-dive SRT scores measured, the data (Figure 3) showed a post-dive decline in the scores of the first attempts ($p<0.05$). The subsequent second, third and fourth attempts were not affected by diving.

A total of 66 cases of DCI were treated at the Slark Hyperbaric Unit, Auckland in the period between May 1996 to April 1997. Case records were available for 55 patients.

Of the 55, five had no SRT scores recorded and these were not included in the study. Figure 4 compares the

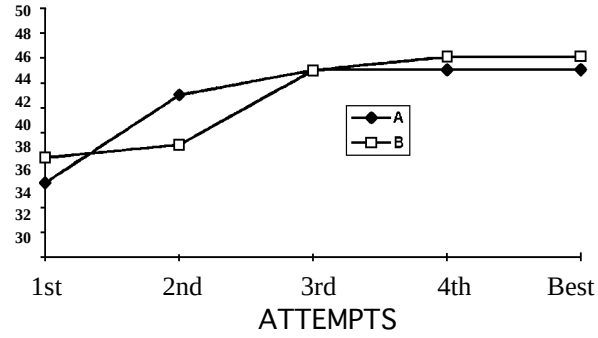

Figure 2. SRT scores of 47 controls tested twice. A denotes the first trial and B the second.

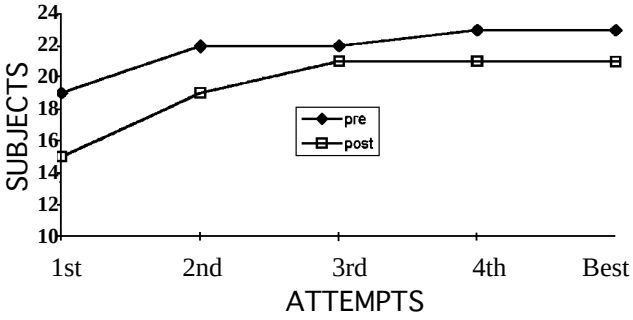

Figure 3. Comparison of the pre-dive and post-dive SRT scores. The number of divers achieving the maximum score (60 seconds) is indicated.

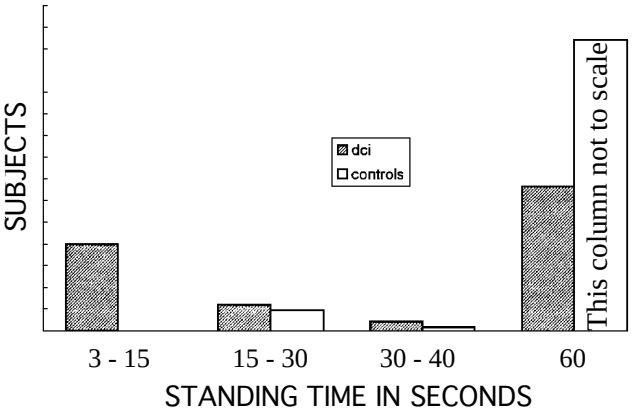

Figure 4. Distribution of SRT scores among patients with DCI and controls

difference in distribution of the SRT scores between the control subjects and those with decompression illness. The performance in the Sharpened Romberg Test in all non-DCI subjects studied (n=101) showed a bimodal distribution with a large majority (95%) achieving a score of 60 seconds and 5% scoring between 16-35 seconds (Figure 4). The patients with DCI also showed a bimodal pattern, with 54% obtaining a score of 60 seconds. The 23 patients who had abnormal SRT scores did poorly with 16 (70%) scoring

TABLE 3

SRT RESULTS IN 23 PATIENTS WITH DCI PRESENTING WITH ABNORMAL SRT

Number of patients	SRT scores	
	Admission	Discharge
9	<= 5 seconds	60 seconds
4	6-10 seconds	60 seconds
3	11-15 seconds	60 seconds
4	16-25 seconds	60 seconds
1	26-30 seconds	60 seconds
2	31-35 seconds	60 seconds

SRT scoring was the best of 4 trials or until 60 seconds were achieved.

less than 15 seconds. The scores of all the patients with DCI who had abnormal scores were less than (or equal to) 35 seconds.

Table 3 shows the SRT scores on admission and on completion of treatment. All patients in this series with abnormal SRT scores on admission had 'normal' scores (60 seconds) upon discharge.

Discussion

The Sharpened Romberg Test is commonly used in the assessment of divers with decompression illness (DCI). In DCI the balance system is involved in a large proportion of patients. Therefore, if found to be abnormal, the SRT is useful as a clinical sign to monitor the progress of the disease during treatment, especially when the patient has only subjective symptoms. However, interpretation of an abnormal SRT score in a diver requires that the attending clinician be aware of other factors which could or could not affect the SRT.

Balance tests are known to improve with practice,[25,29,40] just like any other tests in which skills are involved. In our study population (N=101), the learning effect was evident only between the first and the second attempts within the trial. The subgroup (N=47) which had two separate trials assessed showed no significant difference in their SRT scores. The SRT protocol used appears to provide a good indicator of balance capabilities. Repeat administration of the test showed no learning effect and therefore will not bias the sequential assessment of a patient being treated for DCI.

The pre- and post-dive data (N=25) provided answers to three questions. First, decompression per se causes no deterioration in the SRT score. Therefore, the SRT is probably not a useful or sensitive indicator of decompression stress, be it asymptomatic venous bubbles or subclinical DCI. Second, the feeling of tiredness that divers often experience after diving had no effect on the SRT scores in our study population. Therefore the tiredness that accompanies scuba diving (after 2 dives a day in this context) and the fatigue commonly reported by divers with DCI appear to be pathophysiologically different. Third, the residual vestibular effect (sensation of swaying) after a boat ride in open sea does not cause a significant deterioration in the SRT. However, exposure to severe storm conditions at sea is known to produce a deterioration in balance performance.[41] Only the first post-dive attempts in the sharpened Romberg test were adversely affected (Figure 3). Performances in the subsequent attempts were unchanged from the pre-dive scores.

The distribution of the SRT scores showed a bimodal distribution in both non-DCI controls as well as in those with DCI (Figure 4). However, the majority of patients with abnormal SRT generally had very low scores, with 70% (16/23) scoring less than 15 seconds. There is a considerable overlap in those scoring between 16 to 35 seconds (7 in the DCI group and 5 in controls). It is noteworthy that none had scores between 36-59 seconds. All the subjects who scored more than 36 seconds initially managed to obtain the criterion score of 60 seconds within the allotted 4 attempts. 95% of the normal controls attained the required score, with 5% false positive rate.

TABLE 4

VALIDITY OF THE SRT IN DCI

	DCI	Controls	Total
Abnormal SRT*	23 (21)	5 (4)	28 (25)
Normal SRT*	27 (29)	96 (97)	123 (126)
Total	50	101	151

*Accepting a cut-off score of 40 seconds (in parenthesis) rather than 30 seconds will improve the sensitivity of the test. See text for details.

The 2 x 2 contingency table in Table 4 attempts to define the validity of the SRT. Accepting a SRT score of <=40 seconds as being abnormal would have a sensitivity of 46%, specificity of 95% and a predictive value of 82%. If a score of <= 30 seconds is taken as abnormal, the sensitivity of the test would be reduced to 42% with little change in specificity (96%).

The SRT is resistant to the influence of the factors that were studied , namely practice effect, decompression stress (including post-dive fatigue or tiredness) and vestibular disturbance after a boat ride in mild to moderate sea conditions. Deterioration in SRT scores due to DCI was characteristically in the 16 seconds or less group. If the

cut-off score is increased to 40 seconds the sensitivity will be increased to 46% and specificity 95% (Table 4). It is proposed that the scores of all the attempts should be noted down although only the best result is taken as the SRT score. This is to facilitate future research in this area.

The number of patients used in this study is small and therefore extrapolation of the results to diving medicine in general should be made with caution. For practical reasons the SRT procedure used in this study imposed a limit of 60 seconds as the maximum score. Except for those who scored less than 60 seconds, the true SRT scores for those who attained the 60 seconds were probably much higher. This ceiling effect limits the ability of the SRT to detect small decrements in performance score.

Alcohol is another factor which may interfere with the SRT assessment of diving patients. Fregly and Graybiel found postural equilibrium to be highly sensitive to moderate doses of alcohol (2.2 cc 100-proof vodka per kg body weight).[42] Hyperoxia per se, instead of disease resolution, could be another possibe cause of the improvement seen in SRT scores of the patient treated in the chamber. Further studies should be conducted to evaluate the effect of hyperoxia and lower doses of alcohol on the SRT performance in normal subjects.

In summary, the Sharpened Romberg Test is a useful marker of Decompression Illness. The results of this study show that it is resistant to several potentially confounding factors which are often present during the assessment of a diver with DCI, namely, post-dive fatigue, decompression stress, vestibular disturbance resulting from exposure to swaying motion of dive boat and improvements due to practice or learning effect.

Acknowledgments

The author gratefully acknowledge the invaluable advice and guidance of Professor Des Gorman. Special thanks are also due to Drs Simon Mitchell and Chris Strack for their help with the study. The author also wishes to thank the staff at the RNZN Hospital, and especially those of the Slark Hyperbaric Unit, for their enthusiastic assistance.

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This paper formed the thesis submitted for the Diploma of Diving and Hyperbaric Medicine awarded to Dr Lee in 1998. The study on which this paper is based was carried out when Dr Lee was on a clinical attachment at the Slark Hyperbaric Unit, RNZNH, Auckland.

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THE WORLD AS IT IS

AUSSIE RULES: A PERSONAL OPINION

Douglas Walker

Key Words
General interest, medical conditions and problems, underwater medicine.

The medical profession is no more exempt from fashions than any other human activity, though some of its beliefs, based on ancient texts whose truths were accepted as gospel, persisted unquestioned for centuries. In more recent times fashions in diagnosis and treatment have come and gone more rapidly but have been as unquestioned for a time, as is any dogma, until successfully challenged. In the 16th century Humours defined a person's state of health. In this century Vitamins, (bowel) Toxins, Stress, Viruses or Free Radicles have each in turn been credited with being THE true cause of disease. Indeed, Nature and Nurture are still fighting for supremacy as being critical in defining an individual, with the new discoveries about genes providing equivocal support to one or other side in turn. Diving medicine has not escaped the curse of Accepted Truths, though this may not be immediately apparent to everyone. And now we have the era of Evidence Based Medicine, which rather unjustly assumes that everyone has, until now, formed their opinions out of thin air, or little better. So how does our (sub) speciality rank in this era of questioning ?

In an unusual example of admitting medical uncertainty, the grand division between a diagnosis of Air Embolism or of Decompression Sickness, which was first declared in the 1930s, has been modified, even if not formally abandoned. It is now considered correct to use the diagnostic label "Decompression Illness" for most cases. The reasoning is that the differential diagnosis may be difficult because the two conditions may co-exist as cause of the symptoms, and the basic treatment is the same for both. An additional reason may be that our understanding of the pathological changes in this syndrome is now accepted as being too simplistic and unable to explain, among other things, the response to delayed recompression. In fact there may be a mix of three significant factors, air emboli, decompression produced gas emboli, and tissue bubbles. But is this reason enough to "change the label" to hide our uncertainty or should it be a spur to further research?

The main problem area, in which there are unresolved differences, in basic diving medicine opinions is in defining minimal medical standards for acceptance for diver training. This is to be expected, because there cannot be an absolute standard to cover every field of human activity. Indeed if there should be such a standard developed it is highly unlikely than any single person would satisfy it! We must live with compromise, accepting that the factors of determination and skill shown by some with "disabilities" will prevent them from being labelled "disabled" from activities they wish to pursue. We need to be careful in use of the label "disability" as this may be true only in the context of the degree in which it is present rather than being an absolute. Unfortunately, Diving Medicine has claimed to be able to define the border between the Medically Fit to Dive and the Unfit to Dive. Unfortunately, because the standards are significantly different in different countries and these differences have not been reconciled.

The power which indoctrination wields over decision makers has been well illustrated during past "Workshop" discussions on the importance of a history of asthma in relation to diver safety. The absence of neutral research into this subject is deplorable, and made the more so because it is generally accepted that there are many asthmatic divers in the real world. One problem is that there is so much logic in banning such persons from diving, using compressed gases, that there has been (and continues to be) a reluctance to consider morbidity and mortality data which could be non-supportive of the belief. The problem has not been made any easier by the past claims by the medical profession to be able to define the cut-off point by a medical examination (both the medical history and physical examination) and the pleasure this has given the diving instructor organisations, their insurers, and the legal profession. These non-medical bodies are only too pleased to allow others (doctors) to assume the responsibility for drawing lines in the sand, possible an appropriate description of basing rigid opinions on an insecure base.

Another matter where diving medicine expertise has intruded has been on whether an out of air ascent should be included as an essential element in primary training. Strongly held views have bedevilled attempts to hold a rational discussion of this problem. Here also reference to morbidity and mortality reports, and the collection and examination of data from incidents where an out-of-air situation occurred should be the basis of any discussion. Consideration should be given to whether the protocols of this "training" can reasonably be considered to actually *train*, as contrasted with allowing the person to experience a controlled and supervised trial ascent.

As long as the diving organisations continue to use the term "Advanced Diver" for those who take a second course immediately after their initial course, there will be doubts about their understanding of the critical factors to diver non-survival, the most significant of which is inexperience. This reflects on the validity of present training protocols. There is no justification, however, for the diving medicine community continuing to drag its feet

in the matter of reviewing the advice it gives on safety matters wherein it should have competence.

Samuel Johnson, the great lexicographer, reportedly noted in 1734 that "it is incident to Physicians, I am afraid, beyond all other men, to mistake subsequence for consequence". In conclusion, let me propose, with due acknowledgment to the advertising agency for the Aussie Rules organisation, a remedy to this criticism he so succinctly encapsulated, that we adopt the advice of their advertising and say:

"Evidence based Diving Medicine? I'd like to see that !"

Dr D G Walker is a foundation member of SPUMS. He has been gathering statistics about diving accidents and deaths since the early 1970s. He is the author of the series of Provisional Reports on Australian Diving-related Deaths which have been published in the Journal covering 1972 to 1992. His address is P.O. Box 120, Narrabeen, N.S.W 2101, Australia. Fax + 61-02-9970-6004.

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.

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will be held on the island of **Layang Layang**, Malaysia

Friday April 30th to Sunday May 9th 1999

The Guest Speakers will be Dr Richard Moon (USA), who was a guest speaker at the 1997 ASM at Waitangi in New Zealand and Dr Alf Brubakk (Norway), who attended the 1998 ASM in Palau The Convener of the Annual Scientific Meeting is Dr Chris Acott. The provisional title of the theme of the meeting is *Gas bubble injury and its treatment.*

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

Speakers at the ASM must provide the printed text and the paper on disc to the Convener before speaking.

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LETTERS TO THE EDITOR

THE DEEPEST DIVE FIRST ?

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1/7/98

Dear Editor

I read Bob Halstead's *Asking the right questions* in Dive Log (see page 174). I had never heard of people doing deep bounce dives early in the day intentionally until I spent a month at Cairns last year. Although I had previously dived in Queensland at Mooloolaba, Lady Elliot Island and a lot around the Gold Coast there had never been any pressure to be absolutely strict with doing the deepest dive first.

Although I always plan to do my deepest dive first, it sometimes is not possible and you have to make an informed calculation of how much risk you are at. In North Queensland it got to ridiculous levels but I learnt quickly from the more experienced divers there. Every dive became a bounce dive slightly shallower than the last. Some divers just lied about their depth and times until even they were getting confused. I felt that instead of adding to safety it was rather dangerous.

On one live-aboard with a lot of new and learning divemasters as the staff, we all had a dive on a shallow reef under the boat one morning about 0830. The absolute maximum depth was 6.5 m and most of the dive was about a metre higher than that. When we come on board we, along with at least a dozen others, were told we were not allowed to dive again until tomorrow and we would have to snorkel. There were a lot of unhappy people. One young lad was unable to complete his Advanced course because of it. My wife and I were really pissed off and got a day boat back to Cairns and lost a couple of hundred dollars we had paid out for another night and day.

This was the same live-aboard that had tried to fine me because I had my mask on top of my head. I was putting my reg on my tank at the time on board the boat, a long way from the water, when the young divemaster started crying out "Diver in Distress! Diver in distress!" I actually looked around to see what was happening. It still makes me angry at their stupidity of following rules blindly. My wife tells me to lighten up and let it go.

On a day dive about a week later I was told, after arriving at the site, that I would have to pay an additional $15 to join a guided tour as I did not have my own buddy. (My wife had an ear infection at the time.) We went down to 15.5 m in a big circle and returned to the boat after 20 minutes, as most were running out of air (small aluminium tanks).

An experienced American diver and myself had plenty of air left and we were instructed to buddy up and dive over the shallow reef at the rear of the boat. My wife was snorkelling here and every now and then gave us a little wave as she looked down on us. Maximum depth here was 5 m but much was in 3.5 m. We just pottered around looking in holes etc. Much more like a snorkel than a dive. We spent 43 minutes in the water all up. When we came back on board they banned my buddy from diving again that day. It was only after a number of protests that he was allowed back in later that afternoon. He told me that for the rest of his trip here he would lie about his depths and times.

After a number of other day dives with different operators, I went out with Nimrod III and had a great time. I still did the bounce dives but I may not have had to as they seemed a very sensible outfit with an experienced and slightly older (40ish) divemaster. Many a dive was reported as 25 m when we actually dived at the 14-18 metre mark.

My wife and I try to be careful and safe in our diving. We do not take unnecessary risks. My wife and I also do longish safety stops of at least 5 minutes and usually 10 minutes or more if the dive was deep. We usually have to inform the divemaster as they become concerned if you hang around on the mooring rope longer than 3 minutes. It all may not help but it will not hurt. The point is we try to keep within safe levels.

Stephen Bilson.

Key Words
Environment, recreational diving, risk, safety

Editor's comment

This letter was sent to Bob Halstead who passed it and his paper ASKING THE RIGHT QUESTIONS to the Journal. As readers know, Bob has little time for many regulations applied to the Queensland diving industry, which, in his opinion, have the effect of interfering with sensible diving. Anything which has the effect of making divers lie about their depths and times to be allowed to dive seems unlikely to increase diving safety

While there is evidence that doing the deepest dive last is associated with decompression illness (DCI) we do not know the actual risk of doing the deep dive last. Neither do we usually know the depth and times of the dives (nitrogen load) which preceeded the final deep dive. This information is seldom published.

BOOK REVIEWS

ONE MAN'S WAR
Diving as a Guest of the Emperor 1942
Robert C Sheats.
Best Publishing Company, P.O.Box 30100, Flagstaff, Arizona 86003-0100, U.S.A.
Price from the publishers $US 19.95. 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 .

This is a fascinating book. The foreword written by Leslie Leaney, Executive Director of the Historical Diving Society USA, introduces us to the author who not only survived capture by the Japanese in the Philippines but went on to be the diving supervisor on the USN's Sealab 1 and 2 before retiring from the USN after 31 years service.

The book starts in Manila in 1941 after the attack on Pearl Harbor. Then came the invasion of the Philippines and war in earnest. The author was serving on the submarine tender USS CANOPUS, which had been converted from a passenger liner twenty years earlier. She carried a diving team to work on the submarines.

The Japanese soon overran the Filipino and US forces. Conditions in the prison camps were at best bad and mostly awful. Just before surrender, the US forces had dumped a vast fortune in silver pesos in 36 m (120 ft) of water in Manila Bay. The Japanese knew it was there and wanted to salvage it. But they had no divers. They used Filipino divers but when three had died in the water they refused to work any longer. So the Japanese, who knew from the prisoners' records who were USN divers, rounded them up and moved them to the seaside. The divers had a much better chance of staying alive away from the insanitary camp inland where there was not enough to eat. Being down by the docks there were better chances of "acquiring" food and so surviving. Some divers had misgivings about working for the Japanese but the chance of better food and so a longer life with perhaps a chance to escape usually won the day.

The equipment available was not suited to the depth they were working in and they ended up using an open helmet with the water level just below the diver's mouth. With much effort and skill they worked slowly, sabotaged the boxes of silver and stole as many coins as they could get away with. Later the Japanese found Moro divers who had Siebe Gorman equipment and knew about decompression tables. They were employed on the same job and not being saboteurs raised much more silver per shift than the prisoners. So it was back to the inland camp and dysentery.

Later came transfer to Japan in an overcrowded transport. The author was lucky to survive because American submarines found the convoy and sank most of the ships carrying prisoners. Somehow our diver survived the winter in unheated huts with not enough to eat, which became nothing to eat if the prisoner did not work in the mine as a slave labourer.

The book is a tribute to the desire to survive and the ability to make the best of a series of worsening bad jobs. It gives an insight into the horrors of war and the ability to cope, or not cope, with circumstances beyond one's control.

Every diver should read this book and step back in time to the days before recreational diving.

John Knight

Key Words
Book review, general interest, history.

SCUBA DIVING EXPLAINED
Lawrence Martin, MD
Best Publishing Company, P.O.Box 30100, Flagstaff, Arizona 86003-0100, U.S.A.
Price from the publishers $US 19.95. 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 .

This contribution by Lawrence Martin is the latest of several books which present underwater physiology and medicine in a digestible form. Dr Martin is a respiratory physician and an amateur scuba diver. In the Preface he laments the difficulty recreational divers face in learning more about underwater physiology than they are taught in the usual courses. His book aims to bridge the wide gulf between popular diving reference material and the medical literature for those who seek a deeper understanding of the issues.

The book is presented in "paperback" format and is approximately 200 pages in length. Chapter titles (paraphrased) include: history of diving; the respiratory system; gas laws and physical principles; barotrauma; decompression sickness; oxygen therapy for diving accidents; gas toxicities; dive tables and computers; stress and hyperventilation; mixed gas diving; women and diving; medical fitness for diving; asthma and diving; safety of recreational diving. There is a series of useful appendices which list: DAN services; recreational diving training agencies; US diving related periodicals (Undersea and Hyperbaric Medicine is included but SPUMS Journal is not); distributors of diving books / magazines; and diving websites.

In general, I believe Dr Martin has pitched his prose at exactly the right level. The amateur diving physiologist will feel "challenged" but not baffled by this book. There is no doubt that the various subjects are dealt with in considerably greater detail than in the usual training courses, but the style is clear, concise enough, and entertaining. Jargon is avoided, and there is a glossary of technical terms for those which cannot be eliminated. The material is contemporary, and Dr Martin has clearly kept himself informed of recent trends in thinking. For example, in his discussion of the decompression disorders, he details the debate over the pathological versus descriptive basis for their classification.

The unavoidably technical material is broken up with 35 short vignettes under the banner of "Diving Odds and Ends". The subject material ranges from details of ultra-deep breath hold diving, through whether or not fish sleep, to Sheck Exley's fatal accident. I found these fascinating, and despite 25 years of active diving and interest in the field, there were still items that were news to me.

Did you know that Jacques Cousteau had a pneumothorax? Australia gets a mention in the context of having the most venomous creature on earth (*Chironex*). The technical material is also broken up by "Test your understanding" type questions with answers provided. Unlike many attempts at this type of knowledge validation, these questions neither insult the reader's intelligence nor expect an unreasonably high level of knowledge.

Dr Martin clearly has an orientation to evidence based medicine and often quotes the literature, particularly when discussing contentious topics. This approach provides some useful material for the more serious diving physician and is one of several reasons why I would recommend this book to any of my colleagues. This is one of the few books in this field that has attempted to address the issues of "risk" in diving. Dr Martin spends a lot of time discussing the philosophy of "diving safety" and fitness for diving in risk related terms, and refers to the existing literature in making his arguments. This is an extremely useful discussion, low on dogma and high on pragmatism, and nowhere is he better than in his chapter dedicated to the issue of asthma and diving. This is a "masterpiece" which should be read by all diving doctors. Like Dr Martin, I am an advocate of what he refers to as the "informed consent approach" to fitness assessments in certain "asthmatics" whose history of asthma does not categorise them as clearly "fit" or clearly "unfit". I am now in the habit of giving all such candidates a copy of this chapter to read.

Overall, I rate this as a very good book which is of use to both divers and physicians. There are a few factual inaccuracies, for example, it is claimed that orally ventilating a patient using a pocket mask connected to a free flow oxygen supply will achieve close to 100% oxygen ventilation. One or two of the diagrams are not particularly clear. Nevertheless, these are very minor concerns and I hope the book achieves the success it deserves.

Simon Mitchell

Key Words

Book review, diving medicine, physiology.

Dr Simon Mitchell is the Director of Diving and Hyperbaric Medicine at the Royal New Zealand Navy Hospital, Auckland, New Zealand

MEDICAL EXAMINATION OF SPORT SCUBA DIVERS. THIRD EDITION.

Editor Alfred Bove.

Medical Seminars Inc. 1998

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

Price $US 21.50. 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

This is a new edition of the book initially edited by Jefferson Davis in 1983 which he re-edited in 1986. These early editions were reviewed by Alfred Bove who has now taken the job as editor following Dr Davis' death. David Elliott is the new reviewer.

The new edition is a great disappointment as the format and majority of the text is largely unchanged from the previous edition.

The book is divided into three sections; Otorhinolaryngology and Ophthalmology, Psychiatry and Neurology, Common Medical and Surgical Conditions (involving 10 subsections) with three appendices.

The previous edition had 68 contributors and the new one lists 99 (covering five pages as an appendix). It is difficult to see what they have contributed for, although the text has been expanded from 45 to 60 pages, much of the expansion is due to a wider font. A few new sections have been added, e.g. a page on the "young diver", a page on "recommended convalescent periods" after ophthalmological procedures and an interesting page on cardiac transplantation.

Unfortunately this edition does not address the problems with the previous editions. With so many contributors the text varies from the dull, drab and didactic to the note form and nebulous.

The introduction commendably states that they try to "avoid dogma and provide guidelines" but much is still

left wanting, e.g. a history of Guillain Barre Syndrome is "considered disqualifying" but "individual exceptions based on careful review, may be made". The advice on benign brain tumours concludes "One senior consulting neurosurgeon advises against diving after any of the above surgery".

The section on "Medications and Diving" covers only a third of a page and lacks any specifics. Even reference to ocular medications in the ophthalmology section refers the reader to a 1995 issue of *Survey of Ophthalmology*.

Some of the chapters are poorly laid out and specific conditions are lost in long screeds of text. The names could have been printed in a bold font for easy recognition. There is an index but it is very restricted, e.g. I read about conjunctival-dacryocystorhinostomies (Jones's Tubes) which was something new to me, but it is not listed in the index, so is difficult to find again.

The book purports to be about "medical examination" of sport scuba divers but no mention is made of a methodical and specific system for examining the diver, only a suggested medical history questionnaire is included as an appendix. The book would really be better titled "Lecture Notes on Medical Conditions Relevant to Scuba Divers".

Despite these problems the book contains much useful information and is worthy of a read to revise your knowledge but it is not recommended for doctors wanting to learn how to undertake sport diving medicals

John Parker

Key Words
Book review, diving medicals.

Dr John Parker is the author of THE SPORTS DIVING MEDICAL.

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THE DEVELOPMENT OF THE MINIMUM PRESSURE OXYGEN TABLES

Chris Acott

Abstract

The treatment of decompression illness (DCI) has been hindered by an incomplete understanding of the pathophysiology, the biophysics of bubble formation, inert gas uptake and elimination kinetics. Treatment protocols are based on minimal animal and human trial data and are to be found in military and government documents and so are difficult to review. This paper briefly traces the development of the recompression treatment tables up to the development of USN Tables 5, 5A, 6 and 6A.

Key Words
Decompression illness, history, oxygen, treatment.

Background

The treatment of decompression sickness (DCS) has been hindered by an incomplete understanding of the pathophysiology, the biophysics of bubble formation, inert gas uptake and elimination kinetics.[1,2]

The majority of the data relating to the development of recompression therapy is found in military and government documents and are difficult to review.[3] Reproduction of these various treatment guidelines and tables have varied with the edition of the Naval diving manual used or the particular Navy.[3] In 1978 Berghage prepared a report listing 67 different therapeutic tables used around the world.[4] Some of these were similar or identical, but were named differently, for example the Royal Navy (RN) 62 and the United States Navy (USN) Table 6.

Past therapeutic guidelines have been derived from various Naval protocols for example, in 1976 the Undersea Medical Society (UMS) issued guidelines for the treatment of offshore DCS which were similar to those used by both the RN and USN and are only relevant to military and commercial diving procedures.[5] All these guidelines reflect the view that DCS was an occupational disease confined to either military or commercial diving. There have been no treatment tables designed for recreational divers whose diving practises are totally different, being multilevel, multi-day and multiple dives per day.[6] Naval and commercial divers are treated immediately symptoms appear while with recreational divers there are inevitable delays to treatment.[6,7]

It is difficult to predict the response to recompression. There may be a group of patients who will respond to any recompression and another group who are refractory to treatment. The longer the delay to treatment the worse the initial response to treatment and the probably the poorer the outcome. However, what constitutes a delay to treatment has not been clearly defined.[1,2,6,7]

Data on outcome has largely been anecdotal and based on different variables. Many reports were derived from retrospective studies and often included non-medical opinion about the success of a treatment protocol. Decompression sickness has often been regarded as an accident, rather than a disease, consequently those treating cases have looked for someone, or something, to blame for its occurrence. Whether this mind set has affected the management and outcome of these cases is unknown.

Pathophysiology

Although Boyle[8] demonstrated bubble formation in living tissues in 1670 and Bert,[9] in 1878, showed that these bubbles were nitrogen, early attempts to explain symptoms following a reduction in ambient pressure included such things as reflex spinal cord damage caused by either by exhaustion or cold, frictional tissue electricity caused by compression, or decompression induced organ congestion and vascular stasis.[3,10-12] Even though all the salient clinical features of DCS were established between 1870 and 1910[8] the complete pathophysiology of DCS is yet to be defined.[1,2] The primary event is the formation of intravascular or extravascular bubbles, which can have mechanical (obstructive, disruptive or compressive), physiological or biochemical effects, however, the relative importance of these effects is still being determined.[6]

Outcome studies

Recompression therapy in commercial and military divers has been so successful that any controlled human studies have remained virtually non-existent.[7] The majority of the human studies have been analyses of case histories and outcome. These studies have several weaknesses: they are retrospective, the initial evaluation may be based on a non-medical opinion and subjective terms (i.e. substantial, relapse, recurrence or minor sequelae) are not clearly defined.[13] In addition, before 1985 the USN did not describe a complete neurological examination in its Diving Manual and so earlier reports would have underestimated the occurrence of central nervous system involvement and so an inappropriate treatment table may have been used.[14,15] Outcome data also vary widely because they are based on different variables (the population studied and the sensitivity of the assay) making any comparison between studies difficult. They therefore need to be viewed with an emphasis on the parameters measured. All studies concentrate on the treatment table used; there is little detail about resuscitation, particularly the prevention of secondary central nervous system damage due to an obstructed airway, or the adequacy of any fluid replacement (type, amount and by which route). In 1987 Green et al. reviewed 430 cases of decompression sickness treated by the RN (250 of these were considered to be serious and 180 were pain only) and found only 18 received intravenous fluid (the amount and type of fluid was not mentioned).[15]

The natural history of DCS may sometimes be spontaneous recovery or improvement. Some early studies report spontaneous remission of both pain only and serious symptoms. In 1870 Bauer published a report of 4 deaths in 25 paraplegic patients but the majority recovered within 1-4 weeks.[9] In 1872 Gal published a report in which paralysed patients either recovered over 5 days to 3 weeks or died from the septicaemic complications of bed sores or cystitis.[9] Both Woodward (in 1881) and Blick (in 1909) reported that the majority of pain-only cases and some of the neurological problems spontaneously resolved.[16] Recently, Green et al. reported spontaneous recovery or improvement in 8 cases of Type 2 DCS.[15]

Recent studies of the treatment of recreational divers have altered the data about successful outcome. These divers' dive profiles may be unknown or uncertain, there is a great variation in their medical and physical fitness and there is usually a considerable delay after the onset of symptoms before presenting for treatment. Delay before recompression treatment is thought to reduce success, however, the time period that constitutes a delay has not been clearly defined.[1,2,6,7] Recently, Lam and Yau, treating compressed air workers, suggested that the delay time should be measured from the conclusion of the dive, not from the development of symptoms, to the commencement of treatment.[17] In 793 cases they found that for every hour's delay, using their definition, there was an additional 0.04 bar pressure requirement for pain relief. Early treatment needs well-trained divers and compressed air workers able and willing to recognise early symptoms, an accessible hyperbaric chamber and a readily available team of treatment professionals. In addition, the period of delay after which no benefit from recompression and hyperbaric oxygen can be obtained is uncertain.[3]

Controversial issues

By 1939 recompression had become the accepted method of treatment but there was disagreement concerning its application.[18] Even today similar controversies exist: which treatment depth to be used in unresponsive or deteriorating cases,[19] what is the optimum pressure of oxygen to use and what diluent inert gas to use with oxygen.[3] The use of saturation therapy for non-responders or the repeated use of hyperbaric oxygen after initial treatment is still being debated, but there is no agreement on which hyperbaric oxygen table should be used.[20,21]

Development of the therapeutic tables

Decompression sickness was first described by Triger

in coal miners in 1841.[9,10] Recompression was first proposed as treatment in 1847 by Pol and Watelle, whose patients were coal miners working in compressed air to deepen the mine shafts.[22] Bert (1878), Moir (1889), Snell (1895) and Zuntz (1897) were other early proponents of recompression.[9,10,23,24] Heller et al. in 1907 and Keays in 1909 used recompression on an ad hoc basis.[10,25] Keays showed persistence of symptoms in 14% of caisson workers who were not recompressed compared to 0.5% in those who were.[25] Until 1912, when Ryan published the first treatment regime, the treatment of DCS had been on an ad hoc basis. Ryan suggested a return to 2/3rds the original pressure followed by a slow decompression.[10] In 1917 Levy advocated a return to the original pressure, again followed by a slow decompression.[10] Both regimes had limited acceptance. In 1924 the first standardised recompression tables were published by the USN. This recommended that the diver was rapidly recompressed to 45 pounds per square inch gauge (psig) (approximately 30 msw or 4 bar) with further recompression to 60 psig (approximately 40 msw or 5 bar) if there was no improvement. Decompression was started as soon as the symptoms resolved. The USN published another table before 1937 which recommended recompression to the depth of relief plus 1 atmosphere, decompression from this depth was the diving table air decompression schedule for that depth .[3]

In the 1930s the RN began using oxygen decompression in air dives to 300 fsw (90 msw or 10 bar). This procedure was based on animal experiments (12 goats), human chamber testing (10 divers) and actual naval dives (58 dives).[26]

By 1935 the USN air treatment tables were noted to afford relief only in mild cases so Behnke and Shaw began experimenting with the use of oxygen. They believed that oxygen should be used because it would create a maximum elimination gradient for nitrogen and afford immediate relief of bubble induced ischaemia.[27] Behnke proposed that reluctance to use oxygen had been due to a lack of conclusive experimental data on its efficacy, a lack of suitable facilities for administration and the fact that human tolerance was unknown. Behnke et al. conducted human oxygen tolerance studies using 12 divers. The data showed that 100% normobaric oxygen could be breathed for 6 hours without pulmonary symptoms while convulsions occurred after 3 hours at 3 bar (ATA) and 45 minutes at 4 bar.[27,28] They compressed 26 anaesthetised dogs to 5.4 bar for 105 minutes and then surfaced them in 10 seconds to produce severe cardiopulmonary and neurological DCS. The dogs were then recompressed to 3 bar (20 m) either breathing 100% oxygen or air. They choose 3 bar because of their human oxygen tolerance studies data and the postulated eleven fold increase in nitrogen elimination compared to air at 1 bar. Both groups initially responded well to recompression, but the dogs recompressed on oxygen had a better outcome with fewer recurrences of symptoms. They concluded that oxygen recompression to 3 bar had a better outcome than recompression using air, that 3 bar (20 m or 66 ft) was not an adequate pressure to reverse the CNS signs (paralysis) and that severe DCS caused plasma loss. They noted that the bubbles were eliminated within 1 hour in these dogs but concluded if this was to occur in humans 2 hours would be needed because man's circulation time is twice that of a dog. [28]

Their next experiments were designed to test the pressure needed to prevent or reverse the CNS signs/ symptoms ("to prevent paralysis").[27] Eight dogs were used in 15 experiments and again their model of severe cardiopulmonary and neurological DCS was used. These dogs were recompressed to 60 psig (5 bar or 40 msw) breathing a 50% nitrox mixture, or air if this was not available, oxygen was not used in decompression. In the first 8 experiments 1 dog failed to develop symptoms and so was not recompressed, 3 recovered, 1 partially responded, 2 failed to respond and 1 died following recompression. Overall the data from the 15 experiments showed that in 7 experiments the dogs survived (1 dog was used 4 times but failed to recover in the 4th experiment, 1 dog responded in 1 experiment but failed to respond in a subsequent experiment, 1 dog recovered function after 14 days having fully recovered on a previous experiment, 1 dog required 2 treatments); 6 dogs had an incomplete recovery; 1 died and 1 failed to produce symptoms in 1 experiment but failed to respond to treatment in another. From the data from these 2 groups of experiments Behnke and Shaw concluded that any serious symptoms would require a combination of rapid recompression and hyperbaric oxygen. These two groups of experiments are also important because they were the basis upon which subsequent treatment depth and oxygen pressure have are based .[27]

In 1937 Behnke and Shaw published their oxygen tables based on these animal data. For serious cases the maximum depth of 50 msw (165 ft or 5 bar) breathing either a 50% nitrox mixture or air followed by a decompression to 60 fsw (18 m, 2.8 bar) over 45 minutes where oxygen was to be breathed for 1-2 hours. The patient's response determined the time spent at 165 fsw (minimum 15 minutes and maximum 2 hours). One hundred and sixty five feet (165 fsw) was chosen because:

1 bubble shrinkage would be to 1/6th its surface volume;
2 pressure resolution of all bubbles was thought to require exceedingly high pressures and, by the time the serious cases were recompressed, tissue gas would have diffused into the blood stream limiting its capacity for any further absorption. They preferred to use oxygen at 3 bar for complete elimination of gas emboli.

For mild cases (pain only) they recommended recompression to 60 fsw (18 m or 2.8 bar) breathing 100% oxygen for 1 hour followed by a 30 minute decompression. For unrelieved symptoms they suggested a prolonged stay

at 60 fsw (18 m or 2.8 bar) breathing oxygen for 2-3 hours in every 24. They limited the exposure to 3 bar oxygen in 24 hours to 3 hours.[27] These tables were not published or used by the USN because the USN Bureau of Medicine and Surgery decided that oxygen breathing in a chamber was not "sailor-proof". The risks of an oxygen convulsion and fire were considered too great for it to be used universally by the Navy.[3] However, these tables served a model for future recompression procedures.[3]

In 1939 Yarbrough and Behnke recognised that effectiveness of recompression was related to its prompt application and recommended that for 12 hours following a dive a diver "should not be further removed than 1 hour's travel time".[18] They based this maximum one hour delay on their own clinical and experimental data and data from Keays, Langlois and Bornstein.[18,25]

By now there was agreement that recompression was the treatment of choice, however, at that time there were 4 approaches to the depth to be used:

1 compression to the depth of relief;
2 compression to a depth greater than that required for relief;
3 compression to the depth of the original dive;
4 compression to a depth greater than the original dive.

Yarbrough and Behnke considered that, because the amount of gas in bubble form was unknown, options 3 and 4 (compression to the depth of the dive or deeper than the original dive) could be eliminated from therapeutic consideration. Their guide was the relief of symptoms, and 1 ATA (bar) was added empirically to completely restore circulation to any affected tissue. Using Behnke and Shaw's data[27] the minimum pressure for the treatment of mild cases (symptoms relieved at a depth less than 30 fsw (20 m or 3 bar) became 100 fsw (45 psig, 4 bar or 30 m) with oxygen being used from 60 fsw (18 m or 2.8 bar) if available. Decompression was staged, based on the Haldanian principle, so that the tissue gas pressure never exceeded the ratio of 2:1 compared to the environmental pressure. These tables became known as either the "short oxygen" (when oxygen was used) or "short air" table.[18]

In serious cases immediate recompression to 75 psig (6 bar, 165 fsw or 50 msw) breathing either a 50% nitrox mixture or air was recommended with oxygen being used from 60 fsw (18 m or 2.8 bar) if available. These tables became known as the "long oxygen" (when oxygen was used) or "long air" table. Again decompression was based on the staged Haldanian principle.

Time spent at 165 fsw was to be between 30 and 120 minutes. This was based on;

1 Behnke et al.'s previous clinical and animal data;
2 Borstein's recommendation of 30 minutes;[18]
3 and R H Davis's opinion that it was "useless to wait longer than 2 hours".[18]

If the treatment failed then the patient was immediately recompressed to the pressure of relief (this was usually found to be less than 3 ATA) and maintained for 12-24 hours followed by a slow decompression. This introduced the treatment concept of an overnight soak. These tables were published in the BUMED News Letter in 1944 .[3]

Development of tables 1 to 4

The long oxygen treatment table was used to treat 50 cases of "helium bends" and was successful in 49 patients.[18] However, when it was used to treat 10 air divers there was a 50% symptom recurrence rate with an overall failure rate of 30% in 30 divers treated. Even in human trials the long oxygen table failed in 6 out of 10 divers when tested. The shorter oxygen table, however, was successful in the 6 divers for whom it was used.

By 1945 it was apparent that these tables gave no better results than using the regimes of 1924. These failures led to Van Der Aue et al developing the USN Treatment Tables 1-4 (with Treatment Table 1A and 2A using only air).[29] Development of these tables involved subjecting healthy divers to a 130 fsw (39 m)/60 minute working dive and after a 30 or 60 minute surface interval using the treatment table under evaluation. If the treatment table did not prevent DCS it was modified until no DCS occurred. A total of 84 dives were made using 33 subjects, however, when Table 4 was tested, there were no preceding work dives and the 6 subjects tested all reported fatigue following exposure.[3,30] The short oxygen table became USN Table 1, the short air table was lengthened and became USN Table 1A, 60 minutes of oxygen breathing were added to the long oxygen table (which became USN Table 2) and the long air table was lengthened and became USN Tables 3 and 4. The RN developed their equivalent tables (RN 52, 53, 54, 55) a little later and these tables remained in use world-wide in both commercial and military diving for the next 20 years.[3,30]

In 1947 Van Der Aue et al. reported on the first 113 patients treated with these new tables.[31] Complete relief occurred on the initial compression in 107 cases, 4 had recurrences and 2 had residual problems. The initial success rate with tables 1-4 was excellent with an overall reported failure rate of 6%.

These tables were not subjected to a further review until Slark in 1962 and Goodman in 1964 published studies which showed failure rates of 24-47%.[32,33] In these series, recreational divers accounted for almost 46% of the initial treatment failures. There were no reported failures of Tables 3 or 4 when used promptly on naval divers.

These reports led Goodman and Workman to begin a series of treatments based on moderate pressures of 100%

oxygen. They believed that these "minimal pressure" oxygen tables relieved ischaemia without further exposure to inert gas and provided a maximum gradient for inert gas washout. They also tried to convince diving physicians that deeper treatments were not better because of the decrease in bubble volume vs radius changed little at depths deeper than 18 m (2.8 bar) and that deeper treatments were paralleled by an additional inert gas uptake.[34] Goodman defined what he considered to be the fundamental aspects of the treatment of DCS:

1 compression to reduce the bubble volume and radius to decrease any tissue reaction;
2 relief of focal ischaemia caused by endothelial irritation.[33]

Assuming the strength of Tables 3 and 4 was the 33 fsw (10 m or 2 bar) soak, they began conducting trials at this depth. The patient was compressed to 33 fsw (10 m) breathing oxygen. If all the symptoms were relieved after 10 minutes the patient completed an extra 30 minutes and then decompressed. If relief was not obtained after 10 minutes then the patient was further compressed to 60 fsw (18 m). They treated 150 divers (110 military and 40 civilian) with this regime. The 5 divers (4 military and 1 civilian) treated at 33 fsw had recurrence of their symptoms and had to be treated again.[34] Retrospective statistical analysis of their data showed that oxygen breathing time and depth were related to the treatment adequacy (the minimum adequate exposure time was 30 minutes with a 90 minutes total treatment time). As a result the 33 fsw (10 m) treatment was abandoned and all divers were recompressed to 60 fsw (18 m). Alternation of oxygen breathing with air for periods of 5 to 15 minutes was introduced to reduce the risk of oxygen toxicity. USN Tables 5 and 6 were developed from their data.[34]

Arterial gas embolism

Arterial gas embolism is a relatively recent diving disease. It was clinically defined in the 1930s after the beginning of submarine escape training.[10] Following the development of USN Tables 1-4 it was either treated on Table 3 or 4 which meant a 22 or 38 hour stay in the chamber irrespective of the patient's inert gas burden. Before the development of Tables 3 and 4 gas embolism was treated on the same protocols used for serious DCS.

In 1967 Waite and Mazzone began to re-evaluate the treatment of gas embolism.[35] In a series of experiments they embolised 14 dogs, observing bubble behaviour through a cranial window. Eleven dogs were successfully embolised, 8 were embolised at 1 bar and 3 at 2 bar. Five of the dogs embolised at 1 bar were not treated. Two died within 20 minutes and 3 survived with residual problems. The other 3 dogs embolised at 1 bar were treated with a dive table of 170 fsw (52 m) for 10 minutes with staged decompression. The 3 embolised at 2 bar were surfaced and then treated with the same protocol. All the treated group survived. It was noted that all the bubbles disappeared between 3 and 4 bar with no bubble reappearance following decompression. Because of the prejudices of the USN Bureau of Medicine and Surgery only 6 bar (50 m or 165 ft) had to be used in any treatment tables.

Waite et al. at the USN Submarine Medical Center later modified USN Tables 5 and 6 for the treatment of cerebral arterial air embolism in submarine escape trainees. These tables were called Tables 5A and 6A.[35]

Table 5A was later abandoned in 1976 because it did not allow enough time to assess if there has been any resolution of symptoms.[36] The diver would also not have had enough time to adjust to the thermal stress, noise and narcosis of a rapid compression. These would also interfere with the attendant's assessment.[37]

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ADJUVANT THERAPY FOR DECOMPRESSION ILLNESS

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Key Words
Accidents, decompression illness, drugs, treatment.

Current Knowledge

The definitive treatment of DCI is administration of oxygen in a recompression chamber. However, in recreational diving a chamber is rarely available on site, often necessitating delays of several hours before recompression can be initiated. This is potentially a "golden period" during which simple measures may make a significant difference in outcome.

Surface (First Aid) oxygen

In severe DCI, which can be complicated by aspiration of water or vomitus, administration of oxygen is a standard first aid measure to reverse hypoxaemia and enhance oxygen delivery to under-perfused tissue. Additionally, when breathing 100% oxygen, the partial pressure gradient for diffusion of inert gas from bubble into tissue ("oxygen window") is increased. This has been observed in experimental animal preparations.[1,2] The effectiveness of oxygen administration to injured divers is supported by clinical experience. Analysis of 2,192 recreational diving accidents reported to the Divers Alert Network revealed that 68% of divers who received surface oxygen reported partial or complete resolution of symptoms before recompression versus only 40% who had no supplemental oxygen.

Blood glucose control

Both brain[3] and spinal cord[4] injury can be worsened by hyperglycaemia. The most likely mechanism is accelerated production of lactate producing intracellular acidosis. The effect appears to become significant above a threshold plasma glucose of around 200 mg/dl (11 mM).[5,6] Administration of even small amounts of glucose, for example one litre of intravenous 5% dextrose solution, even in the absence of significant hyperglycaemia, may worsen neurological outcome.[7] Therefore, unless treatment of hypoglycaemia is required, it is best not to administer glucose containing intravenous solutions. If there is reason to suspect hyperglycaemia (e.g. if high dose corticosteroids are prescribed) plasma glucose should be measured, if feasible, and appropriate treatment initiated.

Fluids

Interaction of bubbles with vascular endothelium causes a capillary leak resulting in loss of plasma volume. Haemoconcentration, often of severe degree, has been reported in DCI,[8-11] and post-treatment residual symptoms have been correlated with the degree of haemoconcentration (see Table 1). Fluid administration can replenish intravascular volume and reverse haemoconcentration, thereby increasing tissue perfusion.[8]

Indirect evidence suggests that aggressive hydration during minor surgical procedures can result in more rapid elimination of anaesthetics,[12] from which one might infer that a similar approach in divers with decompression illness may accelerate the washout of excess inert gas. It has been demonstrated that augmentation of central blood volume and cardiac preload using supine position,[13] head down tilt[14] and head out immersion[13,14] significantly increase the rate of inert gas washout. Therefore, fluid administration may be advantageous, even in patients with DCI who are not dehydrated.

Rapid intravenous administration of hypotonic fluids can cause CNS oedema,[15] whereas administration of fluids which are hypo-oncotic but not hypo-osmolar has no effect on CNS water. There is therefore no advantage of colloidal solutions over crystalloids,[16,17] and any isotonic IV fluid without glucose, such as normal saline or Ringer's solution, will suffice. Theoretical objections have been raised to the use of fluids containing lactate (e.g. Ringer's lactate or Hartmann's solution) on the grounds that liver metabolism may be reduced, especially if the patient is hypothermic, and that lactic acidosis can result. However, lactate is metabolised by most tissues, not only by the liver, and the small amounts of lactate in Ringer's lactate solution are unlikely to contribute significantly to acidosis.

TABLE 1

HAEMATOCRIT IN DIVERS WITH DCI AND IN CONTROLS (from Boussuges et al)[11]

		Haematocrit (%)		
	Number	**Median**	**Minimum**	**Maximum**
Controls	16	42.5	39.0	48.0
DCI without neurolgical sequelae	39	42.0	35.0	57.0
DCI with neurological sequelae	19	47.5	32.0	69.5

IV administration of fluid is the most rapid method of rehydration and for critically ill patients it is generally agreed that IV administration is preferable to the oral route. However, there is disagreement about whether there is any advantage to parenteral fluid administration for divers with less severe disease, particularly for divers with pain as the only symptom. In dehydration due to other clinical situations, such as cholera, moderately severe dehydration can be satisfactorily treated using appropriate oral fluids.[18] Therefore, it is argued that many divers with DCI, provided they are alert and not nauseated or vomiting and sufficient volumes of fluid can be ingested without undue interruption of oxygen administration, can be satisfactorily rehydrated orally.

Ingestion of plain water stimulates urine output via a decrease in plasma osmolality and inhibition of antidiuretic hormone (ADH) secretion, producing a false impression of adequate rehydration.[19,20] Therefore a solution containing electrolytes, particularly sodium, is preferable. Maximum water absorption occurs at a sodium concentration of 60 mM and glucose concentration in the range of 80-120 mM. Gastric emptying rate may be reduced by protein or glucose concentrations greater than 5% (252 mOsm/kg). An ideal solution for rehydration in diarrhoea has been suggested as containing approximately 30-60 mM sodium, 70-150 mM glucose and osmolality of around 240 mOsm/kg,[21,22] a mix attained by few commercially available beverages, which are usually low in sodium and high in carbohydrate (see Table 2).

The rate at which rehydration can be achieved after mild dehydration in normal volunteers have revealed mixed results. In one study dehydration of 4% of body weight (12% reduction in plasma volume) was induced by exposure to a hot, dry environment.[19] Administration over four hours of fluid equal to the volume lost, using either demineralised water or glucose-electrolyte solution (sodium 22 mM, osmolality 444 mOsm/kg), failed to normalise plasma volume, although urine output had increased to 180-380 ml/hour. Even after an additional 24 hours of ad lib fluid intake plasma volumes were 2.4-5.5% below pre-test values. On the other hand, in a study of dehydration induced by exercise plasma volume was restored within 20 minutes by ingesting water with sodium chloride (sodium concentration 77 mM) but not until one hour using a sucrose solution.[20]

A palatable oral rehydration fluid with appropriate electrolyte and carbohydrate concentration can be improvised by mixing one part orange or apple juice with two parts water and adding 1 teaspoon of salt to one litre of the mixture. If salt is not available, the appropriate sodium concentration can be achieved by diluting the juice with a mixture of one part sea water and 9 parts fresh water.[23]

Provided that the patient is not vomiting, an intake of 1,000-2,000 ml per hour is safe and tolerable. End points for fluid therapy should include normal haemodynamics and haematocrit. Urine output should exceed 1 ml/kg per hour, bearing in mind that if large volumes of hypotonic fluids

TABLE 2

COMPOSITION OF BEVERAGES

Beverage	Sodium (mM)	Potassium (mM)	Glucose (mM)	Osmolality (mOsm/kg)
Ideal replacement	30-60		70-150	240
Water	0	0	0	0
Apple juice	7.8	19.1	784	730
Club soda	9.7	0.5	0	20
Coca Cola Classic™	1.8	0.0	628	750
Diet Coke™	1.0	1.4	5	10
Gatorade™	23.0	3.0	256	330
Ginger Ale	3.2	0.4	527	535
Orange juice	14.5	28.2	708	793
Powerade™	10.7	3.4	471	499
Snapple™ Kiwi Strawberry	0.0	-	818	818
Sprite™	6.0	0.0	595	607
Beer	2.0	8.0		600
Pedialyte™ (Ross)	45.0	20.0	139	269
Rehydralyte™ (Ross)	75.0	20.0	139	329
WHO-Oral Rehydration Solution	90.0	20.0	167	387

are used, the urine output may be elevated out of proportion to the rehydration. However, fluid should not be withheld just because an ideal liquid is not available.

If fluids are not available rehydration can be simulated by immersion to the neck in water, which redistributes 500-800 ml of blood from the extremities to the thorax, increasing cardiac output. Provided that the diver can be kept warm, head out immersion, although impractical during transport, enhances inert gas washout.[13]

Corticosteroids

Use of pharmacological doses of glucocorticoids to treat neurological DCI has had variable results. In a retrospective review of arterial gas embolism (AGE) Pearson and Goad reported that after initial improvement secondary deterioration occurred less often in divers who had received glucocorticoids.[24] However, glucocorticoids have not been shown to be beneficial in the treatment of head injury,[25-27] or in animal models of decompression illness.[28] In a series of AGE cases analysed retrospectively for a possible relationship between glucocorticoid administration and outcome, no benefit was evident.[29] However, in traumatic spinal cord injury there is evidence that early administration (within 8 hours after injury) of methylprednisolone (30 mg/kg intravenously over one hour followed by 5.4 mg/kg/hour for 23 hours) can improve outcome six months after injury.[30] Such high doses have not yet been specifically tested in DCI, either in animals or humans. Moreover, the only systematic animal studies have used only short term outcomes, with somatosensory evoked responses as the end point, a measurement which, in humans, correlates poorly with clinical neurological function. There are currently no published data providing unequivocal support for the use of corticosteroids in DCI, although the evidence to the contrary may only be due to the lack of a trial using an appropriate dose. Therefore, the issue of efficacy of these compounds in this disease remains an open question.

Lignocaine

In models of AGE in both cats[31] and dogs,[32] lignocaine administration designed to achieve standard clinical plasma drug levels has improved short term neurological outcome. Randomised trials of lignocaine in humans have not yet been reported, although anecdotal reports support its use in DCI.[33,34]

Safe intravenous administration of lignocaine requires an infusion pump and the capability of dealing with untoward effects such as seizures. Early experience with intramuscular administration in the "field" for arrhythmia prophylaxis in acute myocardial infarction suggests IM injection is a safe method of administration of this drug to divers with DCI immediately after the onset of symptoms.[35] Injection of 200-400 mg into the deltoid muscle results in therapeutic plasma concentrations for up to two hours. Routine recommendation of such a regimen would require demonstration of benefit in an appropriately designed trial.

Anticoagulants

Because of the potential for bubble-blood interactions to cause platelet deposition and vascular occlusion refractory to recompression, it has been speculated that inhibitors of platelet function and soluble clotting factors might offer some benefit in DCI. In asymptomatic divers administration of aspirin and other anti-platelet drugs reduces the mild drop in platelets observed after routine dives.[36,37] A single case report of heparin administration to a patient with neurological bends indicated neither benefit nor harm.[38] However, animal studies in which single agents were administered have shown no benefit of anticoagulants, except for one study,[39] in which only a triple combination of indomethacin, PGI_2 and heparin resulted in a beneficial short term effect in a canine model of AGE.

Histological evidence of haemorrhage has been described in arterial gas embolism,[40] inner ear decompression sickness[41] and spinal cord decompression sickness,[42-45] suggesting that antiplatelet drugs or other anticoagulants may actually worsen outcome in DCI. However, in individuals with severe neurological bends and leg weakness, deep vein thrombosis (DVT) and fatal pulmonary thromboembolism have been described.[46] Therefore in these patients some form of prophylaxis against DVT, which may include low dose heparin or low molecular weight heparin,[47] is recommended.

The analgesic effects of aspirin and nonsteroidal anti-inflammatory drugs (NSAIDs) prescribed for the discomfort of pain-only bends may render it difficult to assess the clinical response to recompression.

Body temperature

Animal models of CNS injury have demonstrated that outcome is significantly worsened by hyperthermia.[48] So fever in a patient with DCI should be vigorously treated.

Whether hypothermia may be beneficial has been an open question. In a recently published study of closed head injury (Glasgow Coma Scale 3-7), the effect of induced hypothermia on outcome was examined in 82 patients.[49] Forty patients were in the experimental group and were cooled to 33°C using cooling blankets and chilled nasogastric lavage fluid. Minimum body temperature was achieved on average 10 hours after injury. The patients were kept at 32-33°C for 24 hours then rewarmed. All patients

were mechanically ventilated during the experimental period. Twelve months after injury, 62% of the patients in the hypothermia group and 38% of those in the normothermia group had good outcomes (Glasgow Outcome Score of 4 or 5: moderate, mild, or no disabilities). For patients with severe neurological DCI active cooling might be a modality worthy of investigation.

Future developments

Pressure and oxygen remain the mainstays of treatment for DCI. However, there are relatively few degrees of freedom in the choice of ambient pressure, time of treatment and PO_2. Unless there is a major advance in the prevention of oxygen toxicity it is unlikely that any new treatment tables will offer major therapeutic advantages over current implementations. I believe that the next major improvement in DCI treatment will be in pharmacotherapy.

Fluorocarbons

Both oxygen and inert gases are highly soluble in fluorocarbons. Thus, intravenous administration of these agents in doses sufficient to increase the transport of these gases in plasma should simultaneously increase tissue oxygen delivery as well as accelerate insert gas washout. Animal studies have in fact demonstrated a reduction in mortality in gas embolism.[50,51] Perfluorocarbons may become available for clinical use in other settings, which would facilitate human studies in DCI.

Adjunctive agents

Prolonged anoxia due to interruption of blood supply can produce rapid cell death due to depletion of intracellular energy sources. Reperfusion of ischaemic brain before cell death has occurred can result in rapid recovery of cellular respiration and ATP synthesis and return of electrical activity. However, increased production of oxygen free radicals can lead to neuronal death due to ischaemia-reperfusion and death delayed many days (apoptosis). Understanding of the mechanisms underlying these events may lead to the development of compounds which may improve outcome after DCI. These concepts have been reviewed by Warner.[52]

After CNS injury there is a release of excitatory neurotransmitters such as glutamate, which then facilitates the entry of calcium, which is neurotoxic, into cells.[53] Blockade of voltage-dependent calcium channels by nimodipine and nicardipine has been shown to ameliorate somewhat the damage due to subarachnoid haemorrhage and ischaemic stroke,[54] but to have little effect upon outcome after global ischaemia induced by cardiac arrest.[55]

Calcium entry into cells can also occur with activation of specific glutamate receptors, such as N-methyl-D-aspartate (NMDA), a-amino-3-hydoxy-5-methyl-4-isoxazole propionate (AMPA) and 1-aminocyclopentyl-trans-1,3-dicarboxylic acid (t-ACPD). After an ischaemic insult, blockade of these receptors might conceivably reduce entry of calcium into the cell, thus preserving neuronal function. NMDA receptor blockers can protect against focal insults,[56] and AMPA receptor blockers protect against both focal and global injury.[57-62]

Compounds related to the corticosteroids, but without many of the side effects of corticosteroids ("lazaroids"), have been tested in subarachnoid haemorrhage with both positive[63] and negative[64] results.

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IS THERE A CONSENSUS VIEW ON RECOMPRESSION PROCEDURES ?

A PANEL DISCUSSION WITH AUDIENCE PARTICIPATION

Members of the panel
Drs Mike Bennett, James Francis, Des Gorman, Simon Mitchell and Richard Moon
Moderator Chris Acott

Key Words
Decompression illness, hyperbaric oxygen, mixed gas, research, treatment.

Moderator (Chris Acott)

Decompression illness (DCI) is a multi-system disease. With other multi-system diseases, such as septic shock, we optimise everything, we ventilate them, we maintain their cardiac output, we give them antibiotics, and intensive care nursing. But in spite of all our efforts there is a percentage of patients with septic shock who will die. I wonder whether it is the same with decompression sickness. Perhaps we are at a plateau now, with the best that we are going to get. Perhaps results in the future are not going to improve that much. Would anybody like to comment on that question?

James Francis

There is no eleventh commandment of "Thou shalt get better with recompression".

Des Gorman

I do not think we are at an endpoint of outcome, quite the opposite. There is a wide range of opportunities for improving treatment in DCI, but they may well be pharmaceutical rather than developments of pressure and oxygen. There are people who respond quickly to recompression, and it probably does not matter much what is used. Adding oxygen is a pragmatic, sensible starting point. But remember the reviews of the US Navy Tables, treatment Tables 1 and 1A, they had a very high success rate, 89% first time and 95% eventual success.[1,2]

I think people are overlooking the benefit of additional compression. There is no doubt that we see a small group of people in Auckland who get better with additional compression. If they do not get better at 18 m, they get better at 30 or 50 m. However, I think one can fiddle with pressure and oxygen for the next ten years and not see a dramatic improvement in the absence of earlier presentations. But I think there are significant inroads to be made from pharmaceuticals.

The fundamental problem is that DCI is a disease in which it is very difficult to perform controlled, prospective, randomised trials. We have, with the oxygen-helium trials in Auckland, a number of problems. First of all, how does one blind the attendants when using helium? The patient only has to say one word and the blinding has gone out the window! Yesterday Simon Mitchell presented what appeared to many of you to be a very complex scoring system.[3] However, without such a scoring system, one can end up showing no benefit for quite dramatically effective treatment when using a system as crude as complete, incomplete and no recovery. In our oxygen-helium trial there was no apparent benefit. We are restudying these cases and re-coding them to see if a difference emerges.

How does one obtain the patient group we want? The major weakness of our study was that, if we are to use a gas which is designed to shrink bubbles, we need patients who present early. Despite every effort, our mean time delay is between 2 and 3 days. What is the point of a trial looking at bubble shrinkage when people present after 48 to 72 hours. If we show a dramatic benefit for helium when we re-code the data, I think we are going to have to look very, very carefully at Type 1 errors. One would have to say "What on earth would be the plausible benefit at that period after onset"?

I am not sure how we correct that problem. We have done everything in our power, in terms of education programs, to get people to present early but the denial and culpability issues (which run through the head of the average diver with decompression illness) mean that they are almost unamenable to change.

So we have a disease which is progressive and which presents late. We have amazing heterogeneity in DCI; we have no clinical markers for follow up; we have no magic blood level to measure to show an objective score of outcome. We have, in the case of gas used for treatment, an almost impossible task in blinding them. And the end result is that, with a study like the oxygen-helium study, we have to say that, given our failure to recruit people early, it should be junked. And that is exactly what we are going to do. Round up the data, reclassify it and see if with a more sensitive scoring system, anything emerges. We are almost hoping that it does not, because if it does we will have to try and explain why it did.

The lignocaine study is a lot easier to do. We are doing a lignocaine study on divers, but late presentation is still a problem. We are more interested in the results of the cardiac surgical group, because in them we have pre-morbid data and post-morbid data and a measure of the insult. We have timing of insult and a reasonably homogeneous group in terms of the other phenomena.

The 1995 attempt at consensus went no further than the 1990 attempt at consensus, which was pretty much the same as the 1979 consensus, and my advice then was, as it is now, get the old document, white out the date and just change the date.[4-6]

Moderator (Chris Acott)

Does saturation therapy (which I understand is the only therapy where if anything goes wrong there is a very, very high chance of causing morbidity to the patient and the two attendants who are in the chamber), have any advantage over repeat hyperbaric oxygen up to 20 or 30 treatments, with or without SPECT scans.

Richard Moon

The patients in our retrospective review suffered from the same problem that Des has just pointed out.[7] They arrived at our hospital very late, often one or two days after the event, so a treatment which is designed to optimise gas bubble volume reduction is not likely to offer any particular benefit. I believe that, in general, for patients who show up late, it is not worth the cost and the effort, and frankly the risk, of a saturation treatment. Although, if one has the capability to do it, it can be considered. If one is treating a patient with severe neurological disease, and who is responding dramatically to initial recompression at 18 metres breathing oxygen, it is certainly very tempting to keep the patient in the chamber to administer hyperbaric oxygen repetitively and frequently, more intensively than one can do with repetitive Table 6 treatments. I think that saturation treatment will remain predominantly a tool for the off-shore diving industry and possibly the military, where the capability exists and the time to recompression is very short.

I think if there is a consensus it should be a very simple one. I believe that first aid, pressure and oxygen should be administered to individuals with decompression illness. Also I think that pressure and oxygen should be administered repetitively until there is a clinical response or a "plateau", meaning no measureable clinical improvement. I have seen no convincing evidence that large numbers of treatments offers any benefit. In my opinion the SPECT brain scan data that have been presented as evidence for improvement are not up to scientific standards, in particular because the scans have not been read independently by blinded observers. I think that idea is out on the fringe and should be considered experimental.

Peter Chapman-Smith

How long after the injury it is worth embarking on recompression treatment for sports divers who delay, sometimes they roll up weeks later?

Richard Moon

There are a few "case collections" suggesting that, even several days after the event, one can see objective neurological improvement.[8-12] Whether weeks later one can do the same thing, I have my doubts but I have no direct experience. But, given that hyperbaric treatment is fairly safe, inexpensive and readily available, if somebody was to turn up in my chamber a week after onset, I would probably give it a shot. Two weeks, five weeks later, I do not know.

John Knight

In 1980 SPUMS had a meeting with the Singapore Navy where C L Yap, one of their diving doctors, reported on 58 diving fishermen who, when they developed DCI, remained on board the boat for up to a fortnight before reaching Singapore and treatment.[13] The mean time to treatment was 90 hours. All 11 type 1 cases recovered completely after a RN Table 62 (USN 6). Of the 47 Type 2 cases, who were treated with repeated hyperbaric oxygen, 18 (38.3%) had complete recovery and 20 (42.6%) had more than 50% recovery. Only 3, who had complete paraplegia and bowel and bladder deficits, failed to improve. So about 80%, got useful neurological improvement from their treatment with hyperbaric oxygen as late as three weeks.

Peter Chapman-Smith

What is the role of ambient pressure oxygen in between HBO treatments? We treat people once or twice a day. Is oxygen, at ambient pressure, ever used in between those once or twice a day treatments?

Richard Moon

I cannot answer that, except to say that if one does administer ambient oxygen between hyperbaric treatments, one is much more likely to experience pulmonary oxygen toxicity during subsequent treatments. We and others have seen that. Whether or not there is any difference in outcome, I do not know.

Unidentified speaker

In the 1960s and 70s Carl Edmonds in the Australian Navy was using normobaric oxygen between treatments. He found that this may have decreased the number of repeat treatments that the patient needed.

Unidentified speaker

Is there any place now for saturation treatment? For which patients would you still use the saturation treatment, or the very long air tables?

Richard Moon

I think saturation treatments should be reserved for the patient who presents early, and in a facility which has the necessary hardware and technical and medical support to provide saturation.

Unidentified speaker

Another problem is technical divers, and even military divers, going for deep heliox dives. That is, dives to 80 to 100. What kind of recompression table are you able to recommend for the deep blow up for these divers?

Richard Moon

I assume your question is related to civilian facilities. For deep blow up or technical dives, I think one has to often resort to the deep tables, such as the Lambertsen Table 7A or the US Navy Table 8, which cannot easily be administered in most civilian chambers. In these cases I

think the only thing that one can do is to treat using a table such as USN Table 6 with extensions until there is some kind of clinical response. I would be inclined, if I had a diver very soon after a blow up, to go deep rather than shallow, because the diver is likely to have a large volume of inert gas bubbles, which will be more likely to respond to pressure rather than simply oxygen. But the situation you are describing is uncommon, and it is one for which many civilian facilities lack the capability or the experience.

Jim Marwood

It seems that there is not much we can do other than to induce divers to come earlier. I remember an instructor, a long time ago, who quite early in the course would draw a tombstone on the blackboard and say "That is for any of you who is impatient enough to come up quickly, and especially if you hold your breath!". The PADI course, which so many divers do, stresses the positive and the pleasures of the sport, all very enthusiastically. But an instructor is not, as I understand it, encouraged to emphasise the dangers. I wonder if it would be a good idea to have, somewhere fairly early in the course, a doom and gloom session to bring to notice the importance of reporting any symptoms as early as possible.

Des Gorman

I think that doom and gloom is, unfortunately, why people present late. We all have a concept of culpability which runs through most things we do. We do not understand risk but we do understand blame. A classic example of an Antipodean risk assessment is the young bloke who goes to a party, meets a girl he does not know, decides not to have sex with her for fear of getting AIDS, and then drives home pissed.

Our concepts of risks are related to the outcome. A social acceptability outcome, rather than real level of risk. If we tried to set up a dive shop marketing a risk-related approach to diving, we would not make money. The reason why people go along to the diving schools to learn to dive is because they market safe diving, and that is the only commercially viable form of diving education. Safe diving is what the community wants and what the community demands. Unfortunately, the minute one says to somebody "this is safe diving", one is saying that any adverse outcome is the product of unsafe diving. The end result is that culpability is established at the core of decompression illness. I suspect the doom and gloom link just reinforces that.

The only way, I believe, is to try to shift education to a risk-related basis. But I do not think it will work. Divers are looking for safety. I do not think they would understand a risk-related approach, even if one tried to teach it. The average person is not receptive to that level of sophistication in teaching. My fear about doom and gloom is that it continues to reinforce the false culpability axis which I believe is a major player, but not the only player, in delayed treatment. One of the reasons why people show up with an amazing series of explanations, "I twisted my knee on this", "I hurt my shoulder on that", "I often have a sore back", "It's not uncommon for me not to be able to pee for three days", is rationalising away what they see as being some admission of fault or breaking of some sacred writs.

So I have some misgivings about doom and gloom, and I do not believe that any dive training organisation would be able to shift away from the concept of safe diving. Because it is what the market demands.

James Francis

I would put a slightly different slant on that. Doom and gloom, I agree, is not the way to approach the problem. But I think it is worthwhile taking a pragmatic approach and telling people that, as we all know, diving problems happen. And if it does happen, it is a good idea to tell somebody fairly quickly. I think one can get around this problem of blame by saying "as you all know people get bent regardless of whether they are inside the tables or out". In the Royal Navy divers are taught from a very start that they should report any abnormal symptoms. That is, I think, one of the reasons why military divers do so well from treatment, because, generally speaking, they report early. They are not blamed if they report an illness. One of the reasons is the way the military diving is controlled; if anyone is to blame it will be the dive supervisors, because they have a pretty rigid control over the dive profile which is actually dived. So if the divers do develop DCI they do report it early, and generally they respond well to treatment. If that sort of ethic or culture could be inculcated in the sports diver community, I think we would be on a better wicket than the way things are currently done.

Simon Mitchell

We, in the Royal New Zealand Navy (RNZN), have a policy of taking people deeper after inadequate response, first at 18 m of seawater and then at 30 m. We have seen a lot of cases where inadequate response at 18 m is reversed at 30, or even deeper at 50 m. We have not published that data. The data from the last 3 or 4 years is really the property of another researcher at our unit, and is not something I have looked at writing up. However, the lignocaine trial where we have a similar recompression algorithm, will be written up and published.

We have had three Royal Australian Navy Medical Officers, over the last 5 years, who have arrived with the mindset that one should not go deeper than 18 m under any circumstances or that it is very unusual to do so. All of them left as a proponent of going deeper where there is inadequate response initially at 18 m, because we see it so often. However I have no figures to present to you.

Mike Bennett

There is some data suggesting that people get short

term improvement, but that still does not answer the question of whether there is any difference in ultimate outcome. Extended and/or repeated oxygen treatment at 18 m may produce the same final result in the end, it is just not presented in the same way, as resolution of symptoms on a deeper compression.

Simon Mitchell

I am sure that is true. The problem is one of risk benefit, a similar situation which applies to saturation tables. Richard has recommended that the use of saturation tables should be limited on the basis that they are difficult to do and they present hazards all of their own. Our contention is that the deeper treatments at 30 m and 50 m involve relatively small increases in risk to attendants and in logistical difficulties. On top of that, if it is something one can do early that produces an apparent benefit, then one probably should do it. But I have not got the long term outcome data that would demonstrate a benefit overall.

Richard Moon

Can I again speak to the helium issue? The idea of using heliox as part of a recompression has been around now for 10 or 15 years, and slowly but surely, based on anecdotal reports and personal experience, it is becoming the de facto standard of care, unfortunately, I believe, without the necessary data. It may well be correct that helium-oxygen is more efficacious than oxygen or nitrox, but I think the danger of accepting this notion without the proper data is that a tremendous expense to chambers around the world would be incurred as a result of having to install the necessary capability.

Let me give you a example of the danger of basing ideas on personal anecdotal experience. I have seen a number of severe neurological bends referred to our medical centre after having received a treatment somewhere else, which either did nothing or actually appeared to make the patient worse. Those patients uniformly responded to recompression at our medical centre. I do not really understand why, it must have something to do with the natural history of the disease, but treatment a day later often is more efficacious than the initial treatment.

If we had used helium-oxygen for those second treatments, we would be enthusiastically touting helium-oxygen. So, we must keep an open mind on heliox, but not accept it until we the necessary observations have been made.

Des Gorman

In response to Michael's comments about repeated oxygen treatments, I agree with Simon's stance. I believe that it is inappropriate to wait until tomorrow to try and resolve neurological disease if one has access to treatments that can turn it off today. I think one should pursue recovery vigorously as soon as one possibly can. The idea that one gives them something this afternoon on the basis that we can treat them tomorrow is inappropriate for a young person with a neurological lesion. I think we should be aggressive and try and control the disease as quickly as you can.

An issue that I forgot to mention with the helium study, and one of the things I think we need to address in our lignocaine study and elsewhere, is what is the incidence of long term depression and psychometric anomalies in people admitted to hospital for a broken leg, an abdominal crisis, or after road traffic accident. In other words, are we measuring the effects of hospitalisation per se rather than the results of DCI?. It worries me enormously when I see 40% of our patient load suffering from depression at one month, which is the sort of figures that one sees if one takes the trouble to talk to them. We need to know how many people admitted to hospital for any reason will have similar levels of depression which affect their psychometric performance a month later. I think we need to introduce another control group of people who are age, sex, alcohol and drug matched, who have not been diving at all, but have been admitted for some non-traumatic, preferably non-head injured, reason and discover what is the prevalence of psychometric anomalies and mood anomalies in people admitted to hospital. Looking at studies of CO poisoning, in particular, and decompression illness as well, I believe we are now measuring what may well be the result of a lifetime of stress, and nothing to do with the effects of bubbles. We need to know what the effect is on young people who have a traumatic admission to hospital. Simon and I intend adding such a control group.

Mike Davis

We now have heliox in our chamber. It was not very expensive to put in. The gases are much more expensive than using oxygen, there is not much doubt about that, but the amount of gas one uses with a built in breathing system (BIBS), with a demand supply, for a single patient in one treatment is not particularly great. The table that the RNZN is using, which we have adopted, is shorter than extending a Table 6 and so one saves on costs of staffing. The RNZN table does not require an enormous initial capital cost. It cost us about $NZ 5,000, and we may save a little bit on the treatment compared with an extended table. Cost is perhaps not a concern in that approach, as opposed to saturation therapies, which clearly are not cost effective in terms of the enormous outlay.

I will aslo comment on the "second treatment effect", when commonly one appears to get a significant improvement with the second oxygen treatment. For quite a few years in our old unit in Christchurch, we tended to give divers a Table 6, as their second treatment. In recent times we have adopted the Behnke 18:60:30 table as our routine follow up and I have got my doubts about that. It has been nagging me that perhaps we should go back to doing a Table 6 as the second treatment in any patient who requires more than one treatment and keeping the shorter oxygen treatments for later. I would like to hear the panel's

comments.

James Francis

I agree with your sentiments. The problem is the lack of any properly controlled trials. That is the only way these issues will be resolved and, until the trials are done, it is one opinion against another. It does not matter how many animal experiments one can quote on one side of the argument or the other. There are tremendous difficulties in extrapolating the findings from animal experiments to the human disease. Not just in measuring endpoints, but indeed in the nature of the disease. I am particularly critical of people who extrapolate experiments on small rodents to the human species. There are a few rats amongst us humans, but not enough to make any such trials appropriate.

I think that one of the most exciting things we have heard this week so far was from Simon yesterday.[3] If one is going to try to do controlled trials, one has got to have a sound means of assessing outcome. I wish Simon all success with the model he has come up with. If it does prove to be a reliable, and above all, reproducible measure of assessing individuals, then for the first time we have a got a fighting chance of doing some properly controlled trials. Until they are done we could have this meeting again in another five years and we would reach a similar consensus, which is basically, to say "I do not know".

Des Gorman

Let us design that study though. We could do a study to look at retreatments for patients. How many centres would one need, over what period of time, to achieve a power for a comparative group on follow up treatments? We would have to have virtually every hyperbaric unit around the world, which treats divers, using the same protocol, to acquire, over a 5 years period, enough subjects of homogeneity to make some sensible power study. That is the problem. Just think about the problems and complexities of getting comparative data for follow up treatments. My concerns are the length of time, the number of units and the number of subjects. One needs to study follow up treatments, looking at different options and at homogeneity of the population. It would be a hideous project. James, you can do that one.

I think there are some far more fundamental issues about acute presentation.

James Francis

I do not think one necessarily has to test every single table. One could certainly make a start by looking at, say, with initial treatments, deep versus shallow. One might, after that, do a second study looking at oxygen tables versus heliox tables. As far as retreatment is concerned, one can group them into short tables for retreatment versus longer ones. If one can start to find answers to the broader questions, then one may be in a position to start refining it to specific tables. One needs to decide which is the most important question and look at that one first. Probably the most important question is do you go deep, or do you go reasonably shallow for your first treatment? I have no idea of the answer.

Des Gorman

I agree that one has to choose the right question. But we have a considerable advantage, because we have two relatively large groups of patients, in Australia and New Zealand, who do not become completely well. Having a lot of treatment failures actually makes research easier. With a group in which about 30% have significantly less than complete relief, bringing a practical improvement in that rate, down to 15% say, will allow one to use a much smaller group of people.

David Griffiths

My experience in Queensland is that the majority of people who present to diving instructors after diving are tourists. A few of them are just sent away and told that they have nothing to worry about. The majority are taken seriously and are treated with surface oxygen. Some of these people are a day or so out from port. There is a debate about whether they should stay on surface oxygen and come in on the boat within 12 hours, or whether they should be evacuated by helicopter. Can the panel give advice about who should be helicoptered in? The worrying group is those who, when they have had surface oxygen, feel so much better that they do not come to the chamber at all. Following that group will be difficult. Are they exposing themselves to risk of dysbaric osteonecrosis, or other problems?

David Youngblood

I cannot answer about dysbaric osteonecrosis. There is very little information available on surface oxygen, except in the realm of altitude decompression sickness. The US Air Force, and probably other air forces as well, use surface oxygen as a definitive treatment for altitude DCI when there is complete relief and there were no neurological symptoms. I think it is an open question for the treatment of decompression sickness in divers. Is there any circumstance in which surface oxygen could be the definitive treatment? I think it bears looking at, particularly with minor pain or perhaps even sensory symptoms. However, I think we have all seen cases in which there was an apparent response to surface oxygen and then deterioration once the oxygen was discontinued. So, I think if one wants to incorporate a surface oxygen paradigm into the treatment of bends, it has to make allowance for the problem, what does one do with the patient who gets worse once the oxygen is stopped?

Simon Mitchell

My belief is that one should see them all. We have often had someone ring in with apparently trivial symptoms

and, when they arrive at our unit and we examine them, or get a decent history, we find that the situation is a lot worse than it was first portrayed to us. And we think "Thank goodness we encouraged this person to come", because it is actually quite significant DCI. We see this time after time. I think it is part of the denial response. When people ring up, they are in a situation where it is going to inconvenience others to be evacuated, so they tend to play it down.

James Francis

The only data I am aware of on dysbaric osteonecrosis relates to naval divers. It is the Harrison and Elliott paper of some years ago now, where they looked at 88 cases of dysbaric osteonecrosis.[14] They found that not all of these cases had been bent in the past and, where they had had limb pain, it did not necessarily correlate with the site of their dysbaric osteonecrosis. I think similar findings came out of the Newcastle-on-Tyne registry. As yet we are not aware of an epidemic of dysbaric osteonecrosis in the recreational diving community, so I think that is probably a bit of a red herring at the moment.

Ian Miller

I have an anecdote, a comment and a question.

In Melbourne we have certainly had several patients who had a high cortical function loss, lethargy, not performing well, who have presented 3 to 4 weeks after onset and responded to hyperbaric treatment. We have also had several cases who have had significant higher brain dysfunction, the improvement in which has plateaued out and they have been left with quite severe disease after 8 to 10 treatments using 18 m tables. We elected to try a Comex 30 with heliox on one diver and an 18 m using heliox on another, and in both saw quite dramatic response. That was 10 days to two weeks after the initial presentation.

I think that says something interesting about the variability of the disease, which is my comment with regard to any trials. I think we need to be very careful when we are designing trials, that we recognise that it is a very variable disease and, if we aim to test just a single therapy across the board, or a single approach, we run the risk of losing significant results in a lot of noise. The therapeutic approach may be the right one for a particular subgroup of decompression cases, but may offer no benefit or may indeed be a negative factor for another group. Inevitably we need to be looking for a treatment strategy which is matched to the severity of the disease.

What are the Panel's thoughts on whether we maybe introducing a negative factor into patients by giving them too much oxygen? Whether there may be an element of oxygen toxicity or of oxygen exacerbation of inflammatory or liver peroxidation processes going on, and whether that may be something we need to consider.

Des Gorman

Just briefly Ian, I think the anecdotes you describe are the reason why any clinical trial of decompression illness needs a placebo and a non-diving control group.

The other point, made about oxygen on the surface. If we are going to trial oxygen-helium, that is where we should be trialling it, as a first aid gas. I would recommend a trial of oxygen and heliox as first aid treatment.

About oxygen toxicity. There is no substantive data at present. The reality is Table 5 or 6 produce recovery in 95% of patients, especially if treated early in the Naval context.[14] The question one should ask is, whether that is the appropriate dose of oxygen for someone turning up 2 to 3 days later. That is the heterogeneity I am talking about. I am not arguing that we should not do trials, I am just saying that we need to be reasonably modest in our ambitions, because the potential heterogeneity is so great.

I think the simple answer to oxygen is, that the majority of people who are treated with it early get dramatically better. I have got no idea why. It is a poisonous dose of oxygen we use, there is no question of that. But the clinical response is that they get carried in and walk out. We have to have faith in what we see and not worry too much about problems. When we are looking at outcomes, we have to be very careful not to be confounded by placebo and hysteria and the effect of depression induced by hospitalisation.

David Youngblood

I thought this might be an appropriate time to drop a challenge to this group, and hopefully to a potential multi-national group. Last year you heard about the lobster divers on the Mosquito Coast of Honduras, who are relatively advanced.[15] I have just got back from Nicaragua and there is a very unusual situation there. God's great experiment, I call it. There are 54 boats with a minimum of 30 divers on each, that is roughly 1,500 divers. They are, genetically, almost all the same. They all dive the same profiles. For the first time in diving history we have a denominator. They get absolutely no treatment, so we do not have anything to be confused about and it would be the first aid intervention. We could put teams on various boats at the same time. The season runs from December through to the end of March, although it is a bit rugged to do it. They all dive to 27 m (90 ft) to 36 m (120 ft), they dive all day, they use 16 to 20 tanks a day and they have huge omitted decompression debts. We have just finished installing a chamber in Nicaragua. We need to get there and measure what is happening first and then carefully plan some interventions and see what happens.

Des Gorman

I would like to ask Peter Robinson whether as a health funder, is he worried about the possible shift to more expensive treatments for decompression sickness?

Peter Robinson

I think the short answer is that anything that is going to increase ACC spending is going to be of some concern, if not to me personally, then certainly to the new Minister for ACC. Diving accidents last year consumed about one million dollars. It is the second highest cost group per claim that we have, behind skiing, if you look at recreational issues. In the overall scheme of things, a million out of 1.6 billion budget of outgoing is pretty minimal.

I think that we certainly would want some sort of cost containment, because we do not have a direct levy to pinch money off the people who damage themselves and then consume our resources. New Zealanders will have noticed in the press recently that we have been talking of insurance excesses, so that people become responsible for the first $400. That will be $400 for treatment which comes from the patient. There is also the question of whether we should be levying sports clubs. That is such a bureaucratic and administrative nightmare that I do not think it will happen in the next decade.

Certainly we want efficacy of intervention, and I think one of the first things that will impact upon the people treating divers will be that the contracts will start asking how you assess that one has done a good job. To say "I thought we did well, because it seemed to work last year" is not, I think, a viable funding option for the future.

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PFO AND DECOMPRESSION ILLNESS: AN UPDATE

Richard Moon and Joseph Kisslo

Key Words

Bubbles, cardiovascular, decompression illness, medical conditions and problems.

Introduction

It has been known for many years that blood clots originating in the leg veins can pass from the right to the left sides of the heart via a patent foramen ovale (PFO), resulting in paradoxical thromboembolism. When it occurs it usually manifests as stroke. A clot passing through a PFO is a rare event, and it has traditionally been made at post mortem examination. However, the availability of techniques to detect PFO in live people, in recent years there has been a considerable amount on interest in the role PFO might play in otherwise inexplicable diseases. In 1988 Lechat reported a group of young individuals who had what appeared to be embolic stroke with no other risk factors and found that 40% of these individuals had a PFO demonstrable using bubble contrast echocardiography. The usual prevalence of PFO in the normal population is around 20%, suggesting that embolism through the PFO was the explanation.[1]

The left atrial pressure is higher than the right and the design of the inter atrial septum is a flap valve mechanism. Even with a patent foramen ovale, the doorway, the flap valve should be closed by the normal inter-atrial pressure gradient. How, then, could a PFO result in shunting from the right side to the left? When the left atrial and the right atrial waveforms are examined in detail, there is a small portion of the cardiac cycle in which right atrial pressure actually exceeds the left, during which blood and other materials such as clots could be shunted from the right to the left side of the heart.

Why does this have any importance for divers? After a dive one can demonstrate in some divers, by Doppler techniques, intravascular bubbles on the right side of the heart (venous gas embolism, VGE).[2] Dick Dunford of the Virginia Mason Institute in Seattle has demonstrated that during a week of diving, VGE may exist at some time in virtually every individual studied.[3]

That being the case, if someone does have an inter-atrial communication, the normal filtering ability of the lung may be bypassed, which would allow bubbles to travel from the right atrium into the left, and may then cause arterial occlusion in the central nervous system, or localised activation of a mediator, such as complement. A case report suggested that right-to-left shunting of bubbles through an atrial septal defect (ASD) might precipitate DCI.[4]

After deciding to investigate PFO as a possible risk factor for decompression illness (DCI), our first case was a man who had been diving off the North Carolina coast with his girl friend. After a dive the two of them were driving back to their hotel when he suddenly realised that he did not know the way. A couple of minutes later, as related by the girl friend, he looked quizzically at her and stated that he did not recognise her. Feeling rather disconcerted by this she took him to the hospital. After evacuation and evaluation at Duke Hospital it was evident that he was profoundly abnormal neurologically. He was confused, and had a rash, which he said had occurred after a dive several weeks before, at which time he had also been confused. His MRI showed numerous white spots in the sub cortex and he also had a PFO.

Examination for a patent foramen ovale is quite straightforward. The easiest method is to use transthoracic echocardiography (TTE) and inject a suspension of microbubbles. To make the suspension one can put a three way stopcock into an intravenous line, attach two syringes with a small quantity of air and 5 ml of saline in each and then rapidly flush the solution back and forth between syringes until it goes milky. The bubble suspension is then rapidly injected into a peripheral vein via an indwelling catheter. A few drops of the subject's blood will stabilise the bubbles and permit a better study. A few seconds after injection a cloud of bubbles will be observed traversing the right side of the heart. In the presence of a PFO bubbles will be observed also in the left heart (see Fig. 1). Alternatively, one can use commercially available, stabilised bubbles. The routine is to try it once while the individual is resting comfortably, and if there is no demonstrable shunt, then have the patient perform a Valsalva manoeuvre, injecting the contrast during the release phase. This can demonstrate a shunt which is not visible during normal resting breathing.

There are other ways of doing it, such as using transcranial doppler rather than 2-D echocardiography to detect the bubbles. With this technique, by applying a probe to the head in the appropriate orientation, one can examine the intracranial arteries and observe a pulsatile flow wave, usually in the middle cerebral artery. If one then injects bubbles as described bubbles traversing a PFO can be observed as aberrant spikes in the ultrasound waveform. Others have used transoesophageal echo (TEE), which provides clearer images than transthoracic imaging, and a few additional instances of PFO can be detected using this technique. However, as will be demonstrated below, the minimal right-to-left flow through a PFO which can be demonstrated exclusively with TEE is probably of minor consequence with regard to DCI risk.

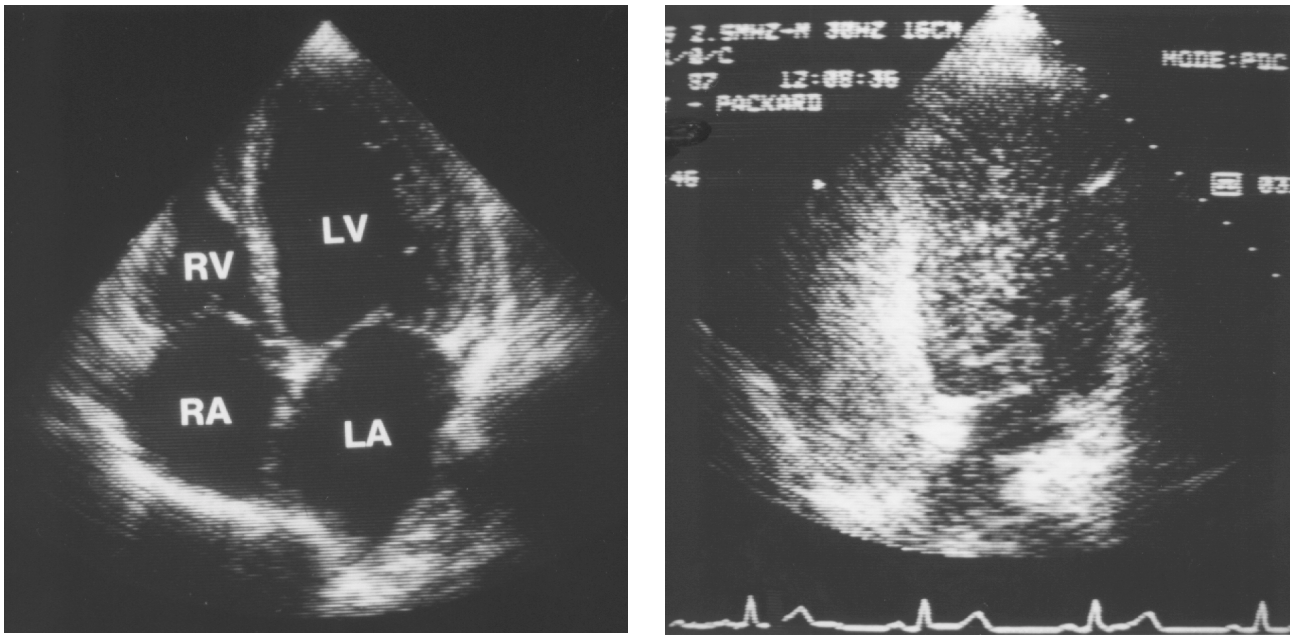

Figure 1. Transthoracic bubble contrast echocardiograph images. On the left, pre-injection, with cardiac chambers labelled. On the right after intravenous injection of bubble suspension in a 48 year old male diver who, a few minutes after a 30 metre 17 minute dive, developed back and epigastric pain, dyspnoea, leg weakness and numbness. He had paraparesis, urinary retention and a T12 sensory level. Bubbles can easily be observed in the left atrium and ventricle.

Findings in divers

In order to examine this problem systematically we identified a group of divers who had varying degrees of predominantly neurological bends. We arbitrarily defined these as serious (cerebral, vestibular or motor weakness), or mild, which included pain, with or without paraesthesia or hypaesthesia. This was simply an operational definition, and was not intended to imply that the latter category is less important than the former.

A total of 91 divers who had had decompression illness and 100 volunteers were studied.[5] Eleven percent of the volunteers had right-to-left shunt during spontaneous breathing and an additional 9% shunted after Valsalva, for a total of 20%. Of the divers with decompression illness, 32% shunted at rest and a total of 43.2% (including those who shunted during resting breathing) shunted after Valsalva manoeuvre. Of the 57 serious cases, as defined above 39% shunted during resting breathing and a total of 47% shunted after Valsalva manoeuvre. There were 31 non-serious cases of which six (19.4%) shunted during spontaneous breathing and a total of 11 (35.5%) shunted after Valsalva.

All subjects underwent colour flow doppler evaluation prior to bubble contrast injection, and few inter-atrial shunts were detectable, confirming the lack of sensitivity of this technique for the detection of PFO.

We studied onset latency and found that the odds ratio was statistically different from 1 in those bends with onset less than 10 minutes and 10-60 minutes after surfacing, but not for those with longer latency onset. I am uncertain as to the significance of this because there is a strong relationship between the severity of DCI and its onset time: serious cases tend to have a shorter time between surfacing and the onset of symptoms. The statistical significance of latency could be because of this correlation.

A similar relationship has been found by Peter Wilmshurst of the UK.[6] He found that 24% of normal divers had right-to-left shunt through a PFO, compared with 65% of those with early onset neurological bends. The prevalence of PFO in divers with late onset bends was not different from control values.

Patent foramen ovale therefore appears to be associated with serious neurological bends (Duke study) and early onset neurological bends (Wilmshurst study) and, at least in our study, there was a relationship between the degree of shunt (resting vs. Valsalva-induced shunt). The reason for this relationship remains an open question. I believe the most tenable hypothesis is that VGE, which would otherwise be filtered by the pulmonary capillaries, may become arterialised in the presence of a PFO. However, there are other hypotheses. It is conceivable that the presence of a PFO is linked genetically to an unrelated factor which predisposes to DCI. In other words the presence of a PFO may be merely a marker for the "real" predisposition, in the same way that xanthomata are not the cause of coronary artery disease, but external markers for the underlying predisposing condition,

hypercholesterolaemia. Another possibility is that DCI induces PFO, or enlarges it. A possible mechanism for this might be right atrial hypertension. However, the only form of DCI in which this is likely to occur is massive venous gas embolism causing pulmonary hypertension and secondary right heart failure, an extremely rare but easily recognisable event. There were no such cases in our series of DCI in which PFO was examined. Therefore I believe that is unlikely.

While surgical correction of a PFO in order to correct one's risk of DCI would be considered too radical by most diving consultants, recent development of techniques to correct cardiac septal defects may have changed the picture. Some years ago, one of our commercial divers with a PFO underwent placement of one the first transvenous occlusion devices. After ascertaining that his neurological exam was normal, and, using bubble contrast echocardiography, that there was no residual right-to-left shunt, we cleared him to return to diving. This technique has been published by Peter Wilmshurst recently in the British Medical Journal.[6]

As a follow-up study we were interested in what would happen to right-to-left shunts when immersed. Divers, particularly professional divers, may spend significant periods of time decompressing in the water. We wanted to know what happened if they were experiencing VGE while they decompressed. We hypothesised that, because of translocation of 500-800 ml of blood from the legs into the thorax (causing an increase in cardiac volume),[8,9] immersion would increase right-to-left shunt through a PFO.

We studied 11 individuals, all of who had a PFO demonstrated by bubble contrast echocardiography, under rest and exercise conditions in the dry and immersed to the neck in water. We measured at end-diastolic and end-systolic left ventricular diameter under resting and increasing exercise conditions and in the supine position. Exercise studies were performed in the dry or immersed to the neck in thermoneutral (35°C) water. Upon immersion there was a significant increase in left ventricular end-diastolic and end-systolic volumes, exactly as one would expect. We used a semi-quantitative measure of the degree of shunt after bubble contrast injection, as follows: "0" represented no right-to-left shunt, "1" represented partial opacification of the left side of the heart and "2" represented total opacification. During a separate sitting we performed the same manoeuvres after placing arterial and pulmonary artery catheters in the same volunteers, and used using the technique of multiple inert gas elimination[10,11] to assess right-to-left shunt (which in this case would include both intracardiac and intrapulmonary shunt). Using either technique, there was no effect of immersion upon the degree of shunt.[12,13] Within the limits of this relatively small study, it appears that neither immersion in water nor supine position increases right-to-left shunt through a PFO.

A recent study

A recent article from the British Medical Journal has created a stir within the recreational diving community in the United States.[14] These investigators examined 87 dive club volunteers, each of whom had made more than 160 recreational scuba dives. Using a 1.5 Tesla scanner each volunteer underwent MRI of the brain, and scans were examined for the presence of subcortical areas of high T2 intensity. In order to detect right-to-left shunt through a PFO, transcranial Doppler, after intravenous injection of bubble contrast, either with or without a Valsalva manoeuvre was used. They diagnosed a right-to-left shunt (RLS) when there were more than 5 bubbles in either middle cerebral artery. Table 1 shows the patient group. Twenty-five individuals (28.7%) had a right-to-left shunt, 62 (71.3%) did not have a shunt, approximately the proportions that one might expect in the normal population. Heights, weights, ages and diving exposures were similar. Cigarette smoking was a little heavier in the group without shunt, while the self-reported amount of alcohol consumed was the same in each group.

TABLE 1

PATIENT DEMOGRAPHICS (Knauth[14]**)**

	RLS*	**No RLS**
Number	25	62
Weight (kg)	69.9	80.0
Height (cm)	174	177
Age (years)	35.4	35.9
Total dives	574	562
Decompression stop dives	89	100
Cigarette smoking (pack-years)	1.8	5.2
Alcohol intake (g/day)	30.9	33.1

* RLS = Left to right shunt

They further classified these shunts as either low or high haemodynamic significance based upon an arbitrary score of either less than 20 bubbles or more than 20 bubbles (Table 2).

TABLE 2

HIGH (HHS) AND LOW (LHS) HAEMODYNAMIC SIGNIFICANCE RIGHT TO LEFT SHUNTS
(From Knauth[14])

Lesions	**HHS**	**LHS**	**No RLS**	**Total**
0	10	11	55	76
1	0	1	7	8
5	1	0	0	1
12	1	0	0	1
16	1	0	0	1
Total	13	12	62	87

When one examines the "0" or "1" lesion group, there is actually no relationship between the existence of a lesion and right-to-left shunt. However, there were three individuals, with 5, 12 and 16 brain lesions, respectively, each of whom had a right-to-left shunt of high haemodynamic significance. The original data can be summarised in Table 3.

TABLE 3

SUMMARY OF RESULTS (Knauth[14]**)**

Lesions	HHS	LHS	No RLS	Total
0	10	11	55	66
1	0	1	7	8
> 4	3	0	0	3
Total13	12	62	87	

HHS High haemodynamic significance RLS
LHS Low haemodynamic significance RLS
RLS Right to left shunt

The authors concluded that right-to-left shunt through a PFO in divers is a risk factor for the development of brain lesions visible on MRI.

There are several reasons why this study cannot be accepted at face value. First, although all of these individuals were divers, they had no non-diving control group, so there was actually no evidence that even if there is a relationship between PFO and brain lesions, it has anything to do with diving. An alternative hypothesis to explain the data is that the lung may be important in breaking down metabolic compounds that may produce MRI lesions. The lesions could also have been due to subclinical thromboembolism. PFO has already been demonstrated to be a risk factor for stroke, presumably by allowing small venous clots to traverse the inter-atrial septum.[15,16] Second, the described relationship depended in this study only on three individuals suggesting an apparent relationship where there may not be one. Finally, the clinical significance of these brain lesions is speculative, and the authors presented no functional data (e.g. psychometric testing) with which to demonstrate clinical relevance. Therefore, although further studies may be warranted, to conclude that PFO is a risk factor for subliminal brain damage in divers is unwarranted.

Conclusions

To summarise, the evidence suggests that the risk of serious neurological DCI or early onset DCI is increased in divers with a resting right-to-left shunt through a PFO. There is, at present, no evidence that PFO is related to mild or late onset bends. This issue raises several questions:

Should all recreational divers be screened for PFO?

No. Even with a PFO the probability of DCI is low, especially those types of DCI that are associated with PFO. A case could be made that screening is appropriate for divers whose work experience is likely to subject them to VGE for prolonged periods (e.g. saturation divers).

If a diver experiences bends frequently and appears to be predisposed to DCI should a bubble echo study be done?

No. If a diver has an intrinsic susceptibility to bends, identification of just one of many possible risk factors (most of which are probably as yet undiscovered) is not useful unless surgical correction is contemplated (e.g. of an ASD).

Should a person with a known intracardiac shunt ever dive?

Because of the extremely high probability of cerebral gas embolism, a person with any significant right-to-left shunt (e.g. Tetralogy of Fallot) should never scuba dive. Because of the small pressure difference between the right and left atria, and the potential for reversal of the usual left-to-right shunt, people with atrial septal defects should also not dive.[4]

In the presence of a PFO, the advice I usually give depends upon the degree to which the individual is risk averse. The most conservative advice is not to dive. The liberal approach states that even if the probability of experiencing serious neurological bends is five fold higher than a person without a PFO and five times a small risk is still small. A middle philosophy is to minimise the probability of VGE, for example by using bottom times that are at most one half of the USN air diving no-stop times.[2]

For individuals with ventricular septal defects (VSD), provided the shunt is unidirectional, left-to-right and not haemodynamically significant, small changes in intracardiac pressures induced by respiratory manoeuvres will not significantly affect the large pressure gradient between the ventricles. Therefore it is extremely unlikely that VGE could enter the left side of the heart via a VSD, and such a condition should not preclude diving.

Questions

Unidentified speaker

In 1991 there was a study from Norway on 120 professional divers who had been examined with MRI, demonstrating that there were not any more bright spots in the MRI scans of professional divers than in a control group.[17]

Secondly in the professional divers the number of bright spots seemed to decrease as the diving career increased. This may of course be the healthy diver effect, that most injured divers leave their job, but it did not look like that because the same divers were used for examination of neurological symptoms and a number of

them had neurological symptoms. Anyway, there was no clear relationship between diving and neurological symptoms from diving and the bright spots.

Reply

That is exactly right. In fact, in that study, the number of white spots was less in the divers than it was in the controls, who were policemen.

Unidentified speaker

Should all prospective divers have a thorough cardiac examination, and have the physician listen carefully for murmurs?

Richard Moon

I believe the answer to your question depends upon the type of diving. For recreational divers I believe that there is no need for a screening examination to look for patent foramen ovale. The only relationship that we have found between PFO and DCI is for serious neurological bends, a rare disorder, and largely attributable to risk factors which are associated with the dive itself, such as depth, bottom time and rate of ascent. On the other hand, for a person who plans to perform dives that have a high risk of venous gas embolism for long periods of time, for example saturation diving, then I would recommend a PFO study.

The method of looking for a PFO must be a bubble contrast echo. Colour flow doppler is insufficiently sensitive. It is impossible to detect inter-atrial shunts on physical examination unless there is a frank atrial septal defect, which produces a fixed split of the second heart sound, or a pulmonic valve systolic flow murmur. These physical signs therefore cannot be used as a method of screening for PFO. However, if there are physical signs of an ASD, the diver needs to be examined more thoroughly using echocardiography.

Unidentified speaker

Two comments. I would like to emphasise that in the Norwegian study, it is my understanding that those controls as you mentioned were policemen and given the combative nature of that work, that may not have been a wise control group to use, as far as head injuries are concerned. But the second thing is I have been desperately seeking some reassurance that the test is not worse than the disease, particularly after hearing Des' work and being familiar with Brian Hills' work. Do we have any assurance that putting these saline bubbles thorough the brain and elsewhere does no harm?

Richard Moon

It appears that in the absence of a pre-existing inert gas load, the transient gas embolism of the degree which is engendered by this test is fairly harmless. In our series of about 170 people on whom we did this test, two experienced transient paraesthesias, but neither had a right-to-left shunt. The general opinion among cardiologists is that it is a safe procedure that does not result in any serious morbidity.

Ian Millar

Any comments on the comparability or preference for testing techniques, given that there were two clearly techniques, one looking at the heart specifically, one sampling the end target organ, and yours using agitated saline, versus recent studies which as I understand it used a contrast medium which has contrast microbubbles in it, which would be significantly smaller than the saline bubbles.

Richard Moon

The sensitivity of the two techniques in detecting PFO in normal subjects appears to be similar. Transoesophageal echo (TEE) has clearer images than transthoracic echo, but it is a little less popular with divers because swallowing the probe is extremely uncomfortable. It has a higher sensitivity,[18,19] presumably because of the increased clarity of the images. There are, at present, no data showing that PFOs seen with TEE that cannot be detected using TTE represent a risk factor for DCI. I feel that such PFOs are probably small and of minimal significance in the pathophysiology of decompression illness.

Paul Langton

Most of the studies that have looked in the neurological series where they have had lots of cases and compared transthoracic and transoesophageal, certainly do find a higher detection rate of PFO from transoesophageal, but at complete loss of specificity. They are detecting lots of small lesions that they can detect in control subjects as well, so the specificity goes out the window with the alleged improved sensitivity.

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A CASE OF RECURRENT DECOMPRESSION ILLNESS

Peter Chapman-Smith

Key Words

Case report, decompression illness, sequelae, treatment.

General practitioners see their patients repeatedly. This puts them in an excellent position for follow up studies on divers who have suffered decompression illness (DCI) to discover what the usual clinical progress is likely to be. Very little has been published about the long term follow up of divers. Case 1 is from my records. Follow up of divers suffering decompression illness treated with recompression is often revealing.

Case 1

A 50 year old mechanic has been diagnosed as DCI on 4 separate occasions. His only other disability has been symptoms of carpal tunnel syndrome. He was treated at the Royal New Zealand Navy (RNZN) Hospital on 3 occasions between 1988 and 1996. He has suffered from a series of subtle but significant disabilities for years.

December 1988

After an evening of moderate to heavy alcohol consumption he did a single dive to 21 m (70 ft) for 60 mins. He ran out of air and made a rapid ascent. 24 hours later he consulted me complaining of skin itch, pain in his hands and feeling very tired and light headed. He had pain at the base of his spine and in the buttocks.

Physical examination was neurologically normal, except for a sharpened Romberg Test (SRT) of 25 seconds. He was slow counting down from 100 by sevens. The audiogram showed a mild high frequency loss R>L.

He was transferred to the RNZN recompression chamber (RCC) at the Naval Hospital in Auckland, about 150 km, where he needed three treatments before his

symptoms resolved. Twenty four hours after his third treatment he still had no symptoms. The discharge diagnosis was Type I DCS with skin and joint involvement.

Unfortunately skin itch, anterior chest tightness and tiredness returned two days later. He was unwilling to go for further treatment. He was advised not to dive for 2-3 months.

He then did 10 uneventful dives before the next problem.

January 1990

He did a single dive, for crayfish, to 21-24 m (70-80 ft) for 35 minutes with a 3 minute stop at 4.5 m (15 ft).

Several hours later he developed a generalised skin itch. Three days later he woke with a dull ache (pain) in his left hip. He developed pain in the left shoulder, left elbow, left wrist and in the knuckles of his left hand later the same day. No joint swelling was noted.

Next day he had reduced sensation over the lateral side the left lower leg and dorsum of the left foot. These changes were only noticed by chance. When there had been no change after 36 hours he contacted the RNZN Hospital directly.

He arrived at the Naval Hospital 6 days after the dive. There had been some improvement in his symptoms and the area of numbness was now hyperaesthestic. No urinary, balance or muscle weakness problems were noted. Physical examination showed no abnormalities. Serial sevens were managed easily and the SRT was normal.

It was decided to recompress him as his symptoms were the same as at the time of his first treatment 12 months earlier. He was given a USN Table 6 with total resolution of symptoms at 18 m. He reported clearer thinking after treatment.

Two days later he telephoned me from home to report all was well.

January 1993

He did his first and only dive since January 1990 to a maximum of 18 m (60 ft) for 45 minutes, in excellent weather, after crayfish. On the boat he was exposed to diesel conditioner containing a biocide similar to nerve gas.

After an Automobile Association call out, and several beers, he developed a moderately severe headache 2-3 hours after the dive. Two days later he saw me as the symptoms felt like his previous DCI. He had slow mentation and short term memory with poor balance, so he was referred to the Naval Hospital where he was recompressed repeatedly, the treatments lasting for 2 weeks.

On discharge he was much better but still had headaches (these continued for months). He was advised not to dive again.

The discharge diagnosis was marked constitutional DCI, with psychosexual dysfunction and short term memory problems.

However he was unable to carry out his occupation, as he was unable to remember what stage he was up to when working on vehicle repairs. He lost self confidence and came under financial pressure. He had a subtle deficit, not visible to others, which was impacting on job performance. A poor prognosis was predicted. He was treated with simple analgesics.

May 1993 Follow up

He had poor balance, appearing to be drunk and staggering with minimal alcohol. His SRT was 3 seconds and he was unable to stand on either leg with his eyes closed. He also had earache (left worse than right) with constant occipital headaches and tinnitus. Memory and cognitive deficits were present. He was depressed.

August 1993 Follow up

His SRT was now normal (30 seconds). His main problems were concentration and short term memory. There wa numbness of the sole of the left foot, and especially heel which ached. His right groin had ached for 3 months but there was no hernia or mass.

September 1993 Follow up

ENT assessment: Mild high tone sensorineural deafness, consistent with noise exposure. Hearing loss and tinnitus worse especially with low background noise. His spouse complained about loud TV. Previous history of noise exposure, shooting (none for 6 years) and occupational as a mechanic.

November 1993 Follow up

The neurologist assessed him as having no neurologic abnormality (!) but depressed. Recommended referral to a psychiatrist.

February 1994 Follow up

Headaches, tinnitus, and memory problems continued. He made silly mistakes at work. Still depressed. Unsure of bladder volume when passing urine. Work output about 60%. Symptoms of peptic ulcer.

January 1995 Follow up

Neurologically normal on examination. Good balance and gait.

July 1996

He was scrubbing the bottom of a boat on snorkel became tired and completed the job using scuba. (2 dives in 3 years.) After 30 minutes at 2 m (6 ft) he dropped the scraper and descended to 6 m (20 ft) briefly to recover it. Ten days later he had an itchy back but his memory was fine.

September 1996

He came to see me for a second opinion. For years he had had pins and needles in both feet, left more than right. He also complained of a burning pain with

defecation. He had a similar pain in both ankles and heels, which were both quite numb with reduced pinprick sensation. He had a prickly sensation in his right eyebrow, both eyelids and inside his mouth. He was taking fluoxetine hydrochloride (Prozac) at night and naproxen. A trial of clonazepam (Rivotril), 0.5 mg mane, was effective within minutes of administration. On this he slept better, the pins and needles virtually went and he was happy for the first time in years.

December 1996

The Accident Compensation Commission refused to authorise any further treatment for DCI. He had constant pain in both shins, hands and feet. Feet numbness had returned. But clonazepam (Rivotril) was effective for skin itchiness. He had variable skin sensation, reduced pin prick anywhere. He still was often dropping tools, forgetting where they were. He had poor libido (marriage fine) but normal erections. Lethargy was constant but his memory was improving.

February 1997

He was still very fatigued. After 2-3 days at work he had to go home to rest. His mood was stable but he had decided to sell the business. His alcohol intake was minimal. He was on fluoxetine hydrochloride (Prozac) 20 mg nocte, clonazepam (Rivotril) 0.5 mg mane and naproxen prn, which had helped. On examination his SRT was 30 seconds and he had a shorter gait step.

April 1997

Hands were seizing up. He had to straighten his fingers out with the other hand. Dropping tools was very frequently through inattention only, a daily hassle. Naproxen was helpful, but he was fed up and the business still for sale. He could not continue with his job.

In short a poor outcome.

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OXYGEN THERAPY EQUIPMENT A THEORETICAL REVIEW

Michael Davis

Key Words

Accidents, equipment, first aid, oxygen.

Summary

The basic needs for oxygen therapy equipment are control of inspired oxygen concentration, prevention of carbon dioxide accumulation, minimal resistance to breathing, efficient and economic oxygen use, adaptability to different gas mixtures and adaptability to different modes of respiration. Understanding the performance characteristics of oxygen therapy devices enables better selection of equipment for diving accident management. Physiological studies have shown that these devices may be subdivided into *fixed performance* and *variable performance* systems. The *fixed performance* devices, when used properly, supply the predetermined oxygen concentration irrespective of the patient's ventilation characteristics. *Variable performance* devices provide variable oxygen enrichment (always less than 100%) depending on the interrelationship of oxygen flow, device factors such as functional apparatus dead space and patient factors such as the peak inspiratory flow rate. For supporting diving operations, ruggedness of construction, simplicity of design and use, ease of training and maintenance and purchase price are all of importance. The newer demand regulator and rebreather systems (both fixed performance) in robust casings are well suited to the early care of diving accidents. However, they are moderately expensive, may require considerable training and carry an obligation on the part of the user to learn, and maintain, airway management skills.

Introduction

Oxygen therapy is an important component of the early management of many medical and trauma emergencies including diving accidents. All ambulance and field medical rescue teams carry oxygen as an integral part of their equipment and there are virtually no emergency situations in medical practice in which oxygen could potentially be harmful administered in high concentrations for short periods.

Unfortunately, medical students have been taught for many years about the potential dangers of oxygen therapy in a small group of patients with chronic obstructive pulmonary disease who are dependent on an hypoxic drive for their continued spontaneous respiratory effort. This view has now been largely discredited.[1] In addition, the pulmonary toxic effects of high concentrations of oxygen,

the so-called Lorraine-Smith effect, have become well recognised and emphasised in teaching. Sadly this has sometimes been misinterpreted to mean that oxygen therapy may do more harm than good. This is an unfortunate failure to recognise that oxygen, like any pharmacological agent, has its own therapeutic range and ratio, an understanding of which is essential to its proper administration. At normal ambient pressure (one bar or less) within the first 12-24 hours of administration of 100% oxygen these issues are almost totally irrelevant to the practice of emergency medicine.

In order to administer oxygen correctly then, an understanding is required of:

a The mechanisms of uptake and delivery of oxygen in the body and the factors that alter its delivery to intracellular systems in vital organs,

b The dose dependent toxic effects of oxygen at partial pressures greater than that in room air, and

c The performance characteristics of oxygen therapy devices.

The reader is referred to recognised texts for an understanding of the first two components.[2,3] This paper is intended to provide an understanding of the function of oxygen equipment from a theoretical viewpoint, with reference on its use under field conditions at normal ambient pressure (**not under hyperbaric conditions**). For a detailed theoretical analysis and an insight into some of the original work the reader is referred to Leigh.[4] For an excellent practical review of commercially available equipment in Australasia and its use, all SPUMS members should carry their own copy of Lippmann's *Oxygen First Aid*.[5]

Basic requirements

The basic requirements for oxygen therapy equipment for field use are summarised in Table 1. Many devices on the market were not designed with these requirements in mind and may fall short of current Australian standards.[6] In addition, few users have any real understanding of whether their equipment meets these criteria nor of the principles underlying oxygen therapy.

One could argue, as has Acott,[7] that this does not matter so long as some degree of enhanced inspired oxygen is administered to the diver patient. This, however, is a nihilistic view. Awareness of the performance characteristics of various devices enables more appropriate selection and purchasing of equipment. As a minimum one must ask three questions:

1 What is the inspired oxygen fraction (F_IO_2)?
2 What is the inspired carbon dioxide fraction (F_ICO_2)?
3 How long, under normal operating conditions will the provided oxygen supply last with this device?

Correct use at sea and for diving activities requires this information.

On the basis of physiological studies, oxygen therapy devices have been classified by Leigh into *fixed performance* and *variable performance* systems with respect to the delivered oxygen concentration.[4] The *fixed performance* devices, when used properly, supply the predetermined oxygen concentration (up to almost 100%) irrespective of the characteristics of the patient's ventilation. *Variable performance* devices give more than 21% but less than 100% oxygen depending on the interrelationship of oxygen flow, device factors such as functional apparatus dead space and patient factors such as the peak inspiratory flow rate (PIFR) and the length of the expiratory pause. These factors result in both between patient and within patient variations of F_IO_2 on a breath by breath basis.

Importance of ventilatory flow and the expiratory pause

Figure 1 shows typical respiratory flow patterns for a spontaneously breathing individual. The respiratory wave-form is essentially sinusoidal with the PIFR occurring during the middle of inspiration. PIFR varies from breath to breath in an individual even when resting (Figure 1) and also from individual to individual. When respiratory rate and minute ventilation increase for any reason, exercise, pain, anxiety, pyrexia, cold, shock, etc., then PIFR increases proportionately (Figure 2). In order to provide a fixed F_IO_2 therefore, an oxygen delivery device must provide flow rates to the patient at least equal to the PIFR. If it does not do so under all normal operating conditions then increased air entrainment with consequent reduction in F_IO_2 and rebreathing leading to both reduced F_IO_2 and increased F_ICO_2 will occur, depending on the type of device being used.

In resting subjects, if a continuous oxygen flow is supplied, oxygen accumulates within the upper airway and in the volume of the apparatus during the expiratory pause. This oxygen also helps wash out carbon dioxide. Since the

TABLE 1

BASIC REQUIREMENTS FOR OXYGEN THERAPY EQUIPMENT

1	Control over oxygen percentage of the inspired gas
2	Minimal accumulation of carbon dioxide
3	Minimal resistance to breathing (both inspiratory and expiratory)
4	Efficiency and economy in the use of oxygen
5	Adaptability to different gas mixes
6	Adaptability to different modes of respiration
7	Sufficient oxygen supplies to meet field requirements

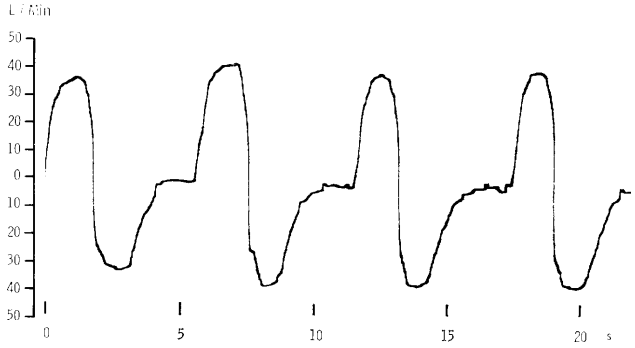

Figure 1. Inspiratory and expiratory flows measured over 20 seconds with a pneumotachograph in a resting healthy subject. Reproduced by kind permission of the author and publisher from Leigh JA, in *Scientific Foundations of Anaesthesia*.[4]

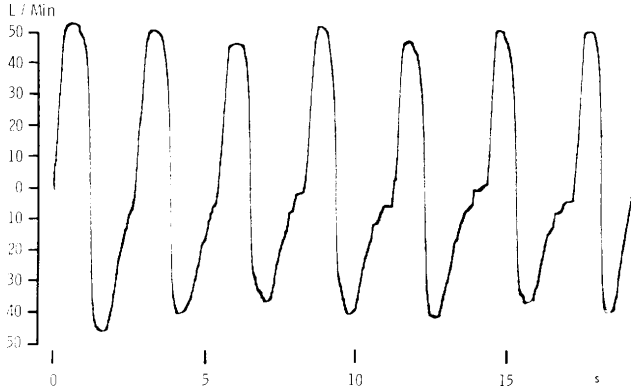

Figure 2. Pneumotachograph of the same subject in a "breathless" state. Note the increase in PIFR and the decrease in the time intervals. Reproduced by kind permission of the author and publisher from Leigh JA, in *Scientific Foundations of Anaesthesia.*[4]

expiratory pause is variable, the amount of oxygen accumulated also varies and so the shorter the pause the less oxygen accumulates. Figure 2 shows that when the respiratory rate increases the expiratory pause virtually disappears. So in some devices both PIFR and respiratory rate will alter F_IO_2 (Table 2).

Variable Performance Devices

Variable performance devices are functionally subdivided into three groups, no-capacity systems, and small or large capacity systems (Table 2). No-capacity systems, e.g. nasal catheter, are not often used in the field, though their inherent simplicity and cheapness have much to commend them over other variable performance devices.

SMALL CAPACITY DEVICES

Here apparatus dead space is added in the form of a mask shell, resulting in rebreathing of carbon dioxide and oxygen. During inspiration, the mask, the volume of which is small relative to tidal volume, empties initially in series with entrained air so the higher F_IO_2s are inhaled at the beginning of inspiration. F_IO_2 then falls markedly and variably during mid-inspiration when PIFR is at its greatest.

This results in a scatter of inspired oxygen concentrations. The extent of this scatter of F_IO_2s may be assessed by multiple breath sampling techniques and plotting the measured oxygen and carbon dioxide concentrations of these separate expiratory gas samples on the O_2/CO_2 diagram as shown in Figure 3. The possible scatter of inspired oxygen concentrations in subjects breathing from a variable performance device is indicated by the broken 'R' lines and in this case varies between 40 and 73%. This variability may be overcome at lower F_IO_2s by employing the Venturi principle in the mask device (Figure 3, left-hand plot).

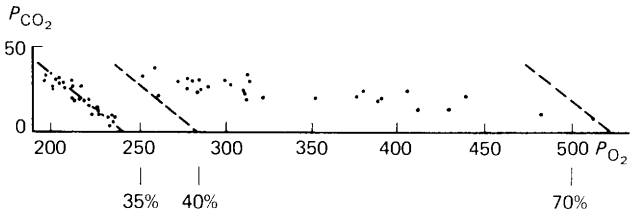

Figure 3. Comparison between fixed and variable performance oxygen systems. A 35% Ventimask has a single R line showing a fixed F_IO_2 of 35% (left-hand sloping dashed line). A variable performance system, MC mask with oxygen flow of 5 l/min, shows a wide scatter of inspired concentrations between 40 to 73% (between middle and right-hand dotted lines). Reproduced by kind permission of the author and publisher from Leigh JA in: *Scientific Foundations of Anaesthesia*.[4]

Rebreathing and apparatus dead space

Functional dead space is often less than the actual physical volume of the device, being that part of the previous expirate which is re-inhaled. Dead space results in rebreathing and the potential for carbon dioxide accumulation which may have deleterious effects on some patients. Functional dead space is increased if the volume of the device is large, the flow of oxygen is low, the expiratory pause is short or the mask is a good fit with reduced air entrainment (or increased resistance to air entrainment through the mask vent).

TABLE 2

VARIABLE PERFORMANCE OXYGEN SUPPLY DEVICES (F_IO_2 affected by patient factors)

System	Characteristics	Examples
No Capacity	F_IO_2 subject mainly to between-patient variation	Nasal cannulae < 3 l/min flow
Capacity	F_IO_2 subject to both between and within patient variation Apparatus deadspace is added, resulting in rebreathing of O_2 and CO_2	
Small capacity		O_2 mask without reservoir bag
Large capacity		O_2 mask with reservoir bag incubators, oxygen tents

LARGE CAPACITY DEVICES

These devices incorporate a rebreathing, or reservoir, bag in the system and their contents empty in parallel with entrained air (Table 2). Since breath by breath times, volumes and resultant flows and resistance to breathing all vary, the performance of these devices is very variable. The shorter the expiratory pause and the greater the expired volume in the bag, the less oxygen will be delivered to the patient and the more carbon dioxide will accumulate.

In summary, the factors influencing F_IO_2 in variable performance oxygen devices are shown in Table 3. Which of these several factors become of practical importance will depend on the device and the way in which it is being used.

TABLE 3

FACTORS INFLUENCING F_IO_2 IN VARIABLE PERFORMANCE OXYGEN DEVICES

1. Type of oxygen device in use
2. Apparatus dead space (rebreathing)
3. Mask/Mouthpiece seal (air entrainment/vent resistance)
4. Peak Inspiratory Flow Rate (PIFR)
5. Expiratory pause

Variable performance devices were the standard for field oxygen resuscitation equipment for decades.

A good example of the type is the Oxy-Viva apparatus with continuous oxygen flow (4-10 l/min) into a simple mask shell such as a Hudson or MC mask.[5] Such devices are unsuitable for divers, but are certainly better than no oxygen at all.

Fixed performance devices

Fixed performance devices are listed in Table 4. The fixed performance devices, when used correctly supply a predetermined F_IO_2 irrespective of the respiratory pattern of the patient. However, even devices classified as having fixed performance may ,under certain conditions, fail to deliver a constant F_IO_2.

As inspiratory flow is sinusoidal in nature, if an oxygen device is to deliver a fixed F_IO_2 then it must deliver the chosen mixture at a rate equal to or greater than PIFR. This may be achieved in one of five ways. Of these high flow gas blender/humidifiers and oxygen concentrators, will not be discussed as they have no application in emergency resuscitation.

HIGH FLOW

Air-entrainment devices

The oxygen fresh gas flow enters the mask through a venturi device and entrains a high flow of air so that the total gas flow into the mask exceeds PIFR under most conditions (Figure 3, left-hand plot). Venturi-type masks are therefore fixed performance devices, but are only efficient in the F_IO_2 range 24-40% and, even then, the oxygen flow into the device may need to be doubled from that recommended by manufacturers to achieve a fixed F_IO_2 since the entrainment ratio remains the same whilst the total mixture flow increases.

For instance, if a venturi-type mask is rated to deliver a F_IO_2 = 0.35 (35%) with a recommended oxygen flow rate of 6 l/min it is easily calculated that the flow of air entrained must be approximately 28 l/min giving a total fresh gas flow rate of about 34 l/min. This is very close to the resting PIFR in Figure 1 and well below that in Figure 2. In the latter case F_IO_2 would fall in mid-inspiration (the most important part of the inspiratory phase for oxygen delivery)

TABLE 4

FIXED PERFORMANCE OXYGEN DEVICES

F_IO_2 is independent of patient factors

System	Method	Examples
High Flow	Venturi operated	Hudson Multivent mask
	Demand regulator	DAN oxygen resuscitator
Low Flow	Constant flow	Some anaesthetic circuits
		Komesaroff resuscitator (OxyDive 1)[16]
		Wenoll oxgen delivery system[17]

unless the oxygen flow is increased to 10 l/min or above. A detailed study of one type of venturi mask has been provided by Woolner and Larkum.[8]

While venturi-masks have some very definite applications in medicine, their use is not indicated in diving accident resuscitation.

Demand regulators

The demand regulator is a very familiar device to scuba divers. Indeed one of the earliest approaches to providing 100% oxygen to diving accident victims utilised an adaptor block placed over a pin-indexed cylinder valve (or screwed into a bull-nosed cylinder valve) to which an ordinary scuba two stage regulator could be mounted by its A-clamp.[5] In those early days (late 1960s) the need for oxygen-compatible cleaning of the regulator was not appreciated by many, thus carrying the risks of fire and oxygen explosions. However only one fire, in a home-made adaptor, has been reported.[9]

The principles of demand regulators do not require explanation here. In spontaneously breathing subjects, these devices are triggered by the negative pressure generated in the airway by the inspiratory muscles and they deliver a fresh gas flow equal to the breather's demands. PIFR is matched and the total gas flow from the regulator equals minute ventilation which in the resting unstressed subject will be in the range 5-8 l/min, but in some situations may be 2-3 times higher than this. Thus the patient's minute ventilation determines the rate of consumption of the oxygen supply.

Demand regulator resuscitators are now widely marketed for diving applications, for instance as the DAN oxygen resuscitator, the LSP Portable Resuscitator and the Laerdal OxiDive 3, all of which utilise regulators manufactured by Life Support Products.[5] When used properly, their main advantage is that close to 100% inspired oxygen (F_IO_2=1) is achieved consistently, though this may be at the expense of high oxygen usage in the distressed patient.

Several factors influence how close to 100% oxygen the F_IO_2 actually achieved will be. Most important is the seal between the mask (or mouthpiece) and the patient's face (lips). Any leaks will result in air entrainment during inspiration and a fall in F_IO_2, particularly during mid-inspiration. It is rare for the seal to be perfect, particularly with face masks, where the design and matching to facial features may be poor. Beards make leaks more likely. Therefore some degree of air entrainment is extremely likely. This has been studied under hyperbaric conditions using the Scott Mask demand regulator where it was shown that a F_IO_2 of 1.0 (100%) was never achieved even under ideal conditions in trained nursing attendants and was less than 0.8 in some cases.[10]

Intermittent positive pressure ventilation (IPPV) in the non-breathing victim is not discussed in this paper. However IPPV is only possible with demand regulator systems where the exhalation mechanism is not automatically opened by positive pressure within the mask or is overridden by a manual triggering mechanism. Pulmonary overpressure injuries and gastric distension are life-threatening complications of IPPV in unskilled hands. The Laerdal MTV-100 (manufactured by Life Support Products) is an example of a demand valve that may be used for spontaneously-breathing patients or for manually-triggered positive pressure breathing and which meets the new Australian Standard 2488.[6] Some existing oxygen kits may not meet this standard.

LOW FLOW DEVICES

Semi-closed Rebreather Systems

All such systems consist of a fresh gas inlet into a circuit containing, at the least, a reservoir for the breathing

gases in the circuit, one or two hoses to deliver the inspired and expired gas volumes to and from the patient and an exhaust valve or opening to release surplus gases from the circuit. In order to economise on fresh gas flows, a system for removing carbon dioxide from the breathing gases is usually incorporated. If one-way valves are placed in the circuit to ensure unidirectional gas flow within the device, then this is called a "circle" system.

All anaesthetic circuits in use today follow these basic design concepts. The basic principles underlying their function are much the same as those outlined recently in the SPUMS Journal by Elliott and Hamilton for diving rebreathers.[11-13]

Again, the principle applies that, in order to deliver a fixed F_IO_2, the device must be capable of delivering the chosen gas mixture at a rate at least equal to PIFR. In a rebreathing circuit this criterion is satisfied as flow is met by partial collapse of the reservoir bag during inspiration, but only if the bag has filled properly during the previous expiratory phase and expiratory pause (see Figures 1 and 2). Once the reservoir bag is filled during exhalation, any surplus gases are dumped from the circuit via the exhaust valve. The great advantage of semi-closed rebreathers incorporating CO_2 removal is the economy of fresh gas flow achieved, with flows of less than one l/min being theoretically possible. In practice, higher flows than this are usually necessary.

This type of equipment is much more complex to use and requires training and repeated practice. Not only is the mask seal (and design) vital in both the spontaneously breathing[14,15] and apnoeic patient for effective oxygen therapy, but problems may arise with circuit integrity. Besides disconnections and other causes of leaks (e.g. splits in delivery hoses or connectors), unrecognised exhaustion of the carbon dioxide absorber, malfunction (usually sticking open) of the one-way valves in the circuit (both of which result in increased F_ICO_2) and malfunction of the exhaust valve may occur. Despite these problems, in the hands of experienced, trained personnel (such as anaesthetists and paramedics) these devices are extremely efficient and effective in terms of oxygen delivery.

An added advantage is that some warming and humidification of inspired gases occurs due to rebreathing of the expired gas and the generation of water vapour and heat by the CO_2 absorber. Dilution of fresh gases by the patient's expirate results in a fall in F_IO_2, the extent and duration of which is dependent on the oxygen fresh gas flow rate and metabolic oxygen uptake by the patient. Even at an initial oxygen flow rate of 8 l/min into a rebreather circle system, and under ideal conditions, it takes several minutes to achieve an F_IO_2 of greater than 0.95.[14,15]

In the diving setting initial oxygen flow rates should be higher than those recommended for general use and the circuit flushed out periodically to enhance nitrogen off-gassing. This is likely to result in an averaged flow rate approaching 3 l/min. Some air entrainment during spontaneous inspiration is inevitable, especially if masks are held to rather than strapped onto the face, and will be greater the lower the fresh oxygen flow rate into the circuit.

One such rebreather circle system now available on the Australasian market incorporates several unique design features, particularly related to the carbon dioxide absorber canister and the exhaust valve, and is marketed by Laerdal as the OxiDive1 resuscitator kit.[5,16] A Swiss designed rebreather for divers (Wenoll oxgen delivery system) was recently described.[16]

Oxygen supply duration

The quantity of oxygen that should be carried by any diving operation depends on:

a the type of oxygen resuscitator to be supplied,
b the distance/time to access medical assistance and
c practicalities such as the space available on a diving vessel.

Table 5 provides approximate durations for several cylinder sizes using three flow rates that are typical of those required for a rebreather circle system (3 l/min), a demand regulator in an undistressed, average-sized diver (6 l/min) and a mask with continuous high flow oxygen to achieve a high F_IO_2 (15 l/min).

TABLE 5

DURATION (IN HOURS) OF OXYGEN CYLINDERS AT THREE FLOW RATES

Cylinder			O_2 Flow Rate l/min		
			3 lpm	6 lpm	15 lpm
Size	Water volume (l)	Contains (m^3)	(Rebreather)	(Demand regulator)	(High flow with mask)
C	2.84	0.49	2.5	1.25	0.5
D	9.5	1.64	9	4.5	1.75
E	23.8	2.26	12.5	6.25	2.5
G	48.0	7.01	39	19.5	7.75

Most oxygen first aid equipment in Australia is supplied with a C size cylinder with a capacity of 490 l, whereas in New Zealand this is not available, the equivalent being the smaller A size cylinder with a capacity of 440 l. For diving locations close to urban areas where running times to shore, helicopter retrieval, etc, are less than two hours an A cylinder is adequate with rebreathers but marginal in supply duration with demand regulators. In all other situations where delay in evacuation over two hours is likely or only a continuous flow device is available, an A size cylinder is quite inadequate. The most convenient cylinder size to carry under these circumstances is the D size which is similar in size to larger scuba tanks and therefore relatively easily handled and stored. In remote regions it may be necessary to carry several of these rather than opting for heavy G storage cylinders.

Other requirements

For wide application in the diving community, it is not only the theoretical performance characteristics of oxygen resuscitation equipment that are important. Of equal importance are ruggedness of construction, simplicity of design and use, the training requirements for safe and effective use, ease of maintenance and the cost.

The recent introduction onto the market of demand regulator and rebreather systems (both fixed performance) in robust waterproof casings is a major step forward in the field care of diving accidents. However, they carry with them a need for considerably more training and an obligation on the part of oxygen attendants to maintain airway management skills. This is especially the case with a circle system resuscitator like the OxiDive1 which is excellent for the trained and experienced physician or paramedic but which the average diver with basic first aid or a DAN oxygen course under his belt would have some difficulty using without full training in its use.

The various performance characteristics, and some of the advantages and disadvantages, of the three main types of oxygen resuscitators commonly available, continuous flow oxygen via a low capacity device, a demand regulator and a rebreather circle system, are summarised in Table 6. Several resuscitators on the market incorporate both the first

TABLE 6

CHARACTERISTICS OF OXYGEN SUPPLY DEVICES

Oxygen device	**Mask with continuous flow**	**Demand regulator**	**Rebreather System**
Device type	Variable F_IO_2	Fixed F_IO_2	Fixed F_IO_2
Oxygen economy	Poor (8-12 l/min)	Moderate (5-15 l/min)	Efficient (2-3 l/min)
Likely F_IO_2	Well below 100%	80-95 %	90- 95 %
Inspired humidity	Drier than air	Very dry	Warm and humid
IPPV *	No	Some types	Yes
Ease of use	Simple	Familiar concept Mask/mouthpiece seal critical	Mask seal and circuit-integrity critical
Problems	Inefficient	Air entrainment Overpressure injuries can occur when using IPPV	Air entrainment Disconnections Reservoir bag collapse CO_2 absorber failure Expiratory valve sticking
Training needs	Minimal	Moderate	Considerable
Cost	Cheapest	Moderate	More expensive Ongoing for consumables

* IPPV = Intermittent positive pressure ventilation

two types of device in a single oxygen regulator. A wide range of commercially available resuscitators is well illustrated by Lippmann[5]

Conclusions

A wide range of oxygen therapy equipment is now marketed and an Australian Standard is in place.[6] A clear need exists for independent assessment of equipment performance to identify those systems and designs most suited to diving operations ranging from recreational shore diving to the off-shore oil industry. This would provide a valuable SPUMS diploma thesis.

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CRITICAL INCIDENT STRESS DEBRIEFING

Jeff Bertsch

Key Words
Accidents, stress, trauma, treatment.

This talk is about trying to prevent post-traumatic stress disorder, which we call PTSD. The hyperbaric unit where I work is very much involved with the diving community. Our marketeers boast the Florida Keys as the diving capital of the world, which is debatable, after having been diving around the world! But we have a large number of diving professionals (dive pros) at work in the area. Dive shops in the Florida Keys put about 10,000 divers in the water a month. As a result our unit sees about 40 diving accidents a year. We have only been open about three years, but around 50% of our cases are acute cases. By that I mean, that the when the diver ascends, he is either unconscious, paralysed or there are other acute or severe neurological symptoms present.

We have done a very good job at this conference talking about providing care, the best care possible, to our injured divers. I would like to shift the conversation just a little bit and talk a little bit about caring for the health care providers. This is something that has not been discussed much in diving and hyperbaric medicine. However, it has been discussed and looked at length and in a great detail involving emergency medical services (EMS) and public safety personnel. I have become involved with this over the past couple of years.

First of all, I would like to define a "critical incident". It is typically an event where there is loss of life or near loss of life. Tragedies, death, serious injuries, threatening situations are all something that we as health care providers and as diving professionals can see. I look at diving professionals as being the first line of health care providers. In our area the care that a diving professional

provides to a diving accident, or diving injury, quite often can help to make our job easier and enhance the outcome of a diving accident.

Post-traumatic stress disorder symptoms have been recorded following dive accidents. I really started noticing PTSD in the years that I worked as a medic following up on diving accidents. When we followed up these accidents we also followed up the instructors who were involved in these accidents and they had a lot of questions. Hyperbaric units that we talked to also had similar reports from their divers and their dive pros. It definitely warranted some closer attention.

In Monroe County, where my unit is, we developed (really we are in the process of developing) a critical incident stress-debriefing team, called a critical incident stress management (CISM) team to work with EMS. However, with such a large diving population, and a lot of requests to my unit from the diving community, we have also expanded our mission to provide debriefings to the diving community.

Let me quickly go over what is critical incident stress debriefing and what is critical incident stress management. It is a confidential, integrated system of interventions designed to prevent adverse psychological reactions from a critical incident. This does not replace or provide psychotherapy at all. What it does is it helps lessen the impact of a major event for the hyperbaric oxygen (HBO) therapy providers or for the diving professionals. It also accelerates normal recovery of normal people who are experiencing high levels of stress after an abnormal event. Again it does not provide psychotherapy. What we use in our program is something called the Mitchell Model. Jeff Mitchell is a clinical psychologist who did his thesis in the US on PTSD and critical incident stress management.

This service does identify those who may benefit from follow up psychotherapy. So there are several stages in which CISM and critical incident stress debriefing is performed. It generally utilises group support in a safe and confidential environment. Initially there can be what is called the defusing, which is a one on one session with a certified peer counsellor, or mental health professional, who has been trained in critical incident management. That offers stress management education and support, establishes a need for formal debriefing, stabilises crew members and dive pros, so they can return back into their normal life and also return to work quickly.

A formal debriefing in a group, say of divers, or of a hyperbaric staff, generally will occur 24 to 72 hours after the incident. This is a confidential, non-evaluative discussion. The best way to work through these stressful situations is to talk, talk, talk it out. Talk about your emotions, thoughts and feelings of the incident and then follow an educational format on stress and how to cope with stress as a follow up to the incident.

What I would like to propose, and this is something we are going to do in our area, is the rationale for divers. CISM has been typically used for EMS and public safety staff. There is some excellent literature supporting the effectiveness of CISM for EMS and for public safety. There is very little literature, if any, involving critical incident stress debriefings for the diving community.

We would like to make this service available to our local community and track the results. This is going to be difficult at best, simply because when we give a debriefing, or defusing, there is absolutely no documentation kept during those sessions and confidentiality is a priority. However, we will be able to keep a list of participants' names and follow up on them one month, three months and six months after the incident and hopefully with that information I will be able to report some future findings.

In conclusion, with critical incidents involving divers, which are a regular occurrence, at least in our area, we hope to make a contribution to preventing post-traumatic stress disorder and at least reduce the risk of burn-out which leads to drop-out for involved professionals in the local diving and hyperbaric community.

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ARTICLES OF INTEREST FREPRINTED FROM OTHER JOURNALS

ASKING THE RIGHT QUESTIONS

Bob Halstead

Key Words
Environment, recreational diving, risk, safety

Many people would like to believe that the secret of a happy life can be reduced to a few rules, which, if held inviolate, would guarantee success. No doubt there are some that feel the Ten Commandments satisfy that requirement. They provide a sense of security to those bewildered by life's complexities and I am not knocking it.

In fact I personally believe that in order to be happy, though human, it is best try to understand human nature and follow the fundamental truths that wise people have passed on through the ages. This is why I always remember my wife's birthday, and never start a dive cruise on a Friday.

Alas humans are far from perfect, and although this fact is well known, many people, proving the point, still confuse the way we should behave, if we were all good and nice, with the way we actually behave, rather more selfish and sinful.

It is this difference of course which has doomed Socialism to the dustbin of history. It is all very well to say "From each according to ability, to each according to need" but human nature is such that, unfortunately, all that this produces is a lot of people with needs and, suddenly, very few with any abilities.

In the world of diving there have been many instances where good people have tried to proclaim the answers to safe and happy diving in a few simple rules. Some of these have merit, "Always come up slowly" springs to mind as a rule that is hard to argue with. However many of the so called Golden Rules are really only superficially correct and ignore human nature. Sometimes the rules provide answers, but to the wrong questions. Let me explain.

There is a rule that is a relatively recent addition to dive safety manuals that goes:-

If you are doing a series of dives in a day you should make the deepest dive first, followed by successively shallower dives.

The corollary covers the conduct of an individual dive:-

If you are making a multi-level dive you should go to the deepest part of the dive first, and then continue to progressively shallower depths.

The reason for this rule is that such conduct produces less nitrogen loading, and therefore reduces the likelihood of decompression sickness.

BUT this marvellous rule presupposes that the question is "I am about to make several dives to various depths in the same day. In which order should I make the dives." With this question the answer is clear - follow the rule!

NOW let us ask a different question and introduce human nature into the equation.

I am going to dive a famous wreck this afternoon at a depth of 30 metres. In the morning we can dive at a reef site. How deep should I dive on the reef?

This is a very real situation as sometimes it is just not possible to reach a particular site in the morning. Don't you just love the question? I suggest you try it on your Dive Master or even better the local Queensland Diving Inspector. What they are going to tell you, I bet, is that the first dive should be deeper than the second, they will follow the rule, when it is obvious that it would be much safer if you only went to 10 m on the first dive.

Of course they could ban the morning dive, and risk mutiny, but what is actually happening in circumstances such as this is that divers are making deep bounce dives, to say 35 m, just so they will be able to dive to 30 m in the afternoon, when they would have been quite happy making a dive to only 10 m. In fact they will make an immediate ascent from 35 m and spend the rest of the dive at 10 m anyway. The important thing is to have 35 m on the computer for the dive master to let you make the afternoon dive to 30 m. Human nature, I love it.

The stupidity of blindly following rules was brought home to me when I was severely criticised because I started one day with a marvellous early morning dive at an anchorage in 2 m of water. My fellow divers insisted, admittedly tongue in mouthpiece, that the rest of the day's dives were to be shallower.

Those of you into live-aboard diving know that when you are really DIVING your computer rarely clears before your next morning's dive. This means that the rule should actually not be limited to only one day. If the dive master is being really conscientious he would insist that, in that circumstance, the first dive of the new day must not be deeper than the last dive of the previous day, and that the computer

must clear before you can go deeper. I just throw this in because it is the logical result of applying the rule absolutely. I admit I have never heard of this actually happening. Perhaps you have, if so I would love to hear from you by e-mail (halstead@internetnorth.com.au)

So to see if the answer is appropriate perhaps we should pay more attention to the question. How about these:-

1 Will the person I am about to buddy with increase or decrease the risk of the dive I am about to make?

2 If my buddy stays on board the boat while I dive, will I be less likely to be left behind by the boat?

3 If I choose to dive only in ideal conditions am I likely to see large pelagics such as Hammerhead Sharks?

4 If I wear a large BCD with all the bells and whistles, will I still be able to swim through the water?

5 If I do not log my sex life, why should I log my dive life?

6 Should I carry my own emergency air supply or let my buddy carry it for me? (As in, should I carry my own spare parachute or let my fellow jumper carry it for me?)

Just a few examples, if you have any of your own please e-mail me and I will publish them with credits.

So in diving, as in life, rules do not replace thinking.

Having published this story I was pleased to receive an e-mail from Stephen Bilson (See Letters to the Editor, page 135). The horror story he tells does demonstrate how important it is to select a good operator for your dive holiday. The bounce diving phenomena has been partly caused by over-zealous application of the deepest dive first rule by at least one of the Queensland Workplace Diving Inspectors, yet again demonstrating how Workplace contributes just as much to decreasing dive safety as it does to improving it. Other contributors to this nonsense are inexperienced divemasters, and operators who do not have standard procedures in place to guide their divemasters.

Reprinted, by kind permission of the Publisher, from Dive Log 119, June 1998.

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ARE YOU AN ACCIDENT WAITING TO HAPPEN?

Breezing off to sea in the first warm days of early summer may be a wonderful release, but if your boat has languished unchecked in the garden since last year, it may prove to be a release from this earthly life! Here are our tips for surviving the new season.

Spring has sprung and every week-end you will see cars heading coastward, trailing just about anything that might float.

Many of these towed vessels have stood idle in gardens, garages and driveways since the previous summer and it comes as no surprise to hear how many come to grief almost as soon as they reach the water. It is a busy time for the coastguard and lifeboat services, and, it is sad to say that, with a little forethought, most of the disasters and near-misses could easily have been avoided.

Many people are prepared to head out to sea in inappropriate weather and aboard unsuitable vessels. Even on a hot day, the sea can be an inhospitable place, and many seagoers are ill-prepared for their adventures.

The first thing to look at is yourself. Are you ready to go to sea? Has your body been idle all winter like so many of those driveway inflatables? Do you get out of breath running for a bus and does your figure reflect a winter of too many pints and chip butties? Maybe you should think about getting into shape!

Divers distinguish themselves by actually exchanging the relative safety of boats for the hazards of swimming in cold and often fast moving water. Add to that the pressures of gas absorption plus the vagaries of sometimes out-of-practice diving techniques and you have a formidable combination.

So, have you done some swimming and some work-up dives in relatively easy conditions, or do you intend to make your first dive of the season in a fast tidal flow at 30 m on a wreck like the Kyarra, off Swanage (a very popular Bank Holiday Monday site)?

Health and fitness should be a number one priority, combined with conditions chosen to be less than daunting at this early time of year. Then there is the question of knowing your own limitations. Be really sure of what you are doing and don't be dragged along by the possibly reckless enthusiasm of your companions.

You should be certain that you have sufficient knowledge to be able to make sound decisions about your diving. People can be full of bravado sitting on a deck in the sunshine. I remember once being almost persuaded by my buddy to discard my lamp before diving a wreck. He

put himself forward as a real expert while on board, but became a very frightened (lampless) diver under water.

Are you going to make sure that you have got slack water when you dive, and will you know the actual site depth? Do you know how to calculate these things or are you going to rely on someone else, possibly to make mistakes on your behalf?

Equipment can let you down. Regulators should be serviced every winter and tried out in your branch pool before committing yourself to the sea. A regulator that has just been serviced can be perfect in the dive store but go into free flow once it smells the sea air.

Even simple equipment failures can ruin your dive and possibly turn pleasure into panic if you are not prepared. A mask that was perfect when you put it away last October can develop a leak where the lenses meet the frame, which only manifests itself under the pressure of depth. Are you able to cope with a mask that perpetually floods with cold water?

Drysuits should be checked for deterioration, too. A small leak may make your dive cold and miserable, but a dump-valve which pulls off in your hand can turn a comfortable drysuit into a sea anchor. You should be sure that all of your equipment is up to the job. Boats are notorious for letting you down and, unlike cars, you cannot walk off somewhere for help. Boats never break down while they are safe at home on their trailers.

The most common cause of engine failure is a worn water pump. It is a part that is easily replaced and should be done routinely.

An uncooled engine quickly becomes a dead engine. Engines should always have enjoyed proper winterising prior to storage but also need a good inspection before setting off for that distant wreck.

Even the tubes of your RIB (rigid inflatable boat) should be thoroughly checked, inside and out. I know of one club that discovered their boat had become a single chamber vessel, due to internal baffle failures, only after they punctured a part of the tube out at sea. It was a very touch-and-go situation with a safe return to shore made possible only by flat, calm water.

Once, as the guest of a local branch, I sat bemused as we wallowed without power, listening to a violent inquiry as to who was responsible for fuelling the empty petrol tanks!

With the advent of electronics, we have come to rely on modern navigational aids. But even electronics sometimes go wrong. Do you know how to get home when the silicon chips are down?

And do you know how to use your equipment properly? I heard of one club outing which distinguished itself by setting off with a new GPS without entering their point of departure. Safety equipment is often ignored until it is needed. In the event of problems, will it all function? Does your radio work and will more than one of you know how to use it? Will you make a radio check with the coastguard?

Flares lie idle and can be unreliable. Replace your smoke and parachute flares before they go out of date and dispose of the old ones in a safe manner.

Do you have a fire extinguisher on board, and is it likely to function properly if you need it in a hurry? The same can be asked of your first-aid kit. It is almost bound to have been plundered in the past and some essentials will need replacing. Are you up to date with first aid techniques?

Finally, it is useful to brush up on your diving skills before heading for the sea. BSAC training has a strong bias towards teaching every diver what to do in an emergency, but because emergencies thankfully seldom arise we can all get rusty. Branch nights and trips to inland sites should not only be used to teach rescue techniques to novices but also to keep one's own skills honed. One of the best ways to keep on top of a practical subject is to teach it to others.

Should the unthinkable happen and a diver become lost or reach the surface in a serious condition, do you know how to react? Do not leave it to others. Enjoy your diving in the knowledge that you have hoped for the best, but planned for the worst!

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The above was written for UK divers but it applies equally well to Australians and New Zealanders looking forward to the end of winter. It also applies, in part, to those SPUMS members, who, like the Editor, have become regular, one-warm-water-trip-a-year, divers. Elderly electronic devices can fail underwater without warning, as did the Editor's very early model all-in-one depth gauge, bottom timer and contents gauge, cutting short a lovely dive.

GLEANINGS FROM MEDICAL JOURNALS

DIABETES

Scuba diving and diabetes; collecting definitive data from a covert population of recreational divers. Interim observations from a long term on-going prospective study. Bryson P, Edge C, Gunby A and St Leger Dowse M. *Undersea Hyperbaric Med* 1998; 25 (Suppl): 51-52

Abstract

Background

Our understanding of the effects of scuba diving on diabetes or the effect of diabetes on a divers ability to dive safely is minimal. The UK Sport Diving Medical Committee allows diabetic divers, who are well monitored and controlled, and who submit themselves for an annual medical, to dive.

Method

In 1991 a data base of British recreational diabetic divers was initiated. A questionnaire was designed which sought to establish the diving activity, health, diabetes control and management, any diving incidents relevant to diabetes, and frequency of a diving medical. Since 1991 the questionnaires have been circulated to medical practitioners, diving medical referees, diving organisations and to diabetic divers. With an *estimated* 90,000 recreational divers in the UK and an estimated 3% (2.5% known, 0.5% unknown) of the UK population suffering from diabetes, a theoretical upper limit to the number of diabetic divers in the UK could be >2,700 and a lower limit could be as high as 500 (at present there are no data on the total numbers of divers with diabetes).

Results

Data from 2,478 dives by 155 diabetic divers (20% females and 80% males), with a mean age of 38 years (16-63) demonstrated that divers, known to the study and suffering from diabetes (13% NIDDM and 87% IDDM), logged an average of 15 dives per year, compared with an estimated UK recreational diver average of >30 dives per annum. Only a small nucleus of respondents have consistently logged >40 per annum. The respondents did not dive continuously year on year. Annually our data shows 38% of diabetic divers are new to the sport, reflecting trends of the diving associations. The majority of the total respondents reported they were under the care of a hospital. However, our data showed 19% had either not had a medical in the last two years, or failed to give the date of their last medical at all, implying their diving medical was not in date.

Conclusion

Our study suggests that gathering definitive data from recreational diabetic divers is a long term project which needs careful and thorough management. Annual follow up of respondents, the facility to evaluate any reason, medical or social, for leaving the sport and the ability to gather data from the growing number of divers with diabetes who are not part of the "club" system, are all factors to take into account and that if not addressed could skew future results.

From

The Hyperbaric Medical Centre, (DDRC-Plymouth), Plymouth, Devon, PL6 8BQ, UK.

Key Words

Diabetes, recreational diving.

DIVING ACCIDENT DATA

INM/BHA diving accident database: analysis of cases 1991 to 30 Sept 1997.
Benton PJ and Glover MA. *Undersea Hyperbaric Med* 1998; 25 (Suppl): 440-41

Abstract

Background

Since 1991 all members of the British Hyperbaric Association (BHA) have forwarded, using standardised reporting forms based upon the descriptive terminology, details of all diving incidents they have treated to the INM for inclusion on a computer

To date the database contains details of 1,422 diving related incidents including 923 cases of decompression illness (DCI). All reports are audited by a diving physician before entry into the database.

The majority of divers were male (84.3%), mean age of male divers 33.8, range 13-71, mean age of female divers 30.4, range 15-50. 1,110 (78%) were amateur divers, 250 (17.6%) civilian professional divers and 62(4.4%) military. Neurological manifestations were present in 721 (78%) cases of DCI, with sensory abnormalities in 545 (59%) and motor deficit in 266 (28.8%) cases. In 139 (15%) cases impairment of higher mental function was reported. Limb pain was present in 454 (49%) cases of DCI, with girdle/back pain in 23 (2.5%). In 140 (15.2%) cases limb pain was the only manifestation of DCI. Constitutional manifestations (fatigue, malaise, headache, vomiting) were reported in 248 (26.8%) cases. Less common

manifestations such as skin 86 (9.3%), pulmonary 36 (3.9%) and lymphatic 8 (0.9%) were also reported. In 471 (51%) cases of decompression illness more than one manifestation was reported. The mean depth of dive prior to DCI was 33 msw with 97 (10.5%) cases of DCI occurring following dives to 50 msw or deeper, 95 msw being the deepest pre-incident dive by an amateur diver. Data for 1995-30 Sep 1997 (n=409) reveals that following initial recompression therapy 216 (52.8%) cases reported complete resolution of symptoms whilst on completion of hyperbaric oxygen therapy (HBOT) 303 (74%) cases reported complete recovery. Residual manifestation were predominantly minor sensory disturbances although 10 (2.4%) cases reported persistent impairment of gait and/or motor disturbance despite aggressive HBOT.

The authors are indebted to the members of the BHA for their continuing support of this study.

From

Institute of Naval Medicine, Alverstoke, Gosport, Hampshire PO12 2DL, UK.

Key Words

Accidents, data, decompression illness.

FLYING AFTER DIVING

Commercial airflight after recompression therapy for decompression illness.
Uguccioni DM, Dovenbarger JA, Hobgood JA and Moon RE. *Undersea Hyperbaric Med* 1998; 25 (Suppl): 36

Abstract

Background

While there is no universal agreement on the appropriate interval before flying after recompression therapy for DCI, a common recommendation is 72 hours. We present a retrospective study of treated DCI to determine symptom re-occurrence during flight.

Methods

All cases in the Divers Alert Network (DAN) database for which treatment was administered from 1993-1995 in either Grand Cayman or Cozumel and was followed by a flight to the US were reviewed. Follow-up was attempted by telephone.

Results

Of 151 cases eligible for the study, follow-up was available on 126. The median number of initial treatments was two (range 1-22). Seventy-four cases (59%) waited less than 72 hours before flight; most of these waited 48 hours (52 cases [41%]); Four individuals (3 %) flew within 12 hours of treatment. Fifty-two individuals (41%) waited 72 hours or more before flying; 17 (%) of these who waited greater than 4 days to travel; one individual waited 14 days before flight. Data are summarised in Table 1.

TABLE 1

Relief After Original Treatment	Yes (N=95)	No (N=31)
Flight < 72 hrs	54 (73%)	20 (27%)
Return/worsening during flight	3	17
Subsequent recompression	2	8
Resolution after 2nd treatment	1	3
Flight >: 72 hrs	41 (79%)	11 (21%)
Return/worsening during flight	6	5
Subsequent recompression	5	1
Resolution after 2nd treatment	3	2

Of the symptoms, which recurred or were exacerbated by flight, nine were severe neurological symptoms, 17 were mild neurological symptom and five were pain only symptoms (*DAN Annual Report on Decompression Illness and Diving Fatalities. 1997*)

Conclusions

This retrospective review shows that there was a subset of divers in both groups who experienced a return or worsening of symptoms with commercial flights after treatment. The return was more likely in those divers who did not have completely resolved symptoms prior to flight and symptom lasted longer in those who flew in less than 72 hours after initial treatment.

From

Divers Alert Network and Duke University Medical Center, Durham, North Carolina 27707, USA.

Key Words

Decompression illness, flying

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Cost in Adelaide $Aust 520.00

Unit 2 Diving Medicine Lectures and
Unit 3 Casualty Paramedical Training.
Unit 2 26/10/98 to 30/10/98
Unit 3 2/11/98 to 6/11/98
Cost $Aust 300.00

Diver Medical Technician Refresher Course
(includes lectures and practical)
26/10/98 to 30/10/98
Cost $Aust 500.00

For further information or to enrol contact
Professor John Williamson, Director, HMU,
Royal Adelaide Hospital, North Terrace
South Australia, 5000.

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THE SEA PEOPLE'S GUIDE TO DIVERS
PART TWO

By Rico

Humans say that to see themselves as others see them is a great blessing. Imagine then what a blessing it would be to see themselves as other species see them. If only we could find a way of giving them a Sea People's view of themselves. Well, actually, we can...

Thanks to the kindness of Rico, the cartoonist, and of Bernard Eaton, the Editor of DIVER, who have agreed to allow this series of typical divers to be reproduced in the SPUMS Journal. Although the featured diver types originated in the UK, we believe that most of them, at one time or another, have attended a SPUMS Annual Scientific Conference.

Demolition Crab

The Demolition Crab is the victim of an unhappy childhood. His parents were spartan in their choice of toys, and "educational value" was to rob him of the fun and novelty every child craves in his playthings. So, like the decorator crab, he embellishes his torso with ornamental baubles in an absurd compensation for his early emotional hardships. The Demolition Crab sports more fins and wings than a '59 Cadillac, and enough chrome barracuda lures for his own private feeding frenzy. His gadgets, straps and trailing consoles help bring back the wide-open spaces to coral reefs everywhere. He rarely loses his buddies, so easy is he to follow down his own cloudy trails of destruction.

Bulldozer-Turtle

The Bulldozer-Turtle is the friendly giant of the diver's world. He is not so much a Popeye: more a benign Bluto. To his buddies he is usually just Big Dave. There is a Big Dave in every dive club. Without him a fleet of heavy plant machinery would have to accompany every club convoy. He can lift a 50-horse Merc into a Land Rover with one hand. He can lift the RIB and trailer while Little Jock changes the wheel, with the crew still aboard. When a diver spots a nice little porthole jammed under a 2-tonne boulder, who else is going to readjust the landscape for him but Big Dave?

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