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OBJECTS OF THE SOCIETY

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

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Abstracts are also required for all case reports and reviews. Letters to the Editor should not exceed 400 words (including references which should be limited to 5 per letter).

References

The Journal reference style is the "Vancouver" style, printed in the Medical Journal of Australia, February 15, 1988; 148: 189-194. In this system references appear in the text as superscript numbers.[1,2] The references are numbered in order of quoting. Index Medicus abbreviations for journal names are to be used. Examples of the format for quoting journals and books are given below.

1 Anderson T. RAN medical officers' training in underwater medicine. *SPUMS J* 1985; 15 (2): 19-22

2 Lippmann J and Bugg S. *The diving emergency handbook*. Melbourne: J.L.Publications, 1985

There should be no full stops after the reference numbers. There should be a space after the semi-colon after the year and another after the colon before the page number and no full stop after the page numbers. The Journal uses two spaces after a full stop and before and after the journal name in the reference. The titles of books and of quoted journals should be in italics.

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Any report of experimental investigation on human subjects must contain evidence of informed consent by the subjects and of approval by the relevant institutional ethical committee.

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SPUMS ANNUAL SCIENTIFIC MEETING 1996

ARTICLES OF INTEREST REPRINTED FROM OTHER JOURNALS

GLEANINGS FROM MEDICAL JOURNALS

SAFE LIMITS: AN INTERNATIONAL DIVE SYMPOSIUM

MEETINGS AND COURSES

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To obtain Diving Incident Report forms write to DIMS, GPO Box 400, Adelaide, South Australia 5000.

The Editor's Offering

In this issue we publish the SPUMS Policy on Technical Recreational Diving (printed on pages 168-170) developed at the well attended Annual Scientific Meeting at Paradise Island in the Maldives. The meeting left the wording up to the Past-President, Dr Des Gorman, assisted by the two guest speakers, Drs David Elliott and R W (Bill) Hamilton, and Drew Richardson, from PADI International, but the directions were laid down over three days of lectures and discussion. The bottom line is that the term "technical diving" is used to cover a multitude of techniques of diving with very different risks for the diver, so divers need to know the risks that they are accepting before diving outside the usual recreational limits. Using enriched air nitrox with open circuits while staying within the safety limits for partial pressure of oxygen (PO_2), which means watching ones depth accurately and not drifting deep by inattention, is no more dangerous than using air in a similar way. Going deep on air can be accompanied by unconsciousness, which can be survived if one is wearing a full face mask but not with the usual recreational equipment. Diving deep on air is dangerous and has been associated with many deaths. If a diver wants to live to draw the pension or retire in style on superannuation deep diving should be avoided. The risks are too high. It was the desire to avoid the risks of deep diving using air, but still go deep because the caves the divers were exploring went down, which led to cave divers in the USA starting to use mixed gases, helium, oxygen and nitrogen to lower the level of narcosis and at the same time reduce the PO_2 to safer levels. One of our guest speakers, Bill Hamilton, has been involved with producing "customised" decompression tables for various groups of recreational divers using special gas mixtures. The other, David Elliott, has supervised many deep dives during his time with the Royal Navy and Shell. His paper on Deep Water Blackout should be read by every diver who is happy to dive deep on air. It was diver incompetence at work deeper than 50 m which led to mixed gases being used by commercial diving companies at greater depths. The divers did better work and the jobs were done satisfactorily and quicker so more economically. Changing to a more expensive breathing gas actually saved money and reduced diving accidents. However the risks to recreational divers using face masks which only cover the nose and eyes are higher than to occupational (the politically correct term for commercial) divers who use a full face mask and surface supply. Many recreational divers run out of air underwater and some die from air emboli during ascent, some go unconscious for various reasons, the regulator drops out of the diver's mouth and he or she drowns.

Nitrox, which is not always oxygen enriched air, is discussed by David Elliott and by Drew Richardson and Karl Shreeves, who outline the PADI Enriched Air Diver Program

We finish reprinting the papers from the Safe Limits Symposium. We suggest that now is the time to pull out the other two issues in which the other papers appeared [1995; 25 (3) and 1996; 26 (2)] and read all the articles one after another to get the full picture. We are grateful to the Workplace Health and Safety Division of the Queensland Government Department of Employment, Vocational Education, Training and Industrial Relations, for being interested enough in diving safety to organise a most useful symposium and then give SPUMS permission to reprint the proceedings so as to bring the symposium to a wider audience.

The two original articles were submitted as diploma theses. It is a pleasure to record that both candidates were successful. If a diver, who could manage a sharpened Romberg test before the dive which brought him to seek medical advice, cannot manage more than 15 seconds the chances are very high that he or she is suffering from decompression illness (DCI). However lasting 30 seconds is not a guarantee that DCI is absent.

Those readers who have read the July-August issue of Scuba Diver will have made their deductions from the girl's story. Carl Edmonds provides a diving medical view of the episode in Scuba Kids. This is enhanced by a well reasoned critique of the follies of teaching young children, under 16, to dive and giving them a certification which allows them to dive with another certificated diver. Though Carl taught his children to dive many years ago they always dived on a buddy line attached to him until they reached years of discretion. We hope that honest opinions backed by evidence will not destabilise the satisfactory SPUMS and the diving industry relationship. The rationale for SPUMS' existence is to improve diving safety, which may occasionally mean pointing out ways to avoid unnecessary problems.

In an aging population, where many over 50s want to learn to dive, doctors have to be sure that these latecomers to diving are fit enough to tolerate the workload. Yet to give everyone over the age of 30 a proper exercise ECG stress test is expensive and not cost effective. Fred Bove dealt with cardiovascular problems and diving (he is a cardiologist) at Castaway Island in Fiji last year. His paper appears in this issue along with Paul Langton's on patent foramen ovale and diving. We still have a number of clinical papers from Fiji whose authors are being coaxed into activity so that the papers can appear in the December issue. This does not mean, however, that you should not write a paper for the Journal.

It is participation by all members contributing papers which makes the Journal what it is.

ORIGINAL PAPERS

A REVIEW OF THE SHARPENED ROMBERG TEST IN DIVING MEDICINE

Ben Fitzgerald

Abstract

The use of the sharpened Romberg test (SRT) was evaluated in injured divers. Over a 12 month period, thirty five divers presenting with decompression illness (DCI) to the Naval Base in Auckland were assessed before hyperbaric treatment and at discharge. Their scores were compared with those of an age and sex matched control group (n=60). Abnormal SRTs were seen in 49% of divers (n=17) before treatment. These results were significantly improved at discharge ($p<0.001$). The results in injured divers were significantly lower than controls at presentation ($p<0.001$), but not at discharge. The SRT is consequently considered to be a valuable examination in divers who suffer DCI. It is proposed that the SRT be conducted in a standardised manner and be scored as the best attempt of four.

Key Words

Decompression illness, investigations, treatment.

Introduction

Neurology only developed as an independent discipline in the second half of the 19th century, due to the delayed discovery of the underlying neuroanatomy and physiology.[1] Moritz Romberg (1795-1873) was involved in early clinical research of the physiology and pathology of the nervous system.[1,2] He is credited with writing the first textbook of neurology and described the sign which bears his name in 1851.[1-3] The earliest description of a modified (sharpened) Romberg test was in 1944.[4]

The Romberg test has become part of the routine assessment of gait and equilibrium.[5] When the sign is positive, it is generally said to be diagnostic of proprioceptive deficiency (disorders of the posterior column of the spinal cord).[6-9] It is also regarded as a useful indicator of vestibular impairment.[10-12] A positive Romberg sign is also found in unilateral or bilateral motor weakness, diseases of the peripheral nerves, cerebellar disease and vertebrobasilar disease.[5,8,9,12,13]

The sharpened Romberg test

In this investigation the subjects wore flat shoes or bare feet and were assessed on a flat surface. They stood heel to toe (tandem position), with their arms crossed so that the open palm fell across the opposite shoulder. The patients closed their eyes once they were stable. They tried to maintain this position for 60 seconds, or for four trials, if 60 seconds was unattainable. A completed test of 60 seconds was scored as 60 for each subsequent trial e.g. if the subject stood for 60 seconds on the first attempt, they would score 60 x 4 = 240. If they completed 15 seconds initially and then 60 seconds on the second attempt, they scored 15 + (60 x 3) = 195. Patients continued with the trial until they had stood for 60 seconds or until they had completed four trials.[11,15]

The SRT, also known as the tandem, augmented or modified Romberg test, is more sensitive and can be quantified.[10-14] This test is generally described as a reliable indicator of a loss of vestibular function,[10,11] cerebral ataxia, sensory ataxia, and sensory neuropathy.[13] The test is more sensitive than the standard neurological examination for assessing changes over time and hence is useful in assessing clinical deterioration.[13]

Two US Naval medical officers assessed the value of Barbey's SRT in the evaluation of ataxia in aircrew in the mid to late 1960s.[4,10,11,15] This has been followed by considerable ongoing evaluation of the test in aircrew and in patients with disequilibrium.[3,10,11,13-17] By the early 1970s, the test was also considered to be useful in diving.[18] It is now used by several Navies (USN, RAN, RNZN) in their diving programs and is regarded as being useful in diving medicine as a baseline and in the assessment of patients with dysbaric illness and injuries.[19-20]

Although the SRT is used in the examination of patients with DCI, the application and interpretation of this test is usually neither standardised nor understood. The SRT requires more skill than the classical Romberg test.[3] One group of researchers suggested that although the SRT was useful in the assessment of gross balance abnormalities, the Stand on One Leg with the Eyes Closed test (SOLEC) was better for fine distinction in balance performance.[3] This claim was not supported by data. For this test the subjects stand with their arms folded against the chest. They stand for 30 seconds, or for 5 trials. Times are added and scored out of 150. A completed test of 30 seconds is scored as 30 seconds for each subsequent trial (as for the SRT). The trial needs to be repeated on each leg (SOLEC-L and SOLEC-R).[11,15.]

It has also been suggested that while the SRT is one of the many available quantifiable, clinical, vestibular function tests, that it has no greater value than the other tests in this group.[18] Again, no trial has been conducted to test this hypothesis.

The SRT has been shown to worsen with increasing age.[3,4,10,11,17] This decline in scores begins after age 40 in males and 30 in females.[11,13] Explanations for this phenomenon include: ageing of the peripheral nervous system causing an impairment of proprioception; a decrease in muscle mass; a decrease in muscle strength; a decrease in exercise levels and fitness; and an increase in the use of prescription medications by the elderly that effect balance and co-ordination when standing.[3,13,17] The reason for the differences between males and females is unknown and may not be real, as the numbers of females tested have been small.

A common criticism of the SRT is that there is a practice effect. This is controversial; one study supported this claim,[16] another disputed the argument[13] and a third study found that while this was the case in controls, it was not true for those with disease (labyrinthine disorders).[11]

Similarly, claims of dominance effects are not supported by a study that showed that there was no difference in performance when using the left foot in front of the right or vice versa and that there was no difference in performing the test with flat shoes on or in bare feet.[3]

The purpose of this study is to examine the SRT by analysing data from a control group of subjects performing the test and from divers with DCI.

Method

Two surveys were conducted. The first was a prospective review of the SRT in 60 control subjects. The second was a retrospective review of the last 35 cases of DCI presenting to the Royal New Zealand Naval Hospital (RNZNH) during 1994 and 1995. Individuals in each group were surveyed to ensure that they did not have any medical problems which could influence the test. Exclusion criteria included: disorders of the posterior column of the spinal cord, vestibular impairment, history of inner ear trauma or damage, unilateral or bilateral motor weakness, diseases of the peripheral nerves, cerebellar disease, vertebrobasilar disease, acute pain of the lower back or lower limbs, back surgery, and either a significant back injury resulting in persisting and current symptoms or a history of balance difficulties. In this study, patients were advised not to practise the SRT.

Subjects

The control group consisted of volunteers from HMNZS PHILOMEL (Navy and civilian) and Naval personnel presenting for routine medical examinations at the Royal New Zealand Naval Hospital (RNZNH). This population was chosen as reasonable age and sex matching was likely given the similar age and gender distribution in the Naval population and the divers treated at RNZNH for DCI.[21] Two patients were excluded from this group due to a past medical history of significant back injury.

The study group were divers treated at RNZNH for DCI. One diver was excluded after it was discovered that he a five year history of vertigo after an episode of inner ear barotrauma. Three other patients were not able to be included because the duty medical officer either failed to measure or did not record the SRT before hyperbaric treatment (these patients had normal scores on discharge).

The data for the divers and controls were compared using unpaired t-tests, assuming equal variance. The SRT scores for divers before hyperbaric treatment and at discharge were compared by paired t-test. A significance level of $p=0.05$ was chosen.

Results

The data for the control group are displayed in Table 1. The data for the divers with DCI are displayed in three tables. Table 2 shows the data at presentation and Table 3 the data at discharge. Table 4 shows the data for only those divers whose SRT was less than 30 seconds at presentation. The SRT mean value is the group mean of the cumulative trial scores. The best mean value cited is the group mean calculated from a subject's best individual trial score, i.e. the best score taken from the four trials.

TABLE 1.

SHARPENED ROMBERG TEST SCORE IN CONTROL SUBJECTS.

	Number	SRT mean	SD	Best [mean]	Best [SD]
Total	60	210.8	47.30	59.1	5.55
20-40 years	54	215.65	39.26	59.78	1.38
Over 40 years	6	167.17	86.74	53	17.15
Male	42	218.93	35.46	60	0
Female	18	191.83	64.72	57	10.02

TABLE 2.

SHARPENED ROMBERG TEST SCORE IN DIVING PATIENTS, AT PRESENTATION.

	Number	SRT mean	SD	Best [mean]	Best [SD]
Total	35	114.17	104.48	32.37	27.00
20-40 years	26	146.35	100.94	40.88	25.24
Over 40 years	9	21.22	38.29	7.78	13.88
Male	27	111.63	106.17	31.52	27.35
Female	8	122.75	105.10	35.25	27.42

TABLE 3

SHARPENED ROMBERG TEST SCORE IN DIVING PATIENTS AT DISCHARGE.

	Number	SRT mean	SD	Best [mean]	Best [SD]
Total	35	201	63.98	54.91	13.53
20-40 years	16	216.96	41.75	58.27	6.12
Over 40 years	9	154.89	93.44	45.22	22.78
Male	27	196.67	69.74	53.70	15.19
Female	8	215.62	38.76	59	2.83

TABLE 4

SHARPENED ROMBERG TEST SCORE IN DIVING PATIENTS WITH AN ABNORMAL RESULT (LESS THAN 30 SECONDS) BEFORE HYPERBARIC TREATMENT [HBO], AND AT DISCHARGE.

	Number	Pre-HBO Best [mean]	Pre-HBO Best [SD]	Discharge Best [mean]	Discharge Best [SD]
Total	17	5.94	7.93	50.88	18.01
20-40 years	9	8.11	9.31	57.56	7.33
Over 40 years	8				

The controls were aged between 18 and 49 years (mean = 28.93 ± 0.98 SD). There were 42 males aged between 19 and 47 (mean = 28.98 ± 1.09 SD) and 18 females aged between 19 and 49 (mean = 28.93 ± 2.09 SD).

The patients with DCI were aged between 17 and 53 (mean = 32.66 ± 1.68S SD). There were 27 males aged between 17 and 53 (mean = 34.89 ± 1.80 SD) and 8 females aged from 17 to 49 years (mean = 25.12 ± 3.57 SD). The mean age of the divers was not statistically different from that of the controls.

The time from the accident to the diver's presentation at the Naval Hospital (and hence assessment) varied from almost immediately after completing the dive to 5 days after diving (mean = 1.59 days ± 1.47 SD).

The group mean of cumulative trial SRT scores for controls was 210.8 (out of 240) ± 47.30 SD. The cumulative score mean for divers on presentation was 114.17 ± 104.48 SD. At discharge, this mean had improved to 201 ± 63.98 SD. The control group mean for their SRT best score was 59.1 (out of 60) ± 5.55 SD. The divers best score mean was 32.37 ± 27.00 SD, at presentation. At discharge, this mean had improved to 54.9 ± 13.53 SD.

A SRT best score at presentation of less than 30 seconds was recorded in 17 (48.57%) divers in this trial. The mean best score in the remaining 18 divers was not statistically different from that of the control group (mean = 57.33 ± 1.51 SD). That is, the best SRT score in divers was either indistinguishable from normal (controls) or less than half the maximum score (mean = 5.94 ± 8.1 SD).

Divers with DCI were almost equally distributed between the two groups (18 and 17 respectively). The mean SRT best score at presentation for the divers was statistically different from the controls ($p < 0.001$). The mean best SRT score at discharge was still significantly less than that of the controls ($p < 0.05$). However, the divers mean best presentation SRT score was significantly different from their mean best discharge score ($p < 0.001$). Amongst the 17 divers with DCI who had a best SRT score less than 30 seconds, 2 had little or no improvement, a

further 3 had improvement to greater than 30 seconds, but considerably less than 60 seconds, while the remaining 12 improved to record essentially perfect best SRT scores.

The difference between divers with DCI at discharge and controls was age related. The mean best SRT score data in divers at discharge was not significantly different from that of the controls, if divers and controls aged 40 years or less were considered only.

Discussion

The results of this study indicate that there are changes induced in the balance system in DCI, which are reflected in the best score with SRT. These changes could involve any or all of the components which contribute towards equilibrium.[5-13,18] The SRT is a quantifiable method of measuring these changes and is consequently useful in the assessment of DCI. It is even possible that the SRT could be used as a "marker" for DCI. The SRT was abnormal in 49% of patients with DCI in this trial. Thus for a patient where the disease process was in question, an abnormal SRT could be used to support a diagnosis of DCI. Grouped scores of the cumulative SRT were less discriminatory and had less value for comparison than the best trial SRT. With the cumulative score, some patients would repeatedly score approximately the same time for each trial. However, the majority have a wide range of scores. The cumulative score had a wider standard deviation. The best trial SRT score provided an isolated view of a patient's ability, and was directly comparable with the results of other divers. It was a more discriminatory measurement.

The data presented here suggest that a score of 48 or greater on a single SRT trial is normal (i.e. mean of controls ± 2 SD). In contrast, previous reports variously state that: a score of better than 30 seconds on any one attempt is normal;[3,12] a single score of 15 seconds is normal;[18] and a score of less than 15 is abnormal, when assessing patients with vestibular dysfunction and major abnormalities of equilibrium.[11,14]

The mean best SRT score for the divers with DCI was just higher than 30 seconds. However, this population was heterogeneous and consisted of almost equal sub-groups of divers with either an essentially perfect best SRT score (mean = 57.33 ± 1.51 SD) or a group with a mean best SRT value of only 5.94 seconds (SD = 7.93).

This suggests that an abnormal result should be regarded as any best SRT score of less than 22 seconds (5.94 + 2 SD). Of the 17 divers with DCI who had a best SRT score of less than 30 seconds, only 5 recorded 15 seconds or better. The other 12 scored less than 10 seconds and 11 were less than 5 seconds. This heterogeneity has been noticed previously amongst divers with DCI.[18]

From these data, and assuming that 99% of patients with an "abnormal" SRT score would be found from the mean of the scores plus three standard deviations, it is proposed that any score of less than 30 seconds be considered abnormal.

The SRT should be performed in the method described in Appendix B to achieve consistency. It is recommended that the test be scored as the best time out of 60 seconds (i.e. the score from the best trial of four). The best SRT score provides a readily comparable value. It also permits assessment of whether it is a normal result.

Divers aged between 20 and 40 years who demonstrated an abnormal initial best SRT score, had a discharge score which was not statistically different from that achieved by the control group. This suggests that the divers' posture had returned to normal and that age may be a risk factor for long term invalidity in DCI. Given that the entire group of divers with DCI showed a mean improvement from presentation to discharge, it can also be argued that the SRT is often a sensitive measure of clinical progress in divers with DCI.

It would be valuable to compare patients with DCI with their dive buddies. The buddies would provide an excellent control group, usually being age, sex and fitness level matched, and having dived the same or similar dive profiles without developing illness. This could provide information on whether the abnormal SRT in divers was due to DCI or was a reflection of decompression stress.

In summary, the SRT is a useful and sensitive test of equilibrium and is especially useful in the assessment of divers. It may even provide a diagnostic "marker" for DCI. It is recommended that the test be performed in the standard format given earlier, recording the best trial time of four attempts aiming at a time of 60 seconds.

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DOPPLER BUBBLE DETECTION AFTER HYPERBARIC EXPOSURE

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Abstract

A review of the literature on the use of transcutaneous Doppler to detect circulating venous bubbles occurring after hyperbaric exposure, with emphasis on the detection of bubbles occurring after relatively small decrements in pressure, is presented. The correlation between circulating bubbles and the occurrence of decompression illness is examined.

Key Words

Bubbles, decompression illness, investigations.

Introduction

Decompression illness (DCI) may occur in many different organisms following a reduction in ambient pressure exposure. The illness is thought to develop as a result of the formation of an endogenous gas phase taking the form of small inert gas bubbles which are widespread throughout the blood and body tissues.[1] The symptoms produced by these bubbles will depend on their size, number and location. Gas bubbles in the microcirculation or moving in the venous circulation may apparently produce no clinical symptoms, whereas a bubble of similar size in the tissues may produce symptoms due to tissue distortion or damage, especially in the nervous system.[2,3] Circulating venous bubbles indicate that gas phase separation has occurred, and that bubbles may exist elsewhere in the body tissues.[4] The growth of gas bubbles in the tissues by gaseous diffusion may produce symptoms some time after the initial decompression has occurred.[2,3] Gas bubbles normally appear and grow after decompression. Rapid decompression or a large gas load, or both, leads to the earlier appearance of bubbles.

The magnitude of the decrement in pressure exposure which can be safely tolerated by humans is of fundamental importance in the field of hyperbaric medicine, where patients and attendants alike are exposed to elevated ambient pressure during routine treatment profiles. The treatment regimes currently in use throughout the world are considered to be safe in that the incidence of DCI in the attendants is negligible, though not zero. However, it is not known if small asymptomatic bubbles may be occurring during these exposures which may cause morbidity in the long term, especially where the attendants have repeated hyperbaric exposure (in some centres more than once daily).

At present, there is no reliable way to detect stationary bubbles located in the body tissues, and although pulse-echo ultrasonic imaging has been proposed for this purpose based on in-vitro studies, it has not gained widespread use in-vivo as it is only possible to examine one region of the body (eg. one limb), the subject must be totally still, and the equipment is large and bulky.[5,6] However, circulating bubbles can be relatively easily detected in the venous and arterial circulation using Doppler ultrasound. This provides a means for assessing whether gas-phase separation has occurred during decompression.

This paper examines the current understanding regarding the use of Doppler ultrasound to detect circulating bubbles and considers their relevance to DCI.

Classification of decompression illness

Interpretation of the studies carried out to date attempting to correlate the incidence of decompression illness to Doppler-detected bubble grade is made difficult by the different classifications of decompression illness used in different establishments. For the purpose of this review, the recent classification developed by the Royal Navy Institute of Naval Medicine workshop at Alverstoke, United Kingdom, in October 1990, will be used.[7] This classification is based on a clinical description of the illness. The term "decompression illness" is used to include both the previous "decompression sickness" and "cerebral arterial gas embolism". The term is prefaced by an evolutionary term (static, resolving, relapsing, progressive) and secondly by the organ system involved, with no attempt to grade the symptoms into a severity hierarchy. For example, a diver who collapsed on surfacing, was initially unconscious and then recovered, would have " resolving neurological decompression illness" and a diver with unchanging shortness of breath and paraplegia would have "static pulmonary and neurological decompression illness".

This new system does not try to classify the decompression illnesses into arbitrary grades of severity as with the previous unsatisfactory "decompression sickness" (DCS) Types I and II.[8] By describing the illness in clinical terms it is less open to variation in interpretation between observers.

Much of the published literature predates this new classification, and previous investigators have used the old Type I and II classification of DCS, with Type I being "mild", Type II being "serious" and many cases not fitting into either Type described as "unclassified".[2,8] This makes interpretation of the available data more difficult, as many symptoms and signs which would now be recognised as manifestations of decompression illness (such as neuropsychiatric changes) were not sought because they did not fit easily into the existing classification. Although more subtle deficits in higher mental function are formally included in the category of "Type II DCS" as manifestations of neurological DCS,[8] they have not been specifically sought in most studies prior to the reclassification.[9-12] This may indicate that cases of DCI were not diagnosed as such unless there were obvious symptoms, such as joint pain, and that many cases of a more subtle nature were overlooked.[4,13,14]

Thus in interpreting the results of studies to date, this possibility should be borne in mind. There is no retrospective way to ascertain if cases of DCI were overlooked. The reported incidence of DCI may therefore be an underestimate.

Doppler ultrasound in hyperbaric medicine

Behnke, in 1942, was the first to propose that silent (asymptomatic) bubble formation occurred during rapid ascent to altitudes of 6,000-8,000 m.[15] The development of medical ultrasound led to the availability of Doppler ultrasound detectors. Spencer and colleagues first detected decompression gas emboli in the arterial and venous blood of sheep following decompression from a 65 m 60 minute exposure to air and venous bubbles were shown to occur before the first stop recommended by the US Navy Exceptional Exposure Tables.[16]

At first it was difficult to detect bubbles in human subjects because the peripheral Doppler detectors could only be located over peripheral veins, and hence only a portion of the venous return was being monitored. A catheter-tip Doppler was developed for implantation into the vessels of humans, but did not gain wide acceptance due to its invasive nature.[16]

The first unequivocal bubble signals were detected by Spencer in 1969 by an external sensor placed over the brachial vein of a subject with DCI involving symptoms in the same arm. These were enhanced by local tissue manipulation, and disappeared after treatment of the DCI with recompression.[16] This finding established that Doppler ultrasound could be used during recompression treatment to optimise both the necessary extent of recompression and subsequent decompression schedule on the basis of the frequency of vascular gas bubbles detected.

It was realised at this time that improved bubble detection could be achieved if a sensor could be developed to detect bubbles flowing in the right ventricular outflow tract and pulmonary artery. The first precordial ultrasonic bubble detector was developed by Spencer et al. in 1971.[17] It incorporated a transducer which focused deeply on the large vessels and heart, thus eliminating confusion from gas emboli signals in the blood vessels of the chest wall itself.

The Doppler ultrasound probes now in common use for the detection of circulating bubbles emit 2.5 MHz continuous-wave signals using two probes of different focal length designed for use on the precordium and the peripheral vessels. The signals obtained can be recorded directly onto audio tape for later analysis.

Clinical significance of Doppler-detected bubbles

Although the ideal decompression would result in no bubble formation, it appears that some degree of gas phase formation occurs in virtually any decompression, as Doppler-detectable bubbles are found in all decompression profiles from short bounce dives to slow, saturation-dive decompressions.[1,13,15] Many of these cases have no symptoms of DCI despite the presence of significant bubbling, which throws doubt on the premise that the presence of detectable venous bubbles equals decompression illness.

Bubble formation not only alters gas elimination rates, but also may lead to DCI.[12] However, any gas remaining in solution following decompression will not of itself produce damaging effects.[15]

Following decompression, bubbles will form early in those tissues most saturated with nitrogen, and as they enlarge and exert greater pressure on the blood vessel walls, these bubbles may enter small veins and capillaries. Spontaneous intravascular bubbles tend to form preferentially in the venous circulation as the blood here is quickly saturated with gas released from the tissues at the capillary level, and the intravascular pressure is relatively low.[2] Bubbles also form early in the microcirculation following a reduction in ambient pressure due to the high gas tension and low intravascular pressure.[1,14] Bubbles located in the microcirculation are initially stationary and may cause tissue hypoxia due to obstruction of blood flow.[2,10] They may eventually embolise to the central venous circulation due to pressure or distortion of the tissue. Bubbles entering the systemic venous circulation are filtered by the lungs up to a maximal bubble load, after which they may completely obstruct pulmonary flow.[1,15,18] It is rare to find bubbles circulating in the arterial circulation, unless there is a right to left shunt present in the pulmonary bed or heart; but if the delivery rate of bubbles to the lungs exceeds a threshold level (0.35 ml/kg/min in dogs),[19,20] some may pass across the pulmonary bed and enter the arterial circulation.[2,3,16] Turbulent flow in the ventricles of the heart may also predispose to cavitation and bubble formation on both the right and left sides of the heart.[21]

The pathophysiology of DCI is thought to be complex and to involve both intravascular and extravascular bubbles, complement activation, histochemical and haematological changes,[3,22,23] so detection of intravascular bubbles is only an indicator that a decompression stress has occurred, not necessarily that clinical DCI will occur.[23] Susceptibility to DCI appears to be an individual trait, as some people will develop DCI with low-grade or no detectable bubbles, whereas others have higher-grade bubbles with no clinical evidence of DCI.[11,12,24,25]

Doppler-detected bubbles are not normally believed to be the cause of DCI, but their presence in the circulation indicates that asymptomatic bubbles may be present in other tissues of the body.[10,11,25] These asymptomatic tissue bubbles may cause subclinical damage which may have long-term effects, for example in the central nervous system.[4,25-28]

Doppler ultrasound mechanisms

Ultrasonic waves have the advantages of being highly directional, easily focused, useful for examining small structures due to their short wavelengths, and they show a large reflection at gas-liquid interfaces, making them very good gas phase detectors.[17] These properties make ultrasound waves ideal for detection of circulating (but not stationary) gas bubbles.[4,29]

The presence of gas bubbles in a liquid causes marked reflection of an ultrasound beam. In addition, bubbles of a given size are found to pulsate under the influence of periodic oscillations of the surrounding medium. This has been proposed as the mechanism for the sound generated by running water in streams.[15]

The transducer is the most important element in the Doppler ultrasound system, determining the operating frequency, the depth of penetration, the size of the ultrasonic beam and the frequency of the Doppler shift recorded.[17] The central element of the transducer is a piezo-electric crystal which changes its dimensions when an electric field is applied to it, or conversely will generate an electric field when it is deformed by vibration. The most sensitive frequency for operation of a transducer is the fundamental resonance frequency of the crystal element. Lower frequencies penetrate deeper into tissues, but result in longer wavelengths and low Doppler shift frequencies. Conversely, too high a frequency will not penetrate deeply enough and will produce Doppler shift frequencies too high to hear. Most Doppler systems use frequencies between 2.5 and 5 MHz, the lower frequencies being used to penetrate to the heart and great vessels, and the higher frequencies being used for more peripheral vessels.[15,17,30]

The Doppler effect is a change in the frequency of an ultrasonic wave when the transmitter, the receiver, or the scatterer are moving with respect to each other. Therefore, reflections from moving gas bubbles have a different frequency from the transmitted frequency. Ultrasonic

Doppler flowmeters only respond to reflections which have experienced a Doppler shift (eg, from moving bubbles in blood) and not to the reflections from stationary structures which have no Doppler shift. The Doppler shift frequency produced by moving gas bubbles is within the normal range for human hearing. The receiver has two inputs, the transmitted wave and the reflected wave. The output frequency of the receiver is the difference between the two inputs, so that there is only an output for the reflections from moving structures.[15]

Doppler signals coming from the receiver can be analysed aurally or displayed graphically in the form of flow velocity waveforms.[31,32] There are several schemes for aural processing in current usage, the most common being those of Spencer and Johanson[9,15] and Kisman and Masurel.[33,34]

The Doppler signal produced by a gas bubble in blood is a sinusoidal narrow band sound wave which sounds to the human ear like a chirp or whistle.[17] The amplitude of the bubble signal is nearly proportional to the radius of the bubble.[30] Due to their strong acoustic interface, bubbles in the blood and other liquids tend to scatter ultrasound more than do solid particles of the same size. The human ear is currently the most accurate signal processor for recognition of the bubble sounds, because the hearing mechanism can distinguish the chirping quality signals at low volumes superimposed on the background noise of Doppler blood flow, and can recognise the extra noises which occur sporadically as a break in the pattern of normal cardiac signals.[15,35]

Doppler bubble detectors can be either continuous wave (CW) or pulsed systems. The CW system is technologically simpler and produces an output in the audio frequency range which conveys both amplitude and frequency information without the need for quantitative calibrations. The pulsed Doppler allows “range gating” to select the penetration depth of the transmitted and received signal which narrows the sample volume and reduces background signals. However, the electronics required are currently complex and expensive and therefore most work has been done using CW Doppler.[6]

Gas bubbles present in the circulation are detectable as long as they are above the resonant size for the ultrasound wave. The minimum detectable bubble size is a function of blood velocity.[15] A bubble of 20 micrometers radius will be detected if the mean velocity carrying it is 55 cm/sec, but the minimum detectable size increases to 90 micrometers if the velocity is only 20 cm/sec. The smallest bubble detectable in the heart and large vessels should be approximately 80 micrometers in diameter, and in the subclavian veins, 10 micrometers.[11,15,30] Hence, bubbles can be easily detected in peripheral veins when they are not detected at the same time by precordial devices.

Nishi determined that small bubbles may not scatter enough ultrasound energy to be detectable above the background noise.[30] He maintained that although it is not possible to deduce the size of the bubbles in vivo, acoustical studies suggest that they must be about 50 micrometers in radius or larger for detection. Hills and Butler[36] measured the size distribution of intravascular bubbles produced by decompression and found diameters of 19-700 micrometers, so the smaller of these bubbles may not be detected by Doppler. Therefore, it is not possible to prove the absence of bubbles, as there may be smaller bubbles circulating which are not detected by the current Dopplers.

Safety considerations

High energy sound waves may affect tissues by several mechanisms. However, no observable effects have been seen to date in intact mammalian tissues from the amounts of power used in medical diagnostic and bubble detection equipment.[15] The intensity of most diagnostic ultrasound equipment ranges from 10 to 100 mWatts/cm^2. Tissue damage does not occur until the intensity reaches 1-100 W/cm^2. Thermal burns may be produced at energy levels higher than 100 W/cm^2.[15]

Also, low pressure areas in the sound wave may produce tearing in liquids, a process which leads to the formation of small cavities in the liquid.[15] This also may result in free radical production and a variety of chemical and biological effects. Theoretical predictions indicate that clinical ultrasound energy levels cannot produce cavitation in the megahertz frequency range. Cavitation has not been demonstrated at frequencies much over 5 MHz. In liquids of low viscosity (eg blood), operation of higher frequency ultrasound in short bursts prevents such cavitation.[15,17] Attenuation of the ultrasonic energy as it penetrates deeper into the tissues provides an additional margin of safety. No untoward effects have been observed in the monitoring of animals or humans by application of the Doppler detector.

Precordial monitoring

The precordial probe is placed at the left midsternal edge, where it overlies the right ventricle and pulmonary artery, and should be well placed to detect any bubbles in the venous circulation.[34] The heart sounds are very loud and clear at this point. Gas bubbles are heard as a “click”, “chirp”, “whistle”, or as a harsher, longer sound depending on their velocity and the angle at which each bubble crosses the ultrasound beam. These sounds are superimposed on the regular heart sounds as sporadic, irregular sounds of different frequency. The bubbles are graded according to the method of Kisman and Masurel (K-M).[33,34]

Monitoring is carried out for 60 seconds at rest (with the patient standing upright) and then for 30 seconds after each of three deep knee bends, the aim being to mobilise bubbles from the tissues with muscle contraction and increase the detection rate.

Monitoring the heart can be difficult as the valvular movement and any turbulent blood flow produces a loud background noise that may mask bubble signals. For this reason, monitoring is also carried out at peripheral sites where the level of background noise is minimal.

Peripheral monitoring

Veins such as the internal jugular, subclavian, inferior vena cava or femoral can be monitored, as bubble signals at these sites are unambiguous. However, they are monitored only as a supplement and not as an alternative to the precordial site, because they do not provide information from the whole body, only the region which they drain.[17]

The probe can be placed over a more peripheral vein such as the subclavian vein at the midpoint of the clavicle, or the femoral vein in the inguinal crease. The aim at these sites is to monitor the soft blowing sound of the venous flow, with the regular arterial pulse superimposed to facilitate bubble grading. A clench of the fist, or contraction of the calf muscles may release a shower of bubbles. The bubble noises are more easily heard at these sites, where there are no cardiac sounds to interfere with detection.[34]

The K-M classification is based on three parameters used to describe the bubble signal; frequency, percentage or duration, and amplitude.[34] Each parameter is assigned a value from 0-4. The frequency represents the number of bubbles per cardiac period, the percentage represents the percentage of cardiac periods at rest having a specified bubble frequency. After movement this is replaced with a duration parameter, which describes the number of cardiac periods following the movement which have a given bubble frequency. The amplitude is graded compared with the amplitude of the cardiac signal. The code is then written as a three digit number, and this is converted to an overall bubble grade I-IV using a table. The correlation of bubble grade between different observers is dependent on the experience of the respective observers.[35]

Despite numerous technical refinements over the last 15 years, the process of Doppler bubble scoring has remained essentially unchanged, with auditory processing by trained observers remaining the mainstay of coding. Butler et al. reported the use of computer-assisted digitisation to provide real-time and replay recordings of Doppler-detected bubbles in both dogs and human subjects, using both the precordial and subclavian sites.[31] They found that the production of visual representation of the Doppler signals by computer digitisation was a valuable aid to the audio-interpretation of the signals, especially when the observers are inexperienced or fatigued. It may also allow cardiac background signals to be more easily distinguished from bubble sounds.

Since it is not practical to monitor divers continuously, Doppler recordings are taken at intervals after the return to the surface. Current information suggests that observations should begin soon after return to the surface and be repeated at half-hour intervals for at least 2 hours, as the peak incidence of bubble formation appears to be between 1-2 hours after the decompression stress.[4,17,37]

Clinical correlation with bubble grade

Many studies have been performed in recent times in an attempt to determine the relationship between circulating bubbles and the clinical onset of DCI. To date, attempts to use Doppler detection methods to guide decompression in human divers have been sporadic and the scattered results difficult to analyse.

Initially, it was hoped that Doppler could be used to control the decompression profiles to prevent DCI, but the studies performed by Eatock and Nishi showed that bubbles tend to form after the decompression has been completed, first appearing up to 1 hour after the decompression stress, with the peak incidence at 2 hours after decompression.[37]

In general, the bubbles are detected before the onset of symptoms of DCI and it is almost certain that decompression-produced bubbles are produced after almost any period of compression, even in dives which produce no symptoms of DCI.

Attention is now focused on trying to predict the probability of development of DCI from bubble frequency.

Spencer studied a group of divers over a range of pressure-time air exposures with Doppler ultrasound in an attempt to determine the direct decompression (decompression to 1 ATA at 60 ft/minute, a rate of 0.3 m/sec) limits for man.[11] He found that there appeared to be a strong individual propensity to form circulating bubbles or venous gas emboli (VGE), which correlated with susceptibility to DCI. No DCI developed without prior detection of precordial VGE. VGE were even found to occur after exposures to pressures of as low as 2 ATA (10 m, 33 ft). After a 60 minute exposure at 2.8 ATA, 4 of 12 divers developed detectable VGE of grade II or greater on direct decompression, although 9 of 12 divers reported skin itching. Only one diver developed limb pains. Spencer found a trend for bubbles to be detected in the venous circulation sooner after a deep dive than after a shallow dive.

For example, after a dive to 1.9 ATA, bubbles were detected in the circulation 15-30 minutes after decompression, whereas after a 7.5 ATA dive, the bubbles were detected after 6-12 minutes.

Neuman et al. studied a group of US Navy personnel during routine hyperbaric chamber operations.[12] Thirty one dives were performed to either 65 msw or 40 msw. There were 5 cases of DCI, all associated with Doppler bubble counts of grade IV. However, Doppler bubble counts of grade IV were also recorded in 12 of the other 27 divers, none of whom had symptoms of DCI.

Powell et al., in 150 man-dives, found that precordially detected bubbles were predictive for limb pain in mixed-gas divers only 50% of the time, but 70% of the divers who developed clinical DCI had no precordially detectable bubbles.[24] They concluded that the presence of venous gas bubbles can be associated with an increased risk of DCI, but that this technique lacks the specificity required for personal dive monitoring.

Eatock collected data from Defence and Civil Institute of Environmental Medicine (DCIEM) and US Navy dives and correlated Doppler bubble grade with probability of developing DCI.[25] Many different dive profiles were used, including mixed-gas diving. In general, bubbles of grade I or less were associated with clinical symptoms of DCI in less than 2% of cases, with an increasing incidence of DCI for grades II-IV. Bubbles of grade IV were associated with a DCI incidence of 50%. However, the author concluded that although the number of intravascular bubbles is a good indicator of decompression stress, the correlation between bubble count and DCI is subject-dependent. Though the presence of bubbles does not necessarily produce symptoms, the possibility of long-term effects cannot be ruled out.

Bayne et al. carried out a double-blind, prospective clinical trial of Doppler bubble detection in simulated diving involving 83 men, of whom 8 developed DCI.[10] They found that diagnosis based only on the Doppler signals had no correlation with clinical diagnosis, but that bubble scores were slightly higher in the DCI group. However, 3 of the 8 divers with clinical DCI had no detectable precordial bubbles, constituting a false negative rate of 38%.

Eckenhoff et al. studied 34 healthy human subjects exposed to shallow air saturation for 48 hours at 1.77 ATA and 1.89 ATA, and then decompressed to 1 ATA in about 2 minutes.[13] Almost all subjects had Doppler-detectable VGE developing up to 4 hours and lasting for up to 12 hours after decompression. The reported incidence of DCI was 27% in the 1.89 ATA group, this being manifested by joint pains only. The incidence of other cases of questionable DCI was approximately 20% in each group, although the symptoms experienced by the individuals in this group were not described. It is interesting to note that the incidence of other symptoms (apart from joint pain) were higher than the reported DCI incidence. Fatigue was reported by 53% of divers, malaise by 26%, myalgias by 15%, headache by 15% and pruritus by 9%. All of these would be classified as symptoms of decompression illness by the new classification, so it is possible that the true incidence of DCI in this study may have been as high as 53%. It is also interesting that those subjects treated for DCI in this study had rapid and complete disappearance of VGE during treatment (USN Table 5), but in an average of 4 hours later, low-grade (grade I) VGE recurred in 3 out of 4 treated subjects. The recurrence of bubbles after a treatment table may indicate that the table is inadequate for treatment, and may correspond with the recurrence of symptoms often seen after the initial treatment of a diver with DCI.

Dunford et al. showed that intravascular bubbles can be detected with Doppler ultrasonic bubble detectors in up to 18% of recreational divers after dive profiles said not to require decompression stops.[38]

Eckenhoff et al. in a study of divers in an underwater habitat determined that 50% of humans can be expected to generate endogenous bubbles after decompression from a steady-state pressure exposure of only 1.35 ATA.[1] VGE were first detected within 1 hour of the decompression and continued on average for 4 hours. None of the subjects developed symptoms of DCI, not even the fatigue or pruritus seen in his earlier study.[13] He comments that substantial formation of an endogenous gas phase can occur without symptoms, although it remains to be proven that asymptomatic VGE after a decompression stress are indeed benign. His data suggested that the incidence of DCI after exposures of 1.38-1.64 ATA was <5%. He concluded that symptoms of DCI will be reported mostly in subjects with Doppler bubble grades greater than grade 3. He also comments that the reduction in absolute pressure of 26% (1.38 ATA to 1 ATA) experienced by the subjects in this study is equivalent to that experienced in ascending from 1ATA to an altitude of 2,500m, which is a pressure commonly attained in commercial pressurised aircraft, and that circulating bubbles may well be a common occurrence in people subjected to this decompression stress.

Sawatzky and Nishi examined the relationship between Doppler detected intravascular bubbles and the subsequent development of DCI in 1,726 dives.[14] They found a definite association between increasing Doppler grade and risk of DCI. Grade II or less bubbles were associated with only a 1-2% incidence of DCI, and Grades III and IV with a 6-11 % incidence of DCI. The maximum recorded Doppler grade was the best indicator for the risk of developing DCI, 90% of cases of DCI having detectable bubbles of grades III or IV. These results indicate that Doppler grade is not a good predictor of which individual subject will develop DCI, but allows an assessment of the risk of DCI to be made, based on Doppler bubble grade. They

concluded that it is necessary for intravascular bubbles to be present for DCI to develop.

The studies performed to date indicate that bubbles of less than grade II are generally not associated with articular pain, but with increasing bubble grade, the incidence of clinical DCI increases. It appears that bubbles may be a precursor of DCI and can therefore be used as an indicator end-point rather than relying on pain or other systemic disturbances for assessing the efficacy of decompression procedures. The Defence and Civil Institute of Environmental Medicine in Canada, where much Doppler research has been carried out on decompression tables, have adopted the policy that dive profiles which produce peak Doppler-detectable bubbles of Grade II or greater in 50% or more of the subjects, probably constitute an unacceptable decompression stress.[4] Unfortunately, cases of DCI still occur with no detectable circulating bubbles, so the policy is only a guide. Also there is great individual variation in the number of bubbles produced between different divers carrying out the same decompression profiles, and in the same divers carrying out the same decompression profiles on different days. Divers who are fatigued before a dive produce more bubbles and have a greater risk of DCI.[6] Other factors known to predispose to DCI include state of hydration, obesity, age, level of stress and infection,[3] and it is likely that these factors may be associated with the production of more circulating bubbles.

In saturation diving on helium breathing mixtures, the correlation between DCI and Doppler-detected bubbles appears to be different from that seen in non-saturation dives. There is a greater incidence of DCI at all bubble grades to the extent that some corrective action has been recommended to modify the decompression schedule in helium dives if the detected bubble grade is greater than grade II.[6]

It must be remembered that in the studies quoted here, which were performed largely in the USA and Canada, the definition of DCI differs from that now in use in Australia.[7,8,14] Joint pain has been required for the clinical diagnosis of DCI until recent times. It is now widely recognised that more subtle effects such as neuropsychological changes and concentration deficits are also symptoms of DCI. One wonders whether the investigators involved missed an appreciable number of subjects with significant DCI in the studies carried out to date, because the subjects had no joint pain. It could be that bubbles of the lower K-M grades may be pathogenetically significant and cause more subtle changes than previously thought.

Summary

The hope that Doppler-detected bubbles could be used as a diagnostic aid for DCI arose from the belief that circulating bubbles caused symptoms of DCI. The current evidence suggests that this is not true. However, circulating bubbles indicate a decompression stress has occurred and may indirectly point to the presence of bubbles elsewhere in the body tissues.[6] Intravascular bubbles may also cause subclinical damage which may have long term effects.[13]

It is certainly clear that circulating bubbles are formed even after decompression from very shallow depths and, although these low-grade bubbles are dismissed as non-significant by the North American authors, it is not known what the long term effect of repeated exposure to such circulating bubbles is. They may be implicated in the neuropsychological changes now being recognised in long-term divers.[4,25-28] Aggressive behaviour, inability to concentrate, poor memory and tiredness now form a symptom complex being increasingly recognised in long term divers, many of whom have never suffered an episode of clinical DCI. Also, dysbaric osteonecrosis occurs in many divers who have never had an episode of clinical DCI and may also be related to chronic exposure to VGE.[28]

The studies performed to date conclude that circulating venous bubbles of less than grade II are probably of little clinical significance, as they are associated with a very low incidence of DCI (<2%), but bubbles of grades III-IV are more significant, being associated with a higher incidence of clinical DCI. Although authorities such as the DCIEM accept bubbles of grade II or less as an acceptable risk in their dive tables and decompression schedules, on current evidence the validity of this assumption should be questioned.

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DIVING DOCTOR'S DIARY

SCUBA KIDS

Carl Edmonds

Case summary

(The full and excellent description of this case report, by the child/patient/diver herself, can be read in Scuba Diver)[1]

At the age of 12 years her father gave her a birthday present, a scuba diving course.

The family were very conscientious and ensured that she was examined by a diving doctor, who also performed lung function and provocation tests at the local hospital. I have not been able to track them down.

The qualification, which she received without difficulty, allowed her to dive in association with another certified diver. This she achieved by her father driving her many hours to the coast, every few months.

During one of these visits she did a dive to 6 m for 37 minutes, totally uneventful. Some 8 hours later, while travelling over the mountain range that surrounds Sydney, she became aware of a tingling in one knee. The usual plea from her father to tell him of anything that could possibly go wrong with a pain in the joint or something like that, was initially ignored but she finally did disclose the tingling sensation.

She was then starting to become a little apprehensive, made worse by reviewing a diving manual which described the symptoms of decompression sickness. At this stage both she and her father were panicking, she in tears and her father driving wildly to return to Sydney.

They contacted the Divers Emergency Service number on their mobile phone, and talked for some time to the doctor. By this stage, with the patient on the phone talking to DES, the whole situation deteriorated. The patient burst into tears and her father took the phone. She was shaking all over and very apprehensive. As she stated "I am going to die here in this car, on some ******* mountain".

The symptoms developed further and she complained of numbness and tingling in all extremities and an increased numbness in the leg.

They then came down from the mountain, but at this stage she was in a bad state with chest pains, difficulty breathing, blurred vision and tiredness etc. She was crying, dad was swearing.

By the time they got to a local teaching hospital, the whole family was close to tears. By then it was all a very big emergency. Whatever else was happening, she was hyperventilating and confused.

The ambulance, the paramedics and the intravenous drips all combined to deliver a live, but very distressed, patient to the Naval base.

Hyperbaric treatment was then given, on the very reasonable presumption and diagnosis of a cerebral arterial gas embolism from pulmonary barotrauma. The patient responded well to this, and there were few, in any, remaining symptoms during the subsequent hospitalisation.

The professional diving physician who saw her did strongly suggest that, because of her asthma (did I not mention that she took Pulmicort regularly?) that she should not continue scuba diving.

She was seen by the "best diving doctors in the state", who gave her and her parents a variety of advice, including references to "wild cat bends" and suggestions to wait until she is a little older before she resumes scuba diving. (This certainly does not say much for the standard of diving medicine in New South Wales.)

It was on the basis of the above report, which has been much abbreviated by me, but is in all its colour and glamour in the Scuba Diver article, that I prepared the following response for the magazine.

Official recommendations

The South Pacific Underwater Medicine Society committee on medical standards for recreational diving met on this subject in 1990. They recommended a minimum age of 16 for scuba diver training. The decision was based purely on safety factors.

The Australian Standards Committee CS/83 reduced the recommended age to 14, to comply with the diving instructor agency's requirements.

The Australian Standard 4005.1 of 1992 states that the selection criteria required that the trainee shall comply with the following:

(a) be at least 14 years of age.
BUT, persons under 14 years of age may in some cases be eligible to train for conditional certification which allows the young person to dive with a certified diver, with consent of parents or guardians.

Under the medical section of the AS 4005.1 it is stated "Children under the age of 16 shall only be medically examined after consultation by the doctor with the parent or guardian to establish the child's physical and psychological maturity. Between the ages of 16 and 18 it is preferable to consult with the parent or guardian before medically examining the child."

Would you allow a 12-14 year old child to:
fly an aeroplane?
drive a motor vehicle?
take out a loan?
be legally responsible for decisions made?
make medical and health judgments?
make life-threatening decisions for himself and others?

If you agree that 12 to 14 year old children should be restricted in this way, then it would be interesting to compare your attitude with that of a similar aged child undertaking scuba diving.

The youngest child that I know of to die as a result of scuba diving was aged seven, but there are quite a few 10 to 14 year olds in the death statistics associated with recreational diving.

The Australian Surf Life Saving Association, whose judgment is not influenced by commercial factors but who is very committed to children's involvement, will not allow active life saving responsibilities until after the age of 15 years, and even then under the supervision of a patrol leader, 18 years or older.

Comment on AS 4005.1

SPUMS recommends a minimum age of 16, with parental informed consent and approval necessary between the age of 16 and 18.

The Australian Standard unfortunately was not prepared by diving physicians. Although there was a representative of SPUMS on the committee, he was greatly outnumbered by the industry and diving training organisation representatives. They have different agendas, and different motivations from physicians. There was no one present with paediatric psychological training.

Looking at the Australian Standard document, it is implied that there is some concern regarding a child's safety, until the age 18. Off loading the responsibility to parents who have no practical knowledge of the risks of scuba diving was a reprehensible act.

I was, and am still, surprised that the committee accepted that an under 14 year old diver could safely "dive with a certified diver". This is clearly inadequate, as it allows one young "conditionally qualified" child to dive with another diver who may be equally inexperienced.

Psychological maturity

This is the main reason why doctors would prefer children not to be given diving certification.

Certification implies that the diver can make informed judgment about dive planning, environmental conditions, equipment use, and the interrelationships of all these. For a dive to be safe, this judgment is sometimes essential. It is related to maturity and experience, not just intelligence.

A child may have no difficulty handling the intellectual content of the diving course, but she or he will have difficulty with its application.

Unfortunately children do not have the same understanding of mortality (death) and the implications of morbidity (disease or accidents) as adults.

Kids are immature. That is what makes a child. They tend to be more immediate in their gratification needs and with a shorter attention span. They are not as good at long term planning as adults. Unfortunately, sometimes the long-term planning will not be needed if the child dies or does significant damage.

Materialistic factors also come into play. Kids are less likely to abort a dive, if they have already committed themselves financially or logistically. With age, judgment does come. Older people see death more clearly.

Psychological reactions are also different in children. Kids react with behaviour that, in adults, would be abnormal. They are far more likely to display anxiety or hysterical reactions, and the control of these is part of the maturation process.

The appropriate response to a life-threatening situation, or even one that is perceived to be life-threatening, is not to burst into tears. Unfortunately this is a child's natural reaction, and is often very successful in obtaining assistance. Tears are not easily seen through a face mask, and in any case, tend to simply add to the large ocean environment. They do not have the same power under water as they do on land, with mummy watching.

Children's reactions are certainly rapid, but not always appropriate for their long-term health and safety.

Endurance and perseverance are characteristics which develop with age. These take over when panic and tantrums have been controlled. Imagination is a characteristic that is endearing, but makes kids susceptible to fear and terror.

Dependency

Children are dependent. They slowly mature to become independent, and act responsibly. Thus they are more likely to rely on the statements and decisions of others, as opposed to deciding what they themselves are capable of doing. This might be all right on a trip to the zoo, but it is not good in open ocean diving.

In the latter environment divers have to be self-reliant and to recognise their own limitations, but also have to be able to act accordingly. They are responsible for the safety and rescue of their companions. Would you really want a twelve year old child being responsible for your safety, or your child's safety?

Children are suggestible and very easily impressed. They can be intimidated directly by their parents, and also by the encouragement and enthusiasm that their parents may give them.

Thus the child might well continue an activity such as scuba diving, to please mum or dad, to impress their parents and peers and to gain attention. These are not good motivations for scuba diving. Kids are very easily intimidated, and for the sake of the child, I would prefer to see an indifferent parental reaction than an enthusiastic one.

Physical immaturity

The problems of good quality equipment purchase include the need to upgrade regularly during the growing years.

There is the likely problem, sooner or later, of the child having to swim against unexpected tides or currents to return to safety. Some children may have this physical ability, but do not have the psychological endurance in such an emergency. Others will have neither.

A small child could have great difficulty in coping with the rescue of a larger "buddy".

With physical immaturity, there is also the problem of increased dangers from certain diving medical disorders. These include such things as hypothermia, gas toxicities, susceptibility to marine venoms, barotraumas, etc.

Medical aspects

The reason that children get "glue ears" is that their Eustachian tubes are narrower and smaller. So are their sinus ostia. So are their respiratory airways. That is the reason why children have far more trouble with barotraumas in aviation, as well as diving, exposures.

Some diseases, such as asthma, are more likely to occur in young children than in late adolescence, when the airways have grown relative to the lung volumes. That is why children sometimes seem to "grow out" of asthma.

Children's upper and lower respiratory passages are much narrower in comparison to the air cavities associated with them. Barotrauma is very common in children exposed to pressure changes.

Many have questioned the safety of exposing children to diseases such as pulmonary barotrauma and arterial gas embolism (one cause of acute decompression illness), especially in children where there is still growth of organs, i.e. where a bubble can do more damage than it would in a full-sized adult.

Such tissues that could be so affected include the brain, inner ear, bone, coronary artery etc. The worry here is that, for the same degree of bubble development, there could be a much greater ultimate damage.

When should children dive?

In my opinion, a child under the age of 16 should only have a "dive experiences" under the following, moderately safe, conditions:

1 They want to, without parental or peer pressure.

2 They are medically fit to do so.

3 A maximum depth of 9 m, to prevent some of the problems referred to above. The 9 m depth will certainly not prevent a child from developing pulmonary barotrauma, cerebral arterial gas embolism or any of the other respiratory tract barotraumas. It will, however, usually prevent decompression sickness manifestations unless the exposure time is excessive.

4 They are trained and taken by a qualified instructor, and under the personal and total control of that instructor (i.e. not three or four trainees together). A buddy line between the child and the instructor is prudent, to prevent panic ascents.

5 After this acquaintance dive, all other dives are only to be carried out in safe environmental conditions, and with the same controls as referred to above (1-4), with an experienced diver of instructor standard taking absolute control.

Giving a certificate to dive to children under the age of 16, other than one which stipulates diving under the above very special conditions is, in my opinion, irresponsible.

Reference

1 Haines S. Why? *Scuba Diver* 1996; July/August: 74-75

Key Words

Case report, decompression illness, fitness to dive, hyperventilation, panic, recreational diving, training.

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

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BREATH-HOLD DIVING
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to be held on 6th and 7th of December 1996 in Paris
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Official languages French and English
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The symposium is to study all forms of breath-hold diving, and the teaching and training courses for each, to increase knowledge of incidents and accidents for better prevention.

Papers are called for on the following subjects

Breath-hold diving physiology
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Characteristics and constraints of underwater competitive sports: synchronised swimming, underwater fin swimming, underwater hockey, target shooting and spear fishing
Specificities of pure breath-hold diving; static test, dynamic test, constant weight, variable weight and "no-limits"; possibility of records and/or of competitions
Organisation and security for deep diving
Training and ventilation control techniques for breath-hold diving
Psychological aspects of breath-hold diving

For further information contact the Congress Secretariat ARCUS AV, 3, rue Rousselet, 75007 Paris, France. Tel + 33-1- 4567-6801. Fax + 33-1- 4567-1788.

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.

Dr PETER McCARTNEY OAM

The Executive Committee of SPUMS congratulates Dr Peter McCartney, a member for over 20 years, on the award of the OAM, in the Queen's Birthday Honours, for services to Hyperbaric Medicine in Tasmania.

CONSTITUTIONAL AMENDMENTS

No member of SPUMS has objected, by writing to the Secretary of SPUMS, to the constitutional amendments (published on pages 80-82 of the June 1996 issue of the SPUMS Journal) which were passed at the Annual General Meeting in May 1996. As a result the amendments were presumed to be acceptable to the membership and came into force on August 2nd 1996.

The revised constitution will be distributed to all members with the December 1996 issue of the Journal, as will the 1996 issue of the SPUMS Diving Medical.

Cathy Meehan
Secretary of SPUMS

MINUTES OF THE SPUMS EXECUTIVE COMMITTEE MEETING

held on 21/4/96 at Paradise Island, Maldives.

Opened 1445 local time

Present

Drs D Gorman (President), C Meehan (Secretary), J Knight (Editor), G Williams (Public Officer), Dr M Davis (New Zealand Representative), C Acott and R Walker.

Apologies

Drs D Davies (Education Officer), S Paton (Treasurer), A Slark (Past President) and J Williamson.

1 Minutes of the previous meeting (11/2/96)

Accepted as a true record after minor adjustments. Proposed Dr R Walker, seconded Dr J Knight. Carried.

2 Matters arising from the minutes

2.1 North American Chapter update. There is no further news from the North American Chapter. The Treasurer will follow this up.

Continued on page 160

24th ANNUAL SCIENTIFIC MEETING AND ANNUAL GENERAL MEETING

12th to 20th APRIL 1997

at the

QUALITY RESORT WAITANGI,

BAY OF ISLANDS, NORTHLAND,

NEW ZEALAND

Theme

PATHOPHYSIOLOGY AND TREATMENT OF DECOMPRESSION ILLNESS

SPUMS Workshop

FIRST AID MANAGEMENT OF DIVING ACCIDENTS

Guest Speakers

Professor Richard Moon, Duke University, Durham, North Carolina, U.S.A., Immediate Past President, Undersea and Hyperbaric Medical Society.

Dr James Francis, until recently Director, Naval Medical Institute, Alverstoke, United Kingdom.

Richard and James are acknowledged as among the world's leaders in decompression illness. Both were major contributors to the 4th edition of *The Physiology and Medicine of Diving* edited by Peter Bennett and David Elliott. Richard Moon is also the Medical Director of DAN (Diver Alert Network) International. This meeting gives SPUMS members an opportunity to hear two outstanding international speakers.

Conference Convenors

Dr Michael Davis and Associate Professor Des Gorman

For further information contact:

Dr Michael Davis

Hyperbaric Medicine Unit , Christchurch Hospital

Private Bag 4710, Christchurch, New Zealand.

Fax +64 3 364 0187. e-mail at hbu@smtpgate.chhlth.govt.nz

Diving Workshops

The diving at The Poor Knights Islands is considered to be amongst the finest temperate water diving in the world. Water temperatures will be about 20°C.

Delegates will travel to the Poor Knights Islands on 3 days on board a large high-speed catamaran, Tiger IV, which can carry around 250 passengers. During the journeys small group workshops, equipment demonstrations and hands-on training sessions related to the workshop theme of the First Aid Management of Diving Accidents will be conducted.

Conference Week Activities

This is a beautiful maritime park region of New Zealand, which is also an area of considerable historical importance. For the non-diver, there is a wealth of land and water-based activities with something for everyone.

The Quality Resort Waitangi is an ideal family venue. A diverse and exciting children's programme has already been developed to suit all ages, as well as excellent baby care facilities.

Bring the whole family for a wonderful New Zealand Holiday!!

Pre- and Post-Conference Tours

Two contrasting and outstanding diving venues will be offered. The Three Kings Islands, situated some 30 miles North of New Zealand, and Fiordland, on the West Coast of South Island.

In addition, self skippered sailing charters at very competitive rates have been reserved and there is an immense range of other holiday opportunities in New Zealand that Fullers Northland's travel division will be able to advise delegates on.

For all travel and New Zealand domestic enquiries, please contact:

Fullers Northland (attention Tania Townsend)

PO Box 145, Paihia, Bay of Islands, New Zealand.

Phone +64 9 402 8802 Fax +64 9 402 7831

PLEASE HELP US TO MAKE THIS A GREAT MEETING BY RETURNING THE NOTICE OF INTENT ENCLOSED WITH THE LAST JOURNAL.

2.2 Maldives Update

2.2.1 Payment for the slide projector to come from funds left over from the Fiji Annual Scientific Meeting (ASM) account. There is to be no reimbursement to general funds from the Maldives conference account for the cost of the letters to EUBS notifying them of the Maldives ASM.

2.2.2 Conferences expenses update. This was discussed. It was proposed that a projector for direct computer presentation be researched for future purchase.

2.2.3 Up date on formalities at the Gala Dinner. Dr Gorman will give a speech. A book prize will be given for the best free paper.

2.3 Copy of Dr Williams' letter of retraction to Allways is needed to complete the records. A copy of this is to be given to the Secretary.

2.4 New Zealand 1997 ASM. A Notice of Intention will be enclosed with the June 1996 Journal.

2.5 Indemnity policy. The Public Officer, Dr Williams will look into this.

2.6 Reprinting of the SPUMS Diving Medical and Statement of Purposes and Rules.

2.6.1 Changes to the SPUMS Diving Medical were discussed. The draft SPUMS policy on medical practitioner certification of fitness for diving was printed in the December 1995 Journal and was open for comment. None has been received. This has now become a SPUMS policy and will be printed in the SPUMS Diving Medical.

2.6.2 Statement of Purposes and Rules has been assessed and proposed changes have been printed in the March 1996 Journal (pages 17-19) for comment. These proposed changes to the constitution will be put to the Annual General Meeting (AGM).

2.7 Role of convenor to be defined and guidelines written (Dr Acott).

2.8 Proposal to form an Ex-Presidents Committee will be put to the AGM.

2.9 A possible honorarium for Treasurer and Secretary was discussed in view of the amount of work that is, at present, required of them. It was decided that with the new computer system, and future technical support, as well as increasing the use of extra help, that the work-load can be reduced. Although the Editor at present receives a honorarium, this was thought to be a different situation as the Editor's position is considered to be more long term.

2.10 A vote of thanks to the Treasurer has been included in the President's report.

2.11 Carl Edmonds Library and the future housing of it will be decided by Dr Edmonds. Dr Knight will communicate with him.

3 Treasurer's report

This was discussed as well as future expected expenses in association with setting the proposed membership fees for 1997.

4 Correspondence

4.1 Letters re full membership to be considered at the AGM. Dr Davis will speak for Quentin Bennett's application and Dr Knight will speak for John Lippmann's application at the AGM.

4.2 Letter from Queensland Division of Workplace Heath and Safety. It was requested of SPUMS to give an opinion on the most suitable form of diving medical examination for all workers in the diving industry, including recreational diving instructors.

5 Other business

5.1 Membership renewal notice and the confusion with regard to the date of commencement of membership. At the top of the renewal notice it states that the SPUMS membership year is January to December. It also states that "If payment is not received by April 30 1996 your subscription lapses and the June Journal will not be sent". The member's name is then taken off the mailing list. This has caused some confusion with members thinking that the final renewal date for membership was April 30th. In order to prevent this from happening the form will be altered and DUE DATE for subscription renewals will be clearly marked on the form. The renewal notice to go out before December. Dr Walker and Dr Meehan will co-ordinate this and Dr Knight will then do the artwork for the new form.

5.2 Diving Doctors List (DDL) update. There will in future be two issues of the DDL, printed in June and December as decided at the Committee Meeting in Cairns in October 1994. The Diver Emergency Service (DES) will hold the register as previously discussed. There will be an asterisk beside the names of Doctors who have

completed a diving medical examiner's course of at least 10 days duration. It was the Committee's opinion that a course of ten days duration or longer gave the additional knowledge and training in diving medicine needed to perform occupational dive medicals according to AS 2299. All doctors on the DDL are considered qualified to perform recreational dive medicals.

5.3 Teleconference costs. The last one cost $1,500. Everyone was reminded to keep business flowing and to be as brief as possible.

5.4 Discussion re tendering process. This has caused a lot of extra work and aggravation, as well as extra expense in teleconference costs. It was discussed that SPUMS should concentrate on the land content and let the individual members arrange their own travel with their own travel agent.

5.5 SPUMS position statement on Diabetes and Diving, written by Dr Williamson, was read. It has been printed in the June 1996 Journal (pages 70-72).

5.6 The postal ballot for the positions of Committee members was counted. The unsuccessful candidates were Drs Roger Capps and Douglas Walker.

5.7 Index of SPUMS Journals. It was proposed that the index be formatted into Microsoft Access (it is at present in Microsoft File for Mac) and be available on disks. With each subscription renewal a disc would be offered for $10 Australian. This would be updated with the completion of each Journal.

Closed at 1645.

MINUTES OF THE ANNUAL GENERAL MEETING OF SPUMS

held on Paradise Island, Maldives,
Friday 26th April 1996 at 1700 local time

1 Minutes of the previous Meeting

The minutes of the previous meeting have been published (SPUMS J 1995; 25 (3): 130-133)

Moved that the minutes be taken as read and are an accurate record. Proposed John Knight, seconded Geoff Long. Carried.

2 Matters arising from the minutes

None.

3 President's Report
Printed on pages 162-163.

4 Financial Statement and Treasurers Report

Printed on pages 164-166.

That the Treasurer's report be accepted and a vote of thanks to Dr Paton for her four years as Treasurer be recorded. Moved Des Groman, seconded John Knight. Carried.

5 Subscription fees for the coming year

Moved that the annual subscription be $100 for members and $50 for associates from January 1st 1997.

Proposed Chris Acot, seconded Mike Davis. Carried.

6 Election of office bearers

A postal ballot of financial members had been held to elect the three committee members. Drs Roger Capps and Douglas Walker were the unsuccessful candidates.

The Committe for 1996-97 is

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

7 Appointment of the Auditor

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

8 Business of which notice has been given

8.1 That Mr John Lippmann be elected to Full Membership of SPUMS. Proposed Dr John Knight, seconded Bill Hamilton. Carried

8.2 That Mr Quentin Bennett be elected to Full Membership of SPUMS. Proposed Dr Mike Davis, seconded Dr John Knight. Carried

8.3 That at this and subsequent annual general meetings opportunity be given for members to raise matters of concern for which prior notice has not been given Proposed Dr Jim Marwood, seconded Dr Gareth Long.

It was pointed out that whatever the fate of the motion no such discussions could be held until the consitution was changed as it specifically bars discussion of matters of which notice has not been given. If successful there will have to be a motion to change the constitution passed at the next AGM.

Carried

8.4 The Committee proposed that the 13 motions for constiutional changes, printed on pages 80-82 of the June 1996 issue of the SPUMS Journal, be accepted.

After the reasons for the changes had been explained the motions were carried.

They will only come into force after a postal ballot of financial members. The constitutional changes have been printed on pages 80-82 of the June 1996 issue of the SPUMS Journal and members have been requested to object, in writing, to the Secrteary of SPUMS if they do not want the proposals to procede. If no member objects, in writing, to the Secretary of SPUMS before August 1st 1996 it will be assumed that the membership has voted, in a postal ballot, in favour of the motions.

PRESIDENT'S REPORT 1996

Farewell and thank you.

There is general consensus that 6 years is a long time for anyone to be the President of a medical society. There is no doubt in my mind that it is time for me to go. This decision will be greeted with relish by some in the Society and in the recreational diving community; especially those who have tried, even to the extraordinary and desperate level of blackmail, to stop the Society from becoming involved in the debate about the health and safety of diving training and practice, an involvement that is surely the raison-d'etre of the Society. Others may be pleased that the perceived Gorman-Acott conspiracy is to end. Those who have worked with the Committee over the last 6 years will know what a myth that is; along with the equally mischievous claims that the Executive, under my influence, has "gone over to the dark side" and embraced the commercial Gods worshiped by PADI and the other diving instructor agencies. I have some very good friends in the recreational diving community, such as Drew Richardson and Terry Cummins, and am very proud of the progress we have made since 1990 on the basis of these personal relationships with respect to the interface between the Society and the recreational diving community. This is something I would like to discuss in some detail later.

Farewells should be positive statements and affirmations of intent, not concentrations on the irritations of the past. Notwithstanding this comment, I have genuinely hated the tendering process we have used for recent meetings, it has been a major and unproductive distraction. It is farcical to ask tour operators to tender 18 months or more in advance of a meeting, to expect hard prices, to make relative judgements about the tenders and then to expect the product to be delivered within a "bulls-roar" of the original tender. This is however the only aspect of the Society's economic reform that I regret or for which I apologise.

It is always nice to cite some legacy of a period of executive management and, in that context, I would like to identify four phenomena that illustrate recent progress. These are the calibre of Annual Scientific meetings, the quality of the Society's Journal, the economic reform of the Society and, most importantly, the now mature relationship that the Society generally enjoys with the recreational diving industry.

Annual Scientific Meetings.

The Society's meetings have always been fun and a trial for our guests. I remember finally getting to present my first paper to the Society in Tahiti when I was the Society's guest along with Andy Pilmanis. Andy had overshot his time by 8 hours (or so it seemed) and I was greeted with a front row that was, with one exception, fast asleep. My first slide was prefaced by a somnolent fart from an esteemed Society member.

Several years later, I was the convenor of the first Palau meeting and hoped that it was possible for a meeting to be both fun and academically worthwhile, the advent of the conference theme and workshop format. Since then the academic standard of the meetings has gone from strength to strength, without any loss of the essential culture of the Society's gatherings. The latter has much to do with the national philosophy, the persona of Misters Leslie and Jones and their confederates in the black-hole gang and the legendary BUMS. The former has been the product of interesting themes, excellent guests, and especially repeat guests such as David Elliott, and the product of very conscientious convenors such as Chris Acott in Rabaul and here in association with Guy Williams, John Williamson at Port Douglas, and David Davies at Fiji. I am to blame for the rest.

The meetings also owe much of their character to the family atmosphere of the Society, that is not to argue that children have been necessarily welcome and especially by their parents. Such an atmosphere will always create a clique phenomenon but I hope that entrance to the clique has never been too difficult for those prepared to become involved. There is no doubt that Geoff Skinner and Adrienne

McKeone are a very real part of that family and have done great things on behalf of the Society. On behalf of all of us in SPUMS, I wish to thank them both for all the help and assistance, and for their contribution to the success of all our recent meetings.

The SPUMS Journal

By the start of this decade, the Journal had became noticeably tired, an opinionated newsletter of limited scientific merit. The challenge was to upgrade the content, without a change in orientation. This has been achieved and to widespread acclaim and is largely due to the efforts of John Knight, who assumed the role of Editor at the time. All credit to you John and thank you. I know that you and I have had some minor differences, for example about the vitriolic nature of some letters to the Editor that we have published, but you should not underestimate the considerable esteem in which your efforts on behalf of the Journal are held.

I am also pleased to see the support that is now being offered to you and that some recognisable succession is in place, to be invoked when needed.

The Society's finances

If the Journal looked tired in 1990, then the Society's finances looked positively exhausted. This is not a direct criticism of the Treasurer of the time, it is simply a recognition that the Society had outgrown its executive infrastructure and management practices. An unsuccessful attempt to contract out our support services followed, and then Sue Paton took up the fiscal cudgels and within a year was reporting a very healthy statement of finances. The Society is in great figurative debt to you Sue, and, given that you are also resigning this year, I would like to take this opportunity on behalf of the Society to sincerely thank you and to wish your successor, Robyn Walker, a long career of happy accounting.

I am also indebted to all of the members of the Executive Committee over the last 6 years and especially to the 3 secretaries who have served the Society in that time, David Davies, John Robinson, who may one day see the humour of being left on the wharf at Palau, and the incumbent, Cathy Meehan, the reigning Queen of Queensland diving medicals.

The recreational diving community

As the leading trainer of recreational divers, both world-wide and in our region, PADI has always been at the forefront of the recreational diver-SPUMS interface. What a long way this interface has come !

I remember the nature of the relationship between diving physicians and the recreational divers at the end of the 1970s and in the very early 1980s when I first went to work at the Royal Australian Navy's School of Underwater Medicine. It was hostile. The hostility was typical of that which is so often a feature of the recreational diving industry, an industry in which ego's often dwarf intellects and experience. The bad feelings in this context were compounded by the following:

the general disregard in which many of the Naval Diving Officers of the time held recreational divers and about which they were happy to tell the media;

the irresponsibility of the media in reporting diving accidents and especially fatalities; and

the personalities of the then leaders of PADI and of the diving medical community. Certainly, there was little trust, very little respect and little or no meaningful dialogue.

It has been a long struggle to break down these barriers and, in the same way in which they were created, they have been "dissolved" at a personal level.

In this context, the advent of Drew Richardson at PADI International, Terry Cummins at PADI Australia and Colin Melrose at PADI New Zealand have been the principal catalysts for the development of a now very mature relationship with the diving medical community.

It was during the SPUMS debate on Emergency Ascent Training that the true nature of the reform became apparent as for the first time PADI "trusted" the Society with data that could have been misconstrued and used to embarrass the diving trainers. The SPUMS Workshop on Technical Diving held here is another illustration of the current level and spirit of co-operation. This is not an argument that everyone is happy with the status quo. There are those, fortunately in the minority, in both the recreational diving community and in the diving medical community who still see the relationship as essentially combative. Let us hope that they pass into extinction with all the other dinosaurs.

Finally, it is with great pleasure that I now hand over the "keys" to Guy Williams. I promise that I will be around in my new capacity as Past-President and hope that I can be of as much benefit to Guy as the very wise Tony Slark has been to me. And so, good luck to you all and see you in New Zealand next year.

Des Gorman
President of SPUMS 1990-1996

TREASURER'S REPORT 1996

The statement of Receipts and Payments for the year ended 31/12/95 show containment of the Society's recurring expenses below that of the preceding year.

The reduction in expenditure is attributable to reduced expenditure on equipment and limiting the number of face to face Committee meetings to one. The equipment purchases made in 1995 were a new printer for the Secretary and a laser printer and A4 monitor for the Editor.

In 1995 subscription income fell slightly from that of the preceding year. As a result of the increase in subscription fees, income should rise by about $1,000. However, funds in excess of this amount have already been committed to the purchase of notebook computers for the Secretary and Treasurer. With the latest software this equipment should greatly improve the Society's database and its efficient operation.

Other significant changes taking effect in 1996 are an increase in the Editor's Honorarium to $15,000 per annum effective from 1/7/95 and closure of the SPUMS funded North American Chapter account, given that North American members now receive their Journals by Economy Air Mail.

Also additional and continuing expenditure is involved with the publication of an index for the SPUMS Journal to mark the 25th anniversary of the Society. To date secretarial expenses alone in this project are in excess of $2,000.

Conference registrations

1995 was the first year that the Society undertook to manage the conference registration. The relevant statement of Receipts and Payments is included with the Auditor's report (see page 166).

A substantial portion of the small profit derived from the 1995 conference has already been utilised to fund the purchase of a slide projector in time for the 1996 Annual Scientific Meeting.

Membership statistics

Current membership is 1,127, slightly less than the 1,190 at the same time last year. Of these 811 are full members.

As in April last year, one third of the members have not yet paid their current annual subscription.

The geographical distribution of the membership remains similar with 825 members in Australia, 111 in New Zealand 102 in North America and 89 elsewhere.

Subscriptions

During my time as Treasurer, as a result of subscription increases since 1992, income has exceeded expenses by about $20,000 annually and the Society's financial position has changed from one of debt to reserves of $88,000 at the end of 1995.

However in the four months to date this year the annual excess of income over expenditure has already been absorbed by the computer purchases and, although income should increase by $10,000, expenses are already $25,000 in excess of the same time last year.

Prudent financial management dictates that the Society's substantial reserves be maintained and that increases in routine expenditure be covered by subscription income. Hence, with the Committee's approval, I am proposing that the annual subscriptions be raised to $100 for full members and $50 for associates effective for the year commencing January 1st 1997.

Conclusion

The Society depends on the voluntary efforts of its office bearers, and although the new computers will ease the burden on the Secretary's and the Treasurer's time, after 4 years as Treasurer, other pressing demands on my time have forced my decision to resign from my position. I take this opportunity to thank the Committee members who have assisted me in my role as Treasurer.

For a relative newcomer to the Society my role as Treasurer has been rewarding in the enormous amount learnt about the workings of the Society and its interaction with its members. Maintaining the database has provided an interesting insight to the global wanderings of many of our members and has shown the increasing influence of, and demand for, the Society's Journal among statutory and scientific bodies seeking reference material in the field of diving medicine. It is hope that the members will ensure that the high standard and image projected by the Journal is maintained and further enhanced in the future.

I am pleased to be able to hand on to my successor, Dr Robyn Walker, a financially healthy Society and I wish our new Treasurer every success.

Sue Paton
Treasurer of SPUMS

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

I have conducted the various tests and checks as I believed are necessary considering the size and nature of the Society and, having so examined the books and records of the South Pacific Underwater Medicine Society for the year ended 31st December 1995, report that the accompanying Statement of Receipts and Payments has been properly drawn up from the records of the Society and gives a true and fair view of the financial activities for the period then ended.

Dated 17th April 1996
Newport Beach, New South Wales 2106

David S Porter
Chartered Accountant

THE SOUTH PACIFIC UNDERWATER MEDICINE SOCIETY
STATEMENT OF RECEIPTS AND PAYMENTS FOR THE YEAR ENDED 31 DECEMBER 1995

		YEAR ENDED 31/12/95		YEAR ENDED 31/12/94	
OPENING BALANCE					
ANZ bank	Access Account	(364)		945	
	ANZ V2 PLUS	68,053		42,499	
			67,689		43,444
INCOME					
Subscriptions		76,061		80,963	
Interest		4,956		2,279	
Advertising and journal sales		979		1,250	
Sponsorship		-		2,585	
Reimbursement of Registration (PNG)		-		6,932	
Refund from CIG (PNG)		-		1,138	
Reimbursement from registration (Fiji)		1,082		-	
Sundry Income		168		-	
			83,246		95,147
			$150,935		**$138,591**
EXPENDITURE					
AGM costs		1,583		1,417	
Donations of 2 oximeters (PNG)		-		7,500	
Secretarial		5,573		5,142	
Stationery and printing		2,067		3,012	
Journal		22,199		23,442	
Postage and facsimile		3,552		5,638	
Conferences and telephone		6,608		10,487	
Equipment (see note)		3,405		6,905	
Miscellaneous/Subscriptions		1,559		1,394	
Bank charges		1,273		1,343	
Audit		540		350	
Editors honorarium		13,978		4,272	
			62,337		70,902
CLOSING BALANCES					
ANZ bank	Access A/c	(3,722)		(364)	
	ANZ V2 PLUS	92,320		68,053	
			88,598		67,689
			$150,935		**$138,591**

These are the accounts referred to in the report of D S PORTER, Chartered Accountant, Newport Beach 2106, dated 17/4/96.

NOTES TO THE STATEMENT OF RECEIPTS AND PAYMENTS FOR THE YEAR ENDING 31 DECEMBER 1995

1 Equipment is written off as purchased.
2 Subscriptions are on a receipt basis.
3 Registration income and expenses for 1995 ASM Fiji are detailed below.

ANNUAL SCIENTIFIC MEETING INCOME AND PAYMENTS

	Year ended 31.12.95		To 17.4.96	
INCOME				
Registrations	29,443		28,943	
Interest received	854		1,184	
		30,297		30,127
EXPENSES				
Allways Dive Expeditions	16,899		23,248	
Safety Sausages	468		468	
Bank charges	82		115	
Castaway Is. Staff Christmas	410		410	
Fiji Compression Chamber	-		250	
Postage, printing & stat.	1,166		1,166	
Secretarial	182		182	
		19,207		25,839
NET INCOME		**$11,090**		**$4,288**
	These funds are represented by closing bank balances:			
ANZ Bank Access Account	1,320		589	
ANZ V2 Plus	9,770		3,699	
		$11,090		**$4,288**

Payments to Allways Dive Expeditions consisted of

$11,389 for costs (in excess of FOC's received) relating to guests speakers, dive escorts, cocktail party, gala dinner and afternoon teas.

$11,859 reimbursement of losses incurred by Allways due to exchange rate drop at the time (27/4/95) of payment of group tour costs. (This was a payment determined by the committee as an isolated ex-gratia payment to Allways in recognition of their work and to finalise this outstanding issue. The payment is not intended to set any precedent nor to be taken as an admission of any liability on the Society's part.)

These are the accounts referred to in the report of D S PORTER, Chartered Accountant, Newport Beach 2106, dated 17/4/96

NZ CHAPTER REPORT

OPENING OF THE HYPERBARIC MEDICINE UNIT, CHRISTCHURCH HOSPITAL

The Hyperbaric Medicine Unit at Christchurch Hospital was officially commissioned on Friday 12th April, 1996. This was an important occasion for hyperbaric medicine in New Zealand. The ceremony, hosted by the new unit's medical director, Dr Michael Davis, and his staff, was attended by several Board Members of the Canterbury Health (CHL) Crown Health Enterprise including the Chairman, Dr Brent Layton. Other guests included the "grandfather" of diving medicine in New Zealand, Dr Tony Slark, Assoc. Professor Des Gorman, Surgeon Commander Courtenay Kenny (Director-General of Naval Medical Services) and other representatives of Royal New Zealand Navy, New Zealand Underwater (NZU), PADI NZ, Dive Industry NZ (DINZ), the South Island Chamber Liaison Committee (SICLC) and the medical and diving

community from the length and breadth of New Zealand. Sadly, the hyperbaric contractors, CABA of Sydney, were unable to attend.

The Rev Maurice Gray, a CHL Board member and Director of the Maori Research Unit at Lincoln University, unveiled the memorial plaque. In considering the purposes for and the methods used in hyperbaric medicine, Mr Gray invoked the Maori name "Te Whare Hau o Te Ha Ora" for the recompression chamber, which he translated into English, as closely as he could, as "the inner sanctum of life". Generous donations to the new unit were presented by SICLC, NZU and DINZ. In a welcoming address at the reception, Des Gorman entertained all present as he recalled some of the rocky road leading to this occasion and enumerated the challenges facing the new service and CHL in the years to come.

NEW ZEALAND CHAPTER ANNUAL SCIENTIFIC MEETING

The Chapter's annual meeting, convened by Michael Davis, was held the following day in the Christchurch School of Medicine and was attended by 35 registrants, including quite a few sport diving instructors, from as far afield as Invercargill and Auckland.

The morning session focused on hyperbaric medicine. Frank Thomas, a first year anaesthetic registrar in Christchurch, opened the meeting with several case histories of carbon monoxide poisoning. These illustrated three aspects of this problematical poisoning, namely, the lack of correlation between admission carboxyhaemoglobin values and the severity of symptoms, the occurrence of delayed or recurrent symptoms and the apparent effectiveness of delayed hyperbaric oxygen (HBO) therapy in some severe cases. These cases laid the groundwork for Des Gorman to discuss current theories of and their evidence regarding the mechanisms of CO poisoning. Much of this has been presented recently in the literature, but his talk brought the meeting right up to date and sketched the way in which research over the next few years is headed.

This was followed by a brief review by Mike Davis of oxygen as a pharmacological agent, illustrated by two examples of dose response data in hyperbaric medicine. It was agreed that we still had a long way to go before understanding how to use oxygen properly! Simon Mitchell then presented the 1995 diving accident data from the Slark Hyperbaric Unit in Auckland, which will be published in the SPUMS Journal.

David Wright, on attachment to the Slark Unit from the Royal Australian Navy, gave a succinct review of the current place of HBO in acute soft tissue sports injuries. His verdict from the literature to date was "not proven". Clearly there is need for good scientific clinical research in this area, despite the mounting popularity of and commercial impetus being given to this mode of treatment.

The morning concluded with the first of two invited presentations from Alexei Korolev, a young Russian medical graduate who has recently emigrated to New Zealand from Moscow. Alexei had spent a decade in hyperbaric medicine and physiology research in the USSR. The vast range of clinical indications for HBO and the huge numbers of patients treated in Russia left the audience shaking their heads in disbelief. The small Christchurch chamber would just about do as one of the medical locks for the chamber complex at the Institute for Hyperbaric Medicine in Moscow!

The afternoon session was devoted to Technical Diving. This was chaired by Simon Mitchell who had many years as a sport diving instructor and scientific diver before taking up medicine. He is SSI's medical adviser in New Zealand. Simon set the scene by defining some of the terminology, summarising the techniques encompassed and highlighting the potential problems that each presents in comparison to open circuit air scuba diving.

A personal historical perspective of semi-closed circuit helium-oxygen diving when he was a student in the 1960's was given by Michael Davis, and is to be published in the Journal. Tim Williams, a clinical psychologist and one of New Zealand's most experienced cave divers, gave a fascinating illustrated talk in which he provided a real insight into why cave divers have been at the cutting edge of technical sport diving. This was followed by a brief summary by Des Gorman on legal and ethical issues in technical diving. A strong message not to lump widely differing diving techniques together as a single entity, so-called "technical" diving, came across at the meeting.

Colin Melrose introduced us to the new PADI Nitrox training program recently released worldwide, whilst Mark Scapens commented on plans for similar courses currently being developed by SSI. Some differences in philosophy between the two training organisations were apparent, and a lively, interesting debate ensued.

The meeting ended with Alexei Korolev talking about sport diving in Russia. Alexei described the two great disasters that have befallen the Russian people during the twentieth century, the Revolution of 1917 and Glasnost! In USSR history every advance in diving technology in the West had been preceded years before by Russian divers and scientists. Essentially, sport diving did not exist in Russia before glasnost, and Alexei's descriptions, despite his limited English, of the lengths to which he and his friends went in order to go diving, their use of the black market, bogus scientific "expeditions" etc. and how he persuaded the authorities to send him to France to do a CMAS Instructor's Course caused much wry amusement.

MINUTES OF THE ANNUAL GENERAL MEETING OF THE NEW ZEALAND CHAPTER OF SPUMS

held in The Bevan Lecture Theatre
Christchurch School of Medicine 13th April 1996.

The meeting opened at 1800 hours.

Present

Drs M Davis (Chairman), C Morgan (Secretary), six full members and several Associate members. Apologies were received from four members.

1 Minutes of the last AGM

Accepted.

2 Matters arising

see 3 below.

3 Chairman's Report

Much of the Chairman's year has been taken up with re-establishing the Christchurch hyperbaric unit and preliminary work towards the main SPUMS Annual Scientific Meeting (ASM) in 1997, for which he is convenor. This has left little time for other SPUMS activities such as publicising the Society to a wider audience. The executive has reviewed the chapter's accounts as minuted last year and the Secretary is in the process of rationalising these. Difficulties have again been experienced in obtaining up to date information on NZ members of the society. Next year's conference in the Bay of Islands will be a landmark event for the Chapter and the Chairman urges all NZ members to attend. Professor Richard Moon from Duke University and Dr James Francis from the Royal Navy had both indicated their willingness to participate.

4 Secretary/Treasurer's report

The financial report was not tabled at the meeting and was presented at a later date (see below).

5 Nominations for 1995-96

The office bearers indicated their willingness to stand for another term and there were no other nominations for these positions.

6 Possible venues for future meetings

These were discussed. There will be no separate Scientific Meeting of the Chapter in 1997 because of the Bay of Islands ASM. For the 1998 meeting, the NZ Naval Medical group volunteered to act as hosts at Devonport. This was accepted with pleasure from the chair.

7 General Business

C Kenny advised that he and others would appreciate viewing the accounts of the 1995 meeting in Tairua. He felt that some members may be eligible for a refund of fees as the diving trip had been cancelled.

D Gorman suggested that there must be ways of increasing membership numbers of the NZ Chapter. He pointed out that other chapters were increasing in membership numbers and that dive instructors should be advised that they were eligible for associate membership. Moved Gorman/Kenny: "that the New Zealand Chapter enter an active recruitment program and that all members try to recruit a colleague". Passed unanimously.

There being no further business, the meeting closed at 1845.

FINANCIAL REPORT

At 1st March 1996 there was $7,016.50 in four New Zealand Chapter accounts. This represents a gain of slightly less than $1,000 over the last financial year due to interest on the savings accounts and profit from the Tairua meeting.

Assets

BNZ Milford	$3,168.70
ASB cheque account	$ 227.73
ASB Savings account	$2,536.40
BNZ (Meeting) account	$1,083.67
Total	**$7,016.50**

C Morgan
Secretary/Treasurer New Zealand Chapter of SPUMS

SPUMS POLICY ON TECHNICAL RECREATIONAL DIVING

Des Gorman, Drew Richardson, Bill Hamilton and David Elliott

Key Words

Diving industry, diving safety, mixed gases, policy, rebreathers, technical diving, training.

Introduction

The Society dedicated the 1996 Annual Scientific Meeting to a Workshop on emergent recreational diving practices, the so-called "technical diving", in recognition of the need for some pragmatic guidance in this area from an organisation with no commercial interest in the activity and as a responsible medical society.

The Society has presented opinions on the subject previously in the form of an Editorial in the Journal.[1] This included two basic statements: first, that the risks involved needed to be understood by aspiring divers and trainees;

and that the Society "would not argue" with attempts at relevant regulation. The latter has to be interpreted in the context of the debate at the time, the nature of the then-intended diving practice and the absence of well-established diving systems outside the conventional (open circuit demand scuba-air diving to 40 msw) recreational diving clubs and instructor agencies when the Editorial was written. It also needs to be emphasised that, with the exception of "employed" divers where a regulated "duty of care" for employers is essential, the Society has never supported the external regulation of recreational diving. Indeed, the Cave Divers Association of Australia, has been and will continue to be advocated by SPUMS as a role-model of effective self-regulation.

The debate about technical diving has often been acrimonious, to the discredit of those involved, and has consequently distracted the attention of the debaters from the essential and necessary description of appropriate and relevant risk management. In addition, many commentators have become obsessed with the nature of the *diver* (e.g.. recreational versus employed) rather than paying attention to the nature of the *diving*. The issue is made more complex by uncertainty as to what is technical diving. Comprehensive definitions consequently include diving practices with widely divergent risks, thus a debate on the "safety" of technical diving *per se* becomes nonsensical.

The SPUMS workshop on technical diving

The SPUMS Workshop on technical diving was free of both acrimony and rancour, indeed it was vigorous and entertaining, much to the credit of all those who presented papers, some of which are published in this edition of the Journal, and those who became involved in the debate. An acknowledgment here of the high quality of conference convening by Drs Guy Williams and Chris Acott is also appropriate. There were no written submissions.

THE SPUMS POLICY ON TECHNICAL DIVING

1 Recognition of technical diving

The Society recognises, but does not necessarily endorse, technical diving. Such diving includes activities "outside" the conventional recreational limits of open-circuit demand scuba-air diving to 40 msw and often involves special techniques, equipment, gas mixtures and decompression procedures. Although a common definition of technical diving limits practice to those which involve a rebreather or a change in breathing gas during the dive (and hence excludes shallow enriched air diving and deep air diving), it is still considered that, for the purpose of risk management, technical diving is not a sufficiently specific term. This is because the types of diving referred to by this title are widely divergent in nature and risk. Instead, a consideration of each type of diving in isolation is necessary. It also follows that unique training and diving conduct measures are necessary for each type of diving activity.

The Society believes that the critical issue in assessing diving practice is the nature of the practice and not the intent or employment status of the divers.

2 Risk management in technical diving

Many of the following comments are generally applicable to diving, but are especially important in the context of technical diving and hence are included in this policy statement. In general, the Society encourages those who engage in any form of diving to have the requisite training, experience, attitude, equipment and support (both in the water and at the surface), operational planning and organisation to be able to dive "safely".

The Society believes that before anyone undertakes any form of diving education or diving practice, it is important that:

the health and other hazards associated with either the diving or the training are identified, the associated risks be assessed, in the context of the health of that individual, and that appropriate control measures for these hazards are in place;

the individuals concerned understand and accept (in writing) the risks of that activity and especially in the context of their health; and

where an employer-employee relationship exists, that an appropriate duty of care be exercised in accordance with local health and safety legislation, such as occupational diving fitness standards.

Again, although this is generally true for all diving, the Society also believes that, given the current level of undergraduate education in diving medicine for medical practitioners, some form of post-graduate training is a pre-requisite to the effective conduct of "diving fitness" examinations.

3 Self-regulation of technical diving

The Society encourages recreational diving instructor agencies and dive organisations to evaluate critically all forms of diving technique that they intend to teach or practice. This recognises the current low rates of risk involved in conventional recreational diving as defined above. It is also reasonable to assume that effective control (i.e. risk management) of emergent diving techniques will result in the following:

individual morbidity and mortality rates and associated costs to local health systems will not increase;

relevant health and life insurance premiums will, at worst, remain unchanged and, at best, may decrease;

the public perception of recreational diving in general will either be maintained or improved;

and, consequently, there will be no substantial stimulus for any increase in external regulation of recreational diving activities.

Although the Society believes that occupational health and safety agencies should be encouraged to produce codes of diving training and practice, especially for operational dive organisation and planning, and technical codes for such things as equipment design, gas standards, gas mixing and testing, it is strongly recommended that these be seen as templates and that recreational diving groups become self-regulating. It is also recommended that this regulation should be visible in the form of standards and activities such as independent audits of incidents and accidents as part of an overall quality management program.

The Society is also concerned at the currently extravagant, and occasionally inaccurate, advertising of diving equipment and practice made by some manufacturers and training agencies and at the likely consequent misleading of the diving public. A self-regulated code of practice is recommended in this context. Members of the Society are also reminded that regulations concerning the accuracy of advertising do exist in most countries and that they should be active in alerting the relevant regulators.

4 Specific forms of technical diving

Compressed air diving is not recommended deeper than 40 msw. Deep air diving below these depths in pursuit of individual or community records is considered foolish and should be discouraged.

Open-circuit demand scuba enriched air diving in accordance with proposed limits is not considered to represent a significantly greater risk to divers than conventional recreational diving practice as defined above. The PADI EANx (enriched air nitrox) program is acknowledged by the Society as being excellent and is recommended as a benchmark in this context.

Rebreathers currently available to the diving public may have operating instructions that are based on inappropriate assumptions concerning semi-closed diving apparatus and respiratory physiology. This could, and has been shown to, result in hypoxia and equivalent-air-depths that under-estimate the inert gas exposure. All semi-closed diving apparatus should be assessed for inspiratory gas content (at least over the oxygen consumption range of 0.5 to 3 litres/minute) by a suitable laboratory before sale to the diving public. Closed circuit diving apparatus also needs testing, but with a greater emphasis on technical reliability.

5 Treatment of technical diving accidents

The first-aid management of an injured technical diver should be determined by the nature of the injury and will not differ from that recommended for divers in general.

Although the majority of technical divers developing a decompression illness will be well treated with a conventional treatment schedule such as USN 6, the Society encourages medical practitioners who may have to treat such divers to be aware of techniques such as oxygen-helium gas mixtures and saturation decompressions.

References

1 Gorman DF. High-tech diving. *SPUMS J* 1992; 22 (1): 1-2

Dr D F Gorman FAFOM, PhD, is Associate Professor of Medicine and Head, Occupational Medicine, Faculty of Medicine and Health Sciences, University of Auckland, New Zealand. He is the immediate Past-President of SPUMS. His address is 52 Albert Road, Devonport, Auckland 9, New Zealand. Telephone + 64-9-373-7599. Fax + 64-9-308-2379. E-mail d.gorman@auckland.ac.nz .

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Dr Bill Hamilton, PhD, was one of the guest speakers at the SPUMS 1996 Annual Scientific Meeting. He is a diving physiologist with special interests in designing decompression tables. Many of his tables have been used very successfully by technical divers. His address is Hamilton Research, Ltd., 80 Grove Street, Tarrytown, New York 10591-4138. U.S.A. Fax +1-914-631-6134. E-mail 70521.1613@compuserve.com .

Dr David H Elliott was one of the guest speakers at the SPUMS 1996 Annual Scientific Meeting. He is Co-Editor of The Physiology and Medicine of Diving, which was first published in 1969, with the most recent edition in 1993 and is also the Civilian Consultant in diving medicine to the Royal Navy. His address is 40 Petworth Road, Haslemere, Surrey GU27 2HX, United Kingdom. Fax + 44-1428-658-678. E-mail 106101.1722@compuserve.com

LETTERS TO THE EDITOR

THE VALSALVA MANOEUVRE

118 Remuera Road
Auckland 5
New Zealand
24/5/96.

Dear Editor

I was prompted to write this letter after reading the excellent review by Dr David Taylor.[1] Delonca, in an article on methods of equalising middle ear pressures for divers on descent, quotes from a book written by Valsalva called "Tractus de Aure Humana" or *A Treatise On The Human Ear*.[2] In another historical publication I read that in this book about the human ear, Valsalva described his manoeuvre as a means of treating discharging ears in adults. In other words by blowing air up the Eustachian tube it forced pus from the middle ear through the perforated tympanic membrane and this resulted in a cure of the chronic suppurative otitis media.

From my investigations the Valsalva manoeuvre is in fact the method used by divers to force air up the Eustachian tube and this interpretation of the manoeuvre is accepted by all Ear Nose and Throat surgeons. However there seems to be some conflict of opinion with physicians. When I asked a physician what was meant by the Valsalva manoeuvre. he described what Dr Taylor states in his introduction, a forced expiration against a closed glottis. Dr Taylor described a "standardised" Valsalva manoeuvre, which is neither of the above. As far as pressure effects are concerned there is a difference between expiration against a closed glottis and holding the nose and blowing hard down through the nose.

I have often wondered whether Valsalva wrote 2 books, one for the physicians and one for the ear surgeons or whether or not some person many years ago misinterpreted what Valsalva wrote so that the physicians are firmly convinced that their interpretation is the true one and the Ear Nose and Throat surgeons are all wrong.

Dr Taylor states that complications are usually seen when Valsalva is performed either "too forcefully" or "for too long a period." He does not define what he means by either of these indices. I have maintained over the past 30 years that a person should not hold the nose blocked for more than 5 seconds at a time and none of my patients have come to any harm. I consider that carrying out the Valsalva manoeuvre and raising the pressure for less than 5 seconds will not cause any problems and none have been reported to me.

It is difficult to define "forceful" and this leads to many people having problems inflating their ears because they blow gently down through the blocked nose and this does not provide sufficient back pressure to inflate the ears. Some patients I have told to blow hard and they immediately clear their ears within 1 or 2 seconds and their problem is solved. If one states that forceful attempts are to be avoided, then instructors tend to tell their people to be gentle and thus cause problems. Ear surgeons use a Politzer bag to forcefully inflate the middle ear producing much higher pressures than the expiratory muscles can ever produce. So I consider that it is the rare person who is capable of providing sufficient force to cause any trouble in inflating the normal ear. I have had one patient in 45 years.

I disagree that a sudden clearance of a blocked Eustachian tube allows the transmission of pressures into the middle ear chamber driving the tympanic membrane outwards and jerking the stapes (also) outwards. The linkage between the 3 middle ear ossicles is relatively loose to allow for sudden excess movements of the eardrum without transferring forceful movements to the inner ear. The tympanic membrane when moved quickly in or out, as is commonly and frequently done with the pneumatic speculum or with the more violent Politzer bag, does not involve shifting the stapes outwards. In fact if the middle ear pressure is raised, then the stapes is likely to be pushed into the vestibule rather than to be pulled out. That is my explanation for alternobaric vertigo.

The article otherwise is a tremendous achievement by David and will remain as a standard reference to anybody interested in the physiological and patho-physiological effects of the Valsalva manoeuvre whether it be the physicians' definition of the Valsalva or the ear surgeons' definition.

Noel Roydhouse.

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Key Words
ENT, physiology.

This letter has been shown to Dr Taylor whose reply follows.

242 Myers Street
Geelong, Victoria
23/8/96

Dear Editor

Thank you for giving me the opportunity to reply to Dr Roydhouse's letter.

I must confess that I found writing this paper somewhat frustrating. There were many areas which have been poorly written about in the literature. In fact, I had most difficulty in finding references relating to the middle and inner ears and the effects of barotrauma. My review is what I have been able to glean from the available literature.

I agree that Valsalva appears have written two descriptions of his manoeuvre. I found references that Valsalva described the manoeuvre as forced expiration against a closed glottis. I am therefore very interested to hear that he described it as a way of clearing out suppurative middle ear disease. I think that there is probably room for both definitions i.e. one against a closed glottis and another while holding the nose, as divers do.

The "standardised" manoeuvre has an open glottis to allow measurement of pressure yet has the soft palate blocking the transmission of pressure to the nasopharynx and therefore the Eustachian tubes and middle ears. I am confused as to the correct definition of the Valsalva manoeuvre!

It is not possible to really explain to a diver what we mean by "too forcefully" or "too long a period". I could find no help in the literature on this point, apart from a description of the standardised manoeuvre. I think the suggested time limit of 5 seconds is appropriate. If a diver needs to blow harder than he is used to, then he should be aware that perseverance with the manoeuvre may lead to complications.

David Taylor

Key Words
ENT, physiology.

MEDICAL ASSESSMENT OF FITNESS TO DIVE

7 Lyncroft Gardens
Ewell, Epsom
Surrey GU27 2HX, UK
23/8/96

Dear Editor

The Medical Guidance, MA1, used as the basis for fitness assessment of North Sea divers and followed internationally, is about to change. It is now recognised that, apart from police and military divers who are administered separately, there are 5 distinct categories of working diver in the UK: offshore, inland, scientific, media and the professional instructors of recreational diving. Each group will dive using a separate Approved Code of Practice.

A meeting will be held from the 14th to the16th of March 1997 in Newcastle upon Tyne to discuss these changes.

Many of the changes to the Medical Standards for Diving for four of these categories arise from the 1994 Edinburgh meeting and it is proposed that the fifth group, the scuba instructors, have a different examination. Some of the proposed changes in the 1997 version of the Medical Guidance may need interpretation or clarification by the HSE at this meeting.

The academic program will begin on the Friday morning, 14th of March, with Ralph Mavin, HSE's Chief Inspector of Diving, outlining the new Diving Regulations and its various Codes of Practice. The apparent differences between the different Codes of Practice will be highlighted. Dr Stephen Doherty of EMAS will then present the medical views of HSE relating to the procedures and responsibilities required from Approved Doctors for record keeping, appeals and other matters. The competent medical interpretation of the Guidance in relation to each diver being examined will depend upon the knowledge and skills of the examining doctor. The new structure will require appropriate training of Approved doctors to a high standard and, to maintain consistency of medical standards, will need them to maintain continuing medical revision.

A review of the major organ systems will be led by some of the key speakers from the Edinburgh meeting and there will be special emphasis on those aspects of the new Guidance in which some important changes are likely to be made, such as accepting some sports diving instructors with insulin-dependent diabetes. The meeting will be held in the Copthorne Hotel, Newcastle upon Tyne, situated beside the river some 10 minutes from the main railway station and linked by a regular metro service to Newcastle's international airport. The meeting will close at lunchtime on Sunday 16th of March, 1997.

This will be a milestone meeting for all Medical Examiners of Divers. PGEA and CME approvals are expected.

Details from Biomedical Seminars, 7 Lyncroft Gardens, Ewell, Epsom, Surrey GU27 2HX, UK. Fax + 44-181-786-7036. E-mail 106101.1722@compuserve.com .

David Elliott

Key Words
Fitness to dive, medical standards, meeting.

BOOK REVIEWS

PADI ENRICHED AIR DIVER MANUAL
PADI. PO Box 25011, Santa Ana, California 92799-5011, U.S.A.

Key Words
Mixed gas, nitrogen, oxygen, recreational diving, training.

This is one of the PADI series *Improve your Scuba*. It is the manual for the PADI Enriched Air Diver program. The list of acknowledgments at the beginning of the book is long and includes three members of SPUMS, Drs Des Gorman, Chris Acott (both on the Executive Committee of SPUMS) and Ray Rogers. Five people, including Drew Richardson (who is well known to members who have attended Annual Scientific meetings in the last 6 years), shared the writing and produced an accurate, comprehensive and easily understood manual.

The reader is led through the advantages and disadvantages of EANx. The suggestion that EANx will noticeably reduce the incidence of decompression illness is given short shrift. The advantage of EANx is increased bottom times at relatively shallow depths. That the major disadvantage is CNS oxygen toxicity, is quite rightly emphasised. Then the equipment, the precautions required to produce EANx and the need to check that the oxygen percentage actually is what is written on the label is discussed. Then there is a discussion on managing oxygen exposure, with emphasis on not exceeding the recommended oxygen partial pressure of 1.4 bar. This section also includes the consideration of lung toxicity as well as CNS toxicity. The use of the PADI Recreational Dive Planner (RDP) is assumed and covered well. However the description of the use of the RDP Wheel for multilevel dives using EANx takes careful reading and requires the diver to calculate equivalent air depths (EADs) underwater. If the reviewer took up EANx diving to extend bottom times he would be investing in a computer which handles EANx to avoid having to remember or calculate EADs!

It is unfortunate that the United States of America still clings to the British Imperial system of measurement, when even the United Kingdom has gone metric. As a result the book is compelled to calculate every formula and example, first in metric and then in imperial measurements. As all our readers know, calculating depths and pressures as multiples of 10 is much easier than as multiples of 33 or of 14.7. As the authors point out, depth and partial pressure of oxygen must be calculated carefully before the dive, and adhered to during the dive, for safe use of nitrox, which PADI quite truthfully refers to a Enriched Air or EANx. It is a pity that one dive instructing organisation has decided to call its oxygen enriched nitrox "Safe Air", while this might be true of the risk of decompression illness it is quite untrue when exceeding expected depths. PADI is to be congratulated on its choice of name.

Throughout the book there are exercises testing the reader's understanding of each chapter. If the text has been carefully read these are simple to work out correctly. Failure to get the right answers (these are at the end of the test) is a clear indication to the student to go back and read the chapter again. At the end of the book is a straightforward knowledge review to be filled in and handed to the instructor at the first training session.

In spite of much searching the reviewer could only find one error, in an illustration on page 31, where the text says 14 m in pressure group C and the circle on the table is in the pressure group B column. Perhaps it is a deliberate mistake to make the reader think!

Someone who has learnt everything in this book and had it reinforced by further instruction should have the basic knowledge about how to dive safely with EANx. But with only two dives under instruction one doubts whether the candidate will have the required skills to stay at or above the planned depths, to analyse mixtures for their oxygen content accurately and to calculate EADs and oxygen exposures correctly every time.

Navies allot much more time to teaching divers to use EANx than PADI is offering and naval divers undergo a lot more training with scuba than recreational divers do. One hopes that recreational divers are much more obsessive about depth control than the average SPUMS member at the Annual Scientific Meeting, but that happy state of affairs is unlikely.

That said this manual is an excellent start to PADI's venture into the enriched air diving world.

John Knight.

THE CASE FOR COMPUTERS
John Shuman
Agora, Inc., 105 W. Monument Street, Baltimore, Maryland 21201, U.S.A.
Available from Undercurrent Books, PO Box 831806, Richardson, Texas 75083-1806, U.S.A.

Key Words
Computers, diving safety.

This small, 47 page, booklet has grown out of 5 reviews of dive computers published in Undercurrent in 1995 and adds another 6 computers. In one advertisement in the

British diving magazine *Diver* (August 1996) there were offered computers by Aladin (5), Bridge, Cochrane (4), Oceanic (7) and Sea and Sea. Shuman's book covers computers by Cochrane, Oceanic (2), Scubapro (3), Sea and Sea, Uwatec (Aladin in the UK) and US Divers (3). It appears that he has chosen them as examples of three types of dive computer, basic, air integrated and adaptive.

The majority of computers in use fall into his basic category, being able to give information about depth and time, no-stop time remaining, nitrogen out-gassing and next dive schedules. The newer ones record a number of dives for playback and show time to flying.

Air integrated computers are the author's middle category. Showing how much longer the air supply will last is an excellent advance and making this or no-decompression time remaining the warning setter should provide safety. However divers can fixate on the air time remaining figures and ignore the decompression requirements.[1] In the discussion on hoseless air integrated computers no mention is made of the problems some have when the diver is in the "line of sight" between the transmitter and wrist worn computer, which is no longer receiving the transmissions.

The final category is adaptive decompression computers which adjust the calculation of nitrogen uptake and excretion for such things as rapid ascent, cold water and heavy work-load (calculated from air consumption).

The real value of the book is the first chapter. Here the author discusses how American sports divers really dive, taken from personal observation. He categorises 6 safety related skills for divers, dive planning, establishing accurate bottom time, controlling the rate of ascent, performing safety stops, measuring dive depth and group reliance. Group reliance is a concept that I have not met before and from the discussion it is obviously an "unsafety" factor. One example quoted is where a dive guide looked for the computer with the longest available second dive in the group and then used that person's available dive time for the group. Reading the stories of "experienced divers" diving from boats off California and Hawaii should be compulsory for all those interested in safe diving. No wonder the author states "Many divers ignored (or were deficient in) key safety-related skills". The author uses the frightening casualness of his fellows to emphasise that a computer will provide accurate depths and times, help with dive planning and ascent rate control and enable a diver to dive more safely. Seeing that only one of his unsuspecting observed divers ascended the last 4.5 m (15 ft) in 15 seconds I am surprised that he did not recommend using a computer with an audible ascent rate warning.

No book on diving computers can be up to date for more than a few months as new versions appear at short intervals and different algorithms are slipped into the new models of earlier computers. This book is an honest attempt to offer one person's views on computers in general and 11 in particular. There is a chapter on the down-side of computers emphasising that computer users often increase their risk of decompression illness by going longer and deeper. This theme runs through the book.

This book should be read by all those interested in diving safety.

John Knight

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SPUMS ANNUAL SCIENTIFIC MEETING 1995

ENERGY EXPENDITURE IN DIVING.

Marius Coetsee

Key words.

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In order to understand energy expenditure during diving it is necessary to have a sound knowledge of the energy systems of the body. Therefore a brief discussion of the energy systems and how they interact with each other is given at the start of this paper to assist readers.

All energy for muscular activity is derived directly from the high energy phosphates stores (ATP and CP) in the body. However, this pool of stored energy is relatively small and must be replenished continually by means of energy production. ATP and CP together would account for less than 10 seconds of all-out exercise. It follows therefore, that activities of a high intensity, lasting longer than 10 seconds. must rely heavily on the adequate production of energy. This would include all the activities of diving.

Replenishment of the energy stores is accomplished through production of energy by the anaerobic and aerobic energy supply systems of the body (Fig 1). During anaerobic production of energy, glucose is used as substrate

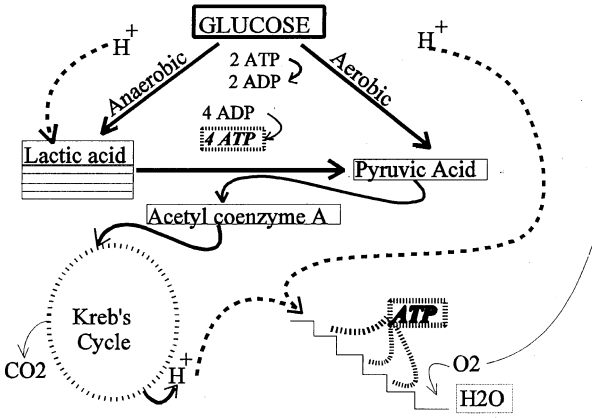

Figure 1. Production of ATP

and is broken down to lactic acid in a process termed glycolysis. During glycolysis two ATPs are consumed but four ATPs are produced, therefore there is a gain of 2 ATPs per glucose molecule. Aerobic energy production through glycolysis can also proceed without the formation of lactic acid, provided oxygen is available in sufficient quantities. Glucose (pyruvic acid) as well as fats and protein can further enter the Kreb's cycle to be metabolised. Hydrogens are released by the chemical reactions in the Kreb's cycle and glycolysis. These hydrogen ions are transported to the electron transport system where, by means of a series of reactions, they release energy in the form of ATP. At the end of the electron transport system hydrogen binds with oxygen, supplied via the aerobic support systems, to form water. If oxygen is unavailable, however, this process will immediately cease to produce energy, thereby leaving only anaerobic glycolysis for energy production with the resultant accumulation of lactic acid. The advantages and disadvantages of the anaerobic and aerobic energy systems are summarised in Table 1.

The demand on the energy systems of the body depend very much on the type of activity to which the body is subjected. The capacity of the energy systems to respond to the demand in turn depend on a variety of factors. By manipulating these factors an individual can optimise the ability to utilise energy effectively.

A high partial pressure of oxygen ($P0_2$) at cellular level together with other factors such as the availability of substrate, sufficient concentrations of oxidative enzymes to catalyse the chemical reactions, optimal pH and temperature all play a crucial role in determining the capacity of the aerobic system to meet the energy needs of the body and therefore determine the relative contribution of the aerobic and anaerobic systems (Fig 2). It must be remembered that if an individual is using more energy than the aerobic system can produce at any specific time, this energy is supplied by the anaerobic system with the resultant accumulation of blood lactate. Unchecked accumulation of blood lactate would soon lead to exhaustion.

TABLE 1

ADVANTAGES AND DISADVANTAGES OF THE ANAEROBIC AND AEROBIC ENERGY SYSTEMS

Anaerobic	Aerobic
Quickly available	Requires up to 2 minutes to function fully
Functions in the absence of 0_2	Requires 0_2 to function
Limited capacity	Very large capacity
Produces lactate which leads to exhaustion	Produces no lactate

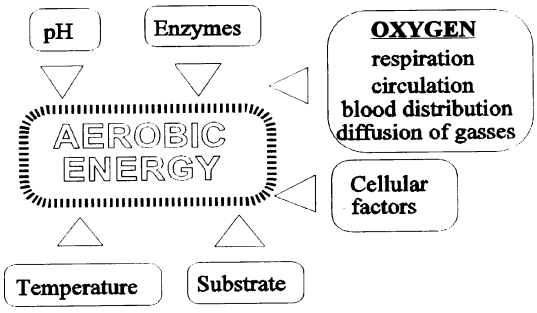

Figure 2. Factors determining the capacity of the aerobic system

In the light of the above discussion of the energy systems of the body, a description of energy expenditure during some aspects of underwater diving follows:

Scuba diving

The average recreational scuba diver always attempts to keep the intensity of the activity as low as possible, to improve air endurance. Therefore, most of the energy is derived from the aerobic system without using the full capacity of this system. The anaerobic system do not play an important role in a normal relaxed scuba dive. This observation is supported by research showing that the peak oxygen consumption (VO2 peak) and peak ventilation volume (VE peak) of scuba trained individuals are significantly lower ($P<0.00l$) during all out scuba fin swimming than the maximal oxygen uptake (VO_2max) and maximal ventilation volume (VEmax) as measured during all out running on the treadmill.[1] The absence of adaptations to the respiratory parameters suggest that the intensity of recreational scuba diving is insufficient to induce adaptations to the aerobic energy system.

However, this does not mean that aerobic and anaerobic fitness should be neglected by recreational scuba divers. Emergencies, such as having to cope with strong currents, will require a much higher reliance on both the aerobic and anaerobic systems and therefore scuba divers are strongly advised to specially train for aerobic and anaerobic endurance. As endurance is highly specific to the muscles that are active, it follows that such training should be fin swimming. Physical fitness remains one of the major contributors to safe diving.

Scuba diving where underwater work is performed or where special circumstances apply such as with very deep diving, brings into effect factors that affects the relative contribution of the energy systems and the ability of these systems to meet the energy needs.

Shore entries through surf can be dangerous to unfit individuals, especially if they also possess relatively low aerobic capacities. While battling through the surf the energy need of the working muscles could easily be higher than can be supplied by the aerobic system, with the resultant accumulation of lactate in the blood. This in turn would tend to lower the pH of the blood. In an attempt to buffer the change in pH, respiration will increase causing a sensation of shortness of breath. This does not only lead to a much greater air consumption but also poses a real danger of panic developing. Proper warm up of the aerobic support systems and especially aerobic fitness would greatly help to prevent this situation from developing.

Underwater hockey

Contrary to common belief, research has shown that energy is derived not from anaerobic but mainly from aerobic sources during underwater hockey.[2,3] Experienced underwater hockey players have a distinct pattern of play, i.e. 10 seconds of submersion and 10 seconds of recovery on the surface.[4] Submerged periods rarely exceed 1O seconds. The intermittent nature of underwater hockey allows for sufficient recovery of the oxygen stores in the body during the 10 second surface interval for the aerobic system to adequately support energy consumption. During maximum intermittent exercise, such as found in an underwater hockey match, the oxygen saturation of arterial blood is maintained above 91.5%.[3] Blood lactate levels of underwater hockey players after 12 minutes of intermittent exercise are about 5.1 mmol/l indicating only a moderate contribution of the anaerobic system to energy supply.[2] Performance (underwater time) seems to be limited by an increase in the urge to breath caused by a significant ($P< 0.001$) increase in $PaCO_2$.[3]

Scuba orienteering

The aim of scuba orienteering is to cover a distance of approximately 600 m in the shortest possible time.

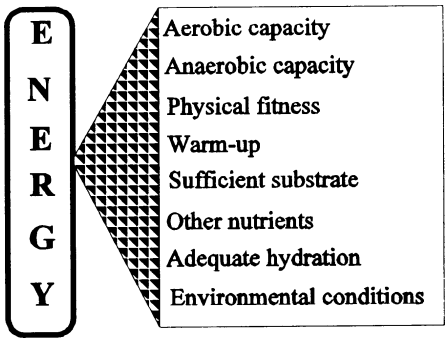

Figure 3. Factors affecting energy supply

Therefore it is understandable that both the aerobic and anaerobic systems would contribute significantly.

Spearfishing

Long underwater breath hold periods during spearfishing necessarily place a much greater emphasis on the anaerobic system for energy supply. The spearfisherman however always tries to conserve energy by limiting physical activity to the minimum.

Discussion

In underwater sports, particularly where a possible shortage of energy might lead to life threatening situations, proper knowledge and effective control of the factors affecting a sufficient supply of energy are very important. The following aspects must be considered by participants to ensure safe and effective participation in exercise (Fig 3):

l Each individual has a maximum potential for oxygen consumption (VO2max) and therefore, for producing energy via the aerobic system. This potential is determined by hereditary factors and can be adapted only moderately through exercise. The physical working capacity of two individuals who are equally fit can differ substantially. Working in a group could mean that some individuals are working at an easy level without getting exhausted while some might be working at a level which, for them, would quickly lead to exhaustion. Instructors and trainers should be particularly aware of this and not expect the same performance from every individual in a group. During group work the pace should be set by the weakest individual in the group. Individuals with relatively low hereditary aerobic potentials should compensate by being aerobically fitter than their "aerobically endowed" counterparts if they wish to achieve the same results.

2 Physical fitness can greatly increase an individual's performance capabilities. Not only do the metabolic systems adapt to become more proficient in producing more energy with the same oxygen consumption but the mechanical efficiency also improves. This means that the available oxygen is used more economically and therefore more work can be performed with the same oxygen consumption.

Unfit individuals can exercise up to approximately 55% of their maximal aerobic capacity before blood lactate starts to accumulate faster than it can be metabolised, with a resultant accumulation of blood lactate concentration. Fit individuals may exercise at a level as high as 85% of their maximal aerobic capacity before lactic acid will start to accumulate in the blood. It must, however, be remembered that adaptations of this nature are highly specific to the type of activity or sport and that very little cross training occurs. In order for divers to improve this capacity they must engage in fin swimming under conditions simulating those conditions they want to get fit for.

3 Proper warm-up plays an important role in the optimal functioning of the aerobic system. Aerobic energy production depends on a sufficient supply of oxygen reaching the muscle cells. However, the systems responsible for the delivery of oxygen to the cells function only at the level required by the intensity of exercise at the time and require time to adapt if the intensity of exercise is changed. If the exercise intensity is suddenly increased, the anaerobic energy system will supply the energy which the aerobic system is temporarily unable to supply and lactic acid accumulates. Provided the increase in exercise intensity is not too great and remains submaximal, the oxygen supply systems will soon adapt to allow sufficient oxygen to reach the cells for the aerobic systems to supply the total energy needs. However, if the intensity increase is too great, sufficient lactate would accumulate to compromise efficient functioning of the aerobic energy system and exhaustion would follow. A proper graded warm-up procedure on the other hand would allow the aerobic system to adapt gradually, with the result that very little lactate accumulates.

In most underwater activities it is easy to warm up by simply starting at a slow pace and gradually increasing the intensity of exercise. During most recreational scuba dives the intensity of the entire dive is low to moderate and therefore there is no need to warm-up. Other dives, e.g. a shore entry, however, would necessitate prior warm-up as the initial activity itself might be so strenuous that no warm-up is possible during the actual activity.

4 Energy production also depends on the presence of sufficient substrate at cellular level. Activities of very high intensity rely mainly on carbohydrate and as the carbohydrate stores of the body is fairly limited it follows that such activities, if carried on for extended periods, might result in hypoglycaemia developing. Intermittent activities, such as found when a number of games are played per day, as in an underwater hockey tournament, may lead to hypokinetic conditions developing if not compensated for through the intake of carbohydrates. Prolonged spearfishing might also result in a shortage of blood glucose developing.

5 Other nutrients such as vitamins, minerals and proteins all play an important role in the production of energy. A shortage in any of these could compromise the production of energy and lead to a reduction in performance. A shortage in these nutrients normally takes some time to develop and a balanced diet should prevent it happening.

6 In order to maintain an optimal medium for metabolic processes, adequate hydration is of utmost importance. Dehydration could severely compromise exercise performance by, amongst others, adversely affecting energy production. Most diving activities comprise lengthy outings in which the individual is subjected not only to environmental factors contributing to dehydration, but also to circumstances that can lead to insufficient intake of water. Diving itself leads to an acceleration of dehydration.

7 Environmental factors such as heat or cold could also affect energy production by creating intra-cellular conditions unfavourable for the chemical reactions of metabolism.

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Dr M F Coetsee's address is Department of Human Movement Science, University of Zululand, Private Bag X 1001, KwaDlangezwa 3886, South Africa. Tel/Fax + 27-351-93916.

CARDIOVASCULAR PROBLEMS AND DIVING

Fred Bove

Key Words
Cardiovascular, fitness to dive, safety, treatment.

In the US the Diving Alert Network has a list of physicians for medical consultation. I am one of the cardiologists on the list and I get about a call a day about some cardiac question that raises issues of safety or fitness for diving. I will discuss the commoner categories such as coronary artery disease, congenital heart disease, valvular disorders, arrhythmias, conduction disorders, pacemakers, some of the common cardiac drugs used in the treatment of hypertension and some other relatively benign medical conditions that raise questions about the use of the drugs rather than the condition itself.

Coronary disease

Coronary disease is the most common cardiovascular problem in divers and in particular divers above the age of forty. This is because of the increased incidence of cardiovascular disease in the population as age increases.

In most cases the history gives the most reliable information about coronary disease. If a patient has a history of myocardial infarction, or angina, coronary disease is present. If they have had bypass surgery or an angioplasty or any other procedure to relieve coronary occlusion, they have coronary disease. We realise now that when we see a single lesion the vessels are more diffusely involved, although all the disease may not show clearly on a coronary angiogram. One can now put small echo probes inside the coronary arteries and visualise the arteries from the inside. When that is done one finds the extent of disease is much greater than the visible narrowing on an angiogram.

Another warning is increased dyspnoea with mild exertion. Some patients do not get pain or tightness with angina, but rather get a sensation of dyspnoea. Dyspnoea on exertion, particularly of recent onset, needs to be thought about as an anginal syndrome, although one has to look for pulmonary disease as well.

Arrhythmia with exercise is a fairly ominous finding and it is always worthwhile asking people if there is a history of sudden death in their families or among relatives, because arrhythmia with exercise can be a variant of angina. Ischaemia not only produces pain or tightness in the chest, but it also can produce primary arrhythmias because of ischaemic irritability of the myocardium. Some patients do not get angina but get ventricular tachycardia as the first sign of angina. People with a history of syncope with exercise need to be considered as a special group and the whole symptom complex has to be sorted out before one can allow them to dive. In the United States, the most common cause of sudden death in diving is coronary disease.

One needs to look at the risk factors which make people more likely to have coronary disease. The common ones are cigarette smoking (the most common reason why people have coronary disease) and then hyperlipidaemia, high blood pressure, stress, lack of exercise and last of all family history which is probably the least important factor except in the very extreme hyperlidaemias.

The incidence of coronary disease increases with age. Oestrogens protect women from coronary disease until the menopause and then their rate of rise of coronary disease exceeds that of males up into the sixties and seventies. The age adjusted rates are higher in women compared with men after the age of fifty. This is important because there are many people in the United States who complete their lifetime work requirements in their fifties or sixties and want to take up a sport. Many people in their late fifties, sixties and even early seventies learn to scuba dive as a recreation. At these ages women are at the same, and probably greater, risk than men from coronary disease. So both males and females need to be tested to be sure that they do not have ischaemic cardiac disease when they start a new program, such as scuba diving, later in life. Age is a risk factor and it is the only one for which there is no pill to reverse.

High blood pressure is a risk factor and one can take a pill to reverse it. High blood pressure means a systolic pressure of greater than 150 mm Hg and a diastolic pressure greater than 95 mm Hg. High blood pressure is an important risk factor which is easy to detect. It is the number that is important. If the number is normal all is well. If the number is abnormal then something has to be done about it, as one should not tolerate high blood pressure, or even borderline blood pressure, because there is an increase in risk. Individuals who have hypertension and hyperlipidaemia, are older, smoke, do not exercise and want to dive are at very high risk. They need screening to be certain that there is no underlying cardiac disease that might put them at risk of sudden death while diving.

A person with coronary artery disease gets a progressive increase in plaque formation and reduction in lumen until an occlusion occurs and an infarct follows. There is not much change in flow reserve until around seventy percent reduction in cross sectional area, so it can be a long time before a person becomes symptomatic. It is important to test people with high levels of exercise because otherwise one cannot tell whether they have occlusions, or partial occlusions.

There are other, rarer, risk factors. For example, a person develops Hodgkin's lymphoma at age 17 and is treated with chest radiation, neck radiation and perhaps a laparotomy to look for nodes. He or she is cured. Twenty years later they want to learn scuba diving, but they have coronary disease from the radiation injury to the coronary endothelium. Someone who has had chest radiation, even though they may appear to be at relatively low risk, ought to be looked at to make sure they do not have intrinsic coronary damage producing ischaemia.

There is also coronary spasm which can cause narrowing on an angiogram. It is not a fixed lesion, but a transient one caused by active smooth muscle in the region of narrowing. Spasm is associated with local endothelial injury. The endothelium produces background vaso-relaxation so when the endothelium is not functioning normally, local spasm can occur and cause ischaemia. Local spasm in a person who has otherwise no coronary disease is very dangerous because if there are no collaterals protecting an ischaemic circulation, this will often induce ventricular tachycardia as the first symptom. Coronary spasm is very difficult to deal with because the usual treatments, such as angioplasty, stenting and even bypass surgery do not work.

An individual with a coronary artery narrowed down to about 90% would be at risk for ischaemic events while performing exercise of any kind and usually has symptoms at rest. Such a person would be at risk for ischaemia while jogging, while playing most sports, and while doing anything related to the physical activity of diving. Such a lesion, if it is known, would prevent anyone from going through a diving program. Could this person test normal? The answer is no, such a lesion would show a filling defect using thallium. Collateral circulation is unlikely to compensate for a lesion of this nature.

One such patient was a fifty five year old businessman, a very aggressive man who refused any further therapy other than anti-coagulation and aspirin. A year later, when we restudied him the narrowing was gone. Thrombosis on a minor narrowing had produced almost complete occlusion. As time went by and the thrombus cleared, the artery reopened and in fact he passed a stress test. He did not want to go diving, but he certainly wanted to go back to playing tennis and jogging, and we let him.

One should ask older candidates for diving if they have angina or if they have chest discomfort. If they experience chest discomfort while having dinner one certainly does not want to clear them for diving unless the chest discomfort is proven to be dyspepsia. If it is true ischaemic chest discomfort at rest then clearly the person should not be allowed to undertake any kind of physical activity until sorted out.

Coronary artery problems

Look for the patient who has risk factors. A forty year old man who runs three marathons a year, has a cholesterol of 110 mg, a normal blood pressure and does not smoke has a very low risk for coronary disease. If on the other hand he is 19 kg (40 lb) overweight, smokes, has diabetes, hyperlipidaemia, hypertension and takes no exercise he is at risk for a first coronary event while diving. History and the risk factors are important.

I think it is most important to look at the individual as a whole. Remember that coronary disease in the absence of any major myocardial injury shows no abnormalities on physical examination. For people with risk factors it is not at all appropriate to do a clinical cardiac examination and a resting electrocardiogram and say that they can dive, because one can not detect coronary occlusion or partial coronary occlusion purely by physical examination alone. One has to generate some stress on the myocardium and determine that the individual does not have ischaemia during that stress. That can be done by history taking if the person does a certain amount of heavy physical exertion every day without any symptoms, or if he is running a marathon three or four times a year and training doing 64-80 km (40-50 miles) a week of running. One asks about the intensity and duration of the exercise, the presence or absence of symptoms and gains the information that the individual can handle stresses. If one does not have that information and if the individual is in a high risk category then one should do a stress test to make sure there is no ischaemia present.

The easiest thing to do is a regular exercise stress test which will identify the person at risk for exercise. It will not always reveal if there is coronary disease. The problem with doing a regular exercise stress test in that one will miss the presence of some coronary disease in about twenty percent of individuals. However if there is critical coronary disease that would cause ischaemia with exercise this will usually be picked up at the rate of 90-95% on a regular stress test. It will not pick up the sub-clinical 40-50% narrowing of an artery but that is not going to cause sudden changes in the patient's status. One can pick this up with a stress test with myocardial imaging using thallium injection. Thallium goes into the myocardium using the same pathway as potassium, so if there is appropriate blood flow it will be imaged as normal distribution in the myocardium. If the individual has an occluded artery at fifty per cent or greater thallium will show that there is a partially blocked artery by demonstrating an area of inadequate uptake in the region of narrowed blood vessel.

If an individual shows severe ischaemia with exercise one must make a judgement about whether it ought to be treated. In the United States, most patients with an ischaemic response on a stress test have a cardiac catheterisation. This behaviour is changing because

outcome data show that there are many patients whose outcomes do not change whether we do an angiocardiogram and intervention or treat medically. Airline pilots with a positive stress test must have an angiogram to be able to go back on flight status. The same applies to military pilots and some other military personnel. If they have a positive stress test they need an angiogram to determine whether it is a true positive and what the extent of disease is. I would not recommend somebody have a cardiac catheterisation just to take up sport scuba diving. If one finds a high risk situation from the initial part of the examination then one could recommend it to the individual who may be at risk from other things such as the workplace or other kinds of activity.

By the time that most sport divers develop coronary disease, they already have a certification card and in the United States there is no need to come back to get recertified or requalified. People show up on a dive boat with a midline sternotomy scar and no other information. Hopefully the conscientious sport diver who has coronary revascularisation will return to a physician before going back to diving. The question that one has to ask about coronary revascularisation is whether it is complete? In some individuals one can identify a single blood vessel, perhaps a very large left anterior descending artery, that could put the patient at serious risk if it occluded. That is an indication to do a revascularisation either by a percutaneous intervention or a internal mammary bypass graft. If that is the only lesion, the ventricle is intact and no infarct has occurred, when that artery is bypassed, the person should have excellent exercise tolerance if revascularisation is complete. To get that individual back in good condition, wait for the wounds to heal and then continue very rigid risk factor modification to prevent any further progression of disease. Such people go back to their workplace without limitations, to their sports and to diving, because when tested there is no evidence of ischaemia.

Most thoracic surgeons nowadays do midline sternotomies for almost everything except descending aortic surgery. They are fairly careful to stay out of the pleural spaces. That is not always possible to do but a large number of patients with midline sternotomies do not have their pleural spaces opened. Once the sternal wound has healed the chest dynamics are pretty well restored. There are many people diving safely after having had a midline sternotomy. If the revascularisation was complete the next question is “Has there been a re-stenosis?”. This is particularly important in angioplasty, stents and anything that is done percutaneously. With balloon angioplasty the plaque is crushed and the artery torn leaving a denuded, torn, segment of blood vessel, so it is no wonder that there is re-stenosis. All these processes have a very high rate of re-stenosis. I think it is lucky that fifty percent of them heal without re-stenosis, but then fifty percent of them heal with re-stenosis. I generally tell a patient that a routine angioplasty has a fifty percent re-stenosis rate in six months. Some people get angina, have an angioplasty done and postpone their annual diving trip by a month so they can get the procedure done. They should really postpone it by six months to get past the early re-stenosis period. Having an angina attack during exercise is not a good thing while diving. They should not be diving until absence of ischaemia can be confirmed by stress testing.

Another question is “Is angina present?”. Bypass surgery and other revascularisation procedures do not always abolish angina. They may raise the threshold because there may have been severe multi-vessel disease and what was taken care of was the worst of the vessels, but not all of them. The patient still has ischaemia and will get angina with enough exercise. But the angina threshold is now high enough to allow a normal life, and return to work, but it may not be adequate to return to a sport like diving. Can one see ischaemia with reasonable workloads, and are there any arrhythmias? Again one would need an exercise test.

Congenital heart disease

People with cyanotic congenital heart disease should not be diving. Most have real trouble just existing. They are prone to pulmonary oedema. They are prone to thrombotic events because of their high haemoglobin, they have very poor exercise tolerance and their arterial oxygen saturation is usually down around 60-70% in many cases.

The atrial septal defect (ASD) is the ultimate residual of a patent foramen ovale. The flap that is supposed to close the opening does not grow, leaving a fixed opening between the two atria. This is a secundum defect. The first septum closes the juncture between the ventricles and the atrium. A septum primum defect is a much more complex lesion, often connecting the ventricles and the atrium, and often resulting in cyanotic heart disease. In secundum type atrial septal defect, the shunt goes from left to right as the left atrial pressure is higher than the right. Later in life when the lungs have been damaged by this high flow, the shunt can reverse. Even when the shunt is primarily left to right anything that raises venous pressure, whether on a long term basis or short term basis, may cause the shunt to reverse. Standing on one’s head, doing a Valsalva manoeuvre and water immersion are all ways that a primarily left to right shunt can instantaneously reverse and carry objects over into left atrium and so into the arterial circulation. Such episodes can cause strokes, so any uncorrected ASD is a contraindication to diving. Cardiologists generally recommend repair even for relatively small atrial septal defects. If the pulmonary blood flow is 1.5 times the systemic blood flow we recommend closing the shunt. In fact, we are getting even more conservative now, because of the risk of embolic events all through life, in the presence of even a small ASD.

Ventricular septal defect (VSD) is the most common shunt-producing congenital anomaly. Most are very small VSDs in the upper septum. They produce small left to right shunts, are inconsequential in terms of haemodynamics and they do not cause paradoxical embolisation. They usually produce a murmur. The only concern we have for a small ventricular septal defect is the risk of infection. These individuals are given prophylactic antibiotics when having dental work to prevent endocarditis, but often surgical repair is not recommended when the shunt is very small. Such a VSD is not a contraindication to diving or any other sport. Haemodynamically significant VSDs require surgical closure.

Patent ductus arteriosus is a left to right shunt into the pulmonary artery, the result of an incomplete closure of the foetal ductus. It does not reverse unless the patient gets severe pulmonary disease later in life and develops pulmonary hypertension. Early in life, until the thirties or forties, these can go undetected. They will produce a small murmur that is not always easy to hear. Eventually somebody will hear the murmur, do an echocardiogram and find the patent ductus arteriosus. This is not a contraindication to diving. They do not produce paradoxical embolisation. The concern is the size of the shunt because a large shunt will ultimately damage the pulmonary vasculature. If an individual has a small patent ductus and it has been decided it is not in need of surgical repair, then he or she can undertake diving without additional risk.

We heard a very good review on patent foramen ovale (PFO) from Paul Langton.[1] Basically there is an oblique hole in the atrial septum to allow arterialised placental blood to go into the left atrium and be distributed by the arterial system in utero. At birth, as left atrial pressure rises above right atrial pressure the two sides of the hole are forced into contact. In most people the flap scars down against the septum and produces a complete closure. Somewhere about 25-30% of the population has some patency of this foramen, however, the flap is always pressed up against the septum so it is haemodynamically inconsequential. There is no shunt unless one does things that force right atrial pressure higher than left atrial pressure, such as standing on one's head, being immersed in water, doing a Valsalva manoeuvre, or a cough. All those can force the foramen open and, if there are objects in the right atrium at the time, they can cross to become arterial emboli. There is a study underway (May 1995) in the US on unexplained strokes in young people. There appears to be a risk that small thrombotic emboli can pass through PFOs and cause strokes.

Wilmshurst[2] reviewed one hundred and nine asymptomatic divers, 24% had PFOs on transthoracic echocardiogram. In fifty divers with early decompression sickness (DCS) he found that 66% of them had a PFO. Late presenting DCS divers had 26% with a PFO, the same as the controls. It is interesting that he had a small group of divers with limb bends who had a low incidence of PFO, but in 14 divers who had skin bends as a primary event, 86% had a patent foramen ovale. This needs to be looked into further because it has not been corroborated by other studies. I know personally people who get skin bends frequently, with no other symptoms. One wonders whether that is related to a PFO. The 66% was statistically significant, although some people argue that the way Wilmshurst did his statistics was not quite valid. However Wilmshurst's data suggests that a PFO may contribute to early decompression sickness.

Cross[3] examined 78 professional and sport divers with echocardiography and about a third (26%) had PFOs. They had all done a lot of diving and none of them had ever had decompression sickness. I think the message here is that is if one dives safely and does not develop a lot of bubbles, it is likely that one will not get a problem even if one has a PFO.

I have done a meta analysis of four studies[2-5] of PFOs and divers (Table 1) which allows calculation of the risk of DCS. The incidence of DCS in sport divers, about 0.03%, is used as a base for the general incidence in the diving population.[6] The risk ratio for DCS is increased by a factor of about 5 for individuals with a PFO. For sport diving the overall risk remains low and the significance of these small differences is questionable. In my analysis there is a slight increase in risk of DCS in divers with PFOs. If one is diving safely it is very unlikely that there will be a problem from a PFO. One third of us here have a PFO and one third of us are not going to get bent this week.

There have been a number of studies on multiple dive profiles. Significant numbers of divers bubble, particularly in multi-dive, multi-day type exposures, four or five dives a day, five or six days a week. One will find a large number of divers who have at least one or two incidents of bubbles in that period of time.

In the DSAT studies of the PADI tables the six dives a day for six days was stopped because there was a single incident of decompression sickness, but a third of the divers had some bubbles at one time or other. In the four times a day for six days about ten percent of the divers had bubbles. When investigators went on some live-aboards and did uncontrolled surveillance of divers who were doing five or six dives a day for six consecutive days they found 18% of the divers had at least once incident of bubbles some time during their exposure. We can bubble, and it appears that bubbling occurs with a large gas load. That makes sense. Now some organisations are recommending no more than three dives a day and to take a break in the middle of a week of diving to get rid of residual nitrogen. The idea is to reduce the number of bubbles formed. I think that if a patent foramen ovale is going to cause trouble it would be in the kind of environment with increasing gas loads over a five or six or seven day period with the possibility of

TABLE 1

METANALYSIS OF PFO AND DCS PAPERS

All DCS

DAN 1994 report estimated total divers at 2,500,000.
1,164 were injured, incidence 0.05%. DCS 871, incidence of DCS 0.03%.

Source	PFO present No DCS	PFO present DCS history	No PFO No DCS	No PFO DCS history
Moon et al. 1989	19	11	157	19
Moon et al. 1991	18	66	72	24
Wilmshurst et al. 1990	26	47	83	50
Cross et al. 1991	26	-	52	-
Totals	89	124	364	93

Sensitivity	Probability PFO present and DCS history	0.571
Specificity	Probability No PFO and no DCS	0.804
Posterior Probability	Probability DCS history and PFO	0.00101
Posterior Probability	Probability DCS history, no PFO	0.00019
Posterior Probability	Probability No DCS, no PFO	0.9998
Posterior Probability	Probability No DCS and PFO	0.9990

Type II DCS

DAN 1994 report estimated total divers at 2,500,000.
1,164 were injured, incidence 0.05%. Type II DCS 644, incidence of Type II DCS 0.03%.

Source	PFO present No DCS	PFO present DCS history	No PFO No DCS	No PFO DCS history
Moon et al. 1989	19	11	157	19
Moon et al. 1991	18	29	72	30
Wilmshurst et al. 1990	26	33	83	17
Totals	63	73	312	66

Sensitivity	Probability PFO present and DCS history	0.525
Specificity	Probability No PFO and no DCS	0.832
Posterior Probability	Probability DCS history and PFO	0.00080
Posterior Probability	Probability DCS history, no PFO	0.00015
Posterior Probability	Probability No DCS, no PFO	0.9999
Posterior Probability	Probability No DCS and PFO	0.9992

Data taken from references 2-6.
Posterior Probability is the inverse of sensitivity and specificity calculated via Bayes Theorem.

bubbling some time during that exposure.

Valvular disease

The important valves are the mitral and the aortic. The pulmonary valve can be stenosed, which is usually a benign condition unless severe, but mitral valve disease and aortic valve disease are still fairly common. People with mild mitral stenosis, a leaking aortic valve, or a prosthetic valve, may all want to go diving.

Mitral valve prolapse is present in probably 10-12% of the population and as a single entity it does not have any consequence. In mitral valve prolapse the mitral valve

leaflets move behind the plane of the AV ring during systole. This process alone is a benign finding. In essence the valve can be considered too big for the orifice and it bulges back into the atrium. At 10-12% prevalence it is similar to being left handed. It would be quite unreasonable to say that everyone left handed has some kind of disease. Arrhythmias or mitral regurgitation can be associated with mitral valve prolapse. There is a very rare incidence of sudden death because of the prolonged Q-T interval syndrome. With arrhythmias, treat the person as an arrhythmia patient, with mitral regurgitation treat as a mitral regurgitation patient and with sudden death syndrome or an identified prolongation of the Q-T interval, do not let them go diving.

The concerns for both mitral and aortic valves are regurgitation and stenosis.

Critically stenosed aortic valves give the heart a relatively fixed output. They are very risky valves during exercise as vasodilatation of the skeletal muscles will reduce peripheral resistance and the blood pressure will drop. As a result the patient gets syncope with exercise and one day may not wake up. Aortic stenosis is associated with syncope and with sudden death during exercise. The question to ask is "Do you get light headed or pass out with exercise". If the response is "Yes", one needs to rule out aortic stenosis. This lesion is dangerous and is a contraindication to diving.

However there are some children with congenital aortic stenosis who grow up getting along fine. I have patients who jog five miles a day in spite of significant aortic stenosis. I say do not do it. And they come back a year later still jogging five miles a day. But for the most part, aortic stenosis, with a critically narrowed valve (about one square centimetre or less) is a contraindication because of the risk of syncope or sudden death during exercise.

Mitral stenosis is of concern because of elevated pulmonary venous pressure. When the cardiac output goes up with exercise the flow across the mitral valve increases, the left atrial pressure rises, pulmonary venous pressure rises and the patient develops pulmonary oedema. This seldom produces sudden death. In patients with mitral stenosis occasionally the first evidence is pulmonary oedema with exercise. The patient with mitral stenosis should not be diving for the same reason that they cannot go jogging or do other forms of exercise, because they get lung congestion. If there is a minimal pressure gradient and no significant obstruction with good exercise tolerance, they can go diving. But moderate to severe mitral stenosis is a contraindication.

Severe mitral regurgitation, like mitral stenosis, will cause elevations of pulmonary venous pressure during exercise witth lung congestion and pulmonary oedema. People with moderate to severe mitral regurgitation should not be diving. When one is immersed in water about 500 ml of blood shifts into the central circulation. This can cause problems. A 58 year old physician, who of course signed off that he was in excellent health and had no medical history, had trouble breathing during a dive. We pulled him from the water in acute pulmonary oedema. Fortunately there was oxygen on board and with that he improved. It turned out that he had significant aortic regurgitation and had actually had endocarditis once. The combination of exercise and the central fluid shift gave him acute pulmonary oedema. We treated him and he was fine, but somehow this physician inadvertently forgot to tell us that he had severe aortic insufficiency!

When mild, aortic regurgitation and mitral regurgitation are of little consequence. If severe they produce heart failure during exercise which means dyspnoea on exertion and sometimes pulmonary oedema during exercise. They seldom result in sudden death during exercise. Mild regurgitant lesions are benign. One has to examine the individual's exercise tolerance and how cardiac function responds to the workload.

Mild stenotic lesions need to be carefully examined to make sure that they are not producing significant obstruction.

There are people with prosthetic valves diving, although the Divers Alert Network (DAN), in the USA, usually tells people with prosthetic valves not to dive. In the absence of any other cardiac disease the prosthetic valve will allow an individual to perform moderate amounts of exercise which is fine for diving. People with prosthetic valves play tennis, jog, play other sports and they do not have serious problems. The issue with prosthetic valves is anti-coagulation. DAN has raised the issue of having haemorrhagic otitis media with an ear squeeze. I have seen twenty or thirty people diving with prosthetic valves and none of them has ever had a problem with bleeding from an ear squeeze. Obviously they need to teach themselves to be careful with their ears. Bleeding does not seem to be a major concern although the individuals need to be warned about injuries and about making sure their ears are clear when they dive. A sinus squeeze would be likely to cause a haemorrhagic sinusitis.

Arrhythmias, conduction defects and pacemakers

People who get supraventricular tachycardia (SVT) with exercise, or spontaneously, should not be diving. SVT with exercise in subjects with accessory pathways can degenerate into sustained tachycardia and ventricular fibrillation. Supraventricular tachycardia is quite common. It appears that somewhere around 60% of young people with attacks of SVT have an accessory bypass track connecting the atrium to the ventricles or the AV node which is inducing the supraventricular tachycardia. They are treatable by catheter ablation or medication and treatment

generally stops the arrhythmia. If somebody has a serious recurring supraventricular tachycardia tell them to have it treated and come back for evaluation when the rhythm has been eliminated.

These arrhythmias can be induced by stimulants, such as caffeine, decongestants that have adrenalin like compounds, amphetamines and stress. Remove the stimulants and often the arrhythmia goes away.

There are some young people, with no evidence of cardiac disease, who have atrial fibrillation. They are well controlled and have excellent exercise tolerance. They may need to be on anti-coagulation because of the risk of embolisation, but they can dive. The problems of anti-coagulation need to be carefully explained.

I do not worry about premature ventricular contractions (PVCs)when they disappear with exercise. If they do not, or the patient has PVCs associated with ischaemia, these PVCs may increase in frequency and induce severe rhythm problems when diving.

There are many pacemakers that sense the need for increased output with exercise and adjust the cardiac rate to accommodate the exercise. Rate controlled pacemakers allow people to exercise normally. For young individuals who need a pacemaker, perhaps after a viral infection destroys the conduction system or congenital heart block, a rate sensitive, dual chamber pacemaker, will allow normal function for all physical activity. The pacemakers are made to withstand pressures to about 39 m (130 ft). As these individuals have otherwise normal hearts they have normal exercise tolerance and can take up sport diving. However they will not get into military or commercial diving.

An implantable defibrillator is implanted because an individual has had an episode of sudden death. Normally sudden death induces unconsciousness and the theory behind the implantable defibrillator is that when one gets a run of ventricular fibrillation blood flow to the brain stops for a few seconds so when the shock comes one does not remember it. Unfortunately in water, going unconscious for twenty seconds before the defibrillator fires means that one is already drowning. I do not advise diving for these people. I know of two divers who entered the water with these devices. One was a seventy year old with severe heart disease, who should not have been diving. His fired and a diving instructor next to him also felt a mild shock! The other was an already certified diver, who at the age of 36 had a sudden death episode and had a defibrillator implanted. He asked, by letter, whether it was reasonable for him to dive. I said that I did not think so, because sudden death in the water is not good for the diver or the buddy. It is very upsetting when your diving buddy dies right in front of you. But while my letter was in the post I received another letter from him saying "All is well, I already did a forty foot dive. Do you think I could go to eighty feet?" I sent him the same letter again saying "Do not do it", but all we can do is advise.

Fitness

One can assess conditioning by measuring maximum oxygen consumption. When one starts exercising there is an increase in oxygen consumption which peaks at a certain work intensity. Everyone has a peak beyond which one cannot increase one's 0_2 intake. Above 70% of one's peak one is into anaerobic exercise, generating a lot of lactate, becoming acidotic and hyperventilating, ultimately one has to stop exercising. The maximum can be varied by conditioning. People with a low maximum oxygen consumption have less exercise tolerance and so are more at risk for getting into trouble when they need to exercise in the water.

The maximum one can achieve declines with age. If you are thirty years old and in reasonably good condition your maximum would be about 40 ml of oxygen per kg per minute. That is about 13 mets. Around age seventy the maximum is down to about 30 ml. The seventy year old diver is not going to be able to handle the extremes of exercise needed to extricate himself from serious problems in diving the way the reasonably fit thirty year old could. The best way to cope with the differences in physical capacity with age is to gain wisdom so that one stays out of trouble.

For conditioning we suggest a target heart rate of 0.7 x (220 - age). Warm up for 5 minutes, exercise at the target heart rate for about 30 minutes then do a five minute cool down. Doing this four or five times a week will get most people in good condition for diving. For a diver it is sensible to do swimming as part of this program because swimming is what one is training for.

Antiarrhythmic Drugs

Most drugs are not of concern in a patient with true heart disease. Someone with heart failure who is on digitalis will not be fit to dive because he has heart failure and poor exercise tolerance. The same applies to diuretics. Perhaps even more, as in a warm environment, with sweating and loss of sodium and fluid by normal means, one does need diuretics. One must warn people taking diuretics that if they are in the tropics they ought to reduce the dose to make sure they do not get dehydrated.

Digitalis is often used, probably incorrectly, to prevent supraventricular tachycardias, particularly in young people. I meet many people in their twenties and thirties who are on digitalis because they had one episode of tachycardia associated with a long night of alcohol intake. I usually stop the digitalis, although if someone has been on a drug for fifteen years it is difficult to get them to stop it.

I do not like to see people diving while taking anti-arrhythmic medications. The reason is that most of them have cardiac problems. The first thing one has to understand is what their rhythm is.

For example we are using amiodarone to prevent atrial fibrillation in people who have paroxysmal atrial fibrillation. Amiodarone is an extremely good drug for managing atrial fibrillation. In fact it is much better than a combination of three or four other antiarrhythmic drugs. A 30 or 40 year old person may be on a small dose of amiodarone to prevent atrial fibrillation, and that is a very effective treatment. A small amount of amiodarone (100 to 200 mg daily) is a benign dose. There is nothing wrong with diving with a small amount of amiodarone. However, amiodarone sensitises the skin to the sun. Many medications have other effects that one needs to warn the patient about. Amiodarone is one that causes skin sensitisation. If somebody takes 800 mg of amiodarone to prevent sudden death syndrome, that is not acceptable, because such doses cause significant problems with the lungs. the eyes, the liver and the thyroid. Large doses of most other antiarrhythmic drugs cause side effects that can be complicated by diving. Small doses, for the most part are not necessary so when a patient is on a small dose of anti-arrhythmic one can be sure, first, that it is not doing very much and, secondly, that the small dose is not going to bother the individual for diving. What one must do is to gently nudge the patient's physician to find out why the patient is still using this medication. In general the antiarrhythmic drugs in small doses are benign, but the question one must ask is what are they being used for.

Anti-hypertensive drugs

Beta-blockers, ACE inhibitors and calcium channel blockers, with or without a diuretic, are the common anti-hypertensive treatments. They are all benign. Beta blockers will reduce exercise tolerance at its maximum. If one is reasonably well conditioned one is not going to need maximum effort so the beta blockers are not going to limit one's exercise capacity. Sometimes the calcium channel blocker will cause orthostasis. I would be reluctant to let anybody taking high doses of antihypertensives dive. But moderate doses which have minimal side effects, are not a problem with any of the medications.

Audience participation

Unidentified speaker

What cardiovascular screening would you do on mature age diving candidates (I judge mature age as anyone older than myself)?

Bove

The age of 40 is a good base line. Well conditioned individuals, who have no risk factors, that is normal blood pressure, not overweight, no diabetes, non-smoker and with a good exercise tolerance by history, between 40 and 50 would not need to be screened with an exercise test.

With risk factors I think one should screen with at least a routine stress test. That is put the patient on a bicycle ergometer or a treadmill, take a continuous electrocardiogram and check the blood pressure while they are exercising. If they do not show ischaemic changes with moderate amounts of exercise, that is exercise up to around 10 or 11 mets, then they are probably fit for diving. But that does not say that they are free from coronary disease. All it says is they do not have a critically obstructive coronary lesion that would put them at risk for any kind of exercise.

Above the age of 50 I would do much the same but I would be more likely to do a stress test even though I knew the patient was exercising adequately. Beyond about 55 I think everybody ought to have a stress test.

Unidentified speaker

What speed of running is associated with 13 mets.

Bove

About an 8 minute mile (4.9 minute km). However this a maximum capacity. A steady capacity would be 70% of 13 mets which is equivalent to a 10 minute mile (6.25 minute km).

Unidentified speaker

The greatest risk, of course, is not at entry into diving, when we get our diving medical done at 17 and we are fit. We should screen 40 year olds and upwards before they start diving. But what should we do for aging divers ?

Bove

A good history of physical activity, with low risk factors, eliminates the need for stress testing.

Unidentified speaker

Is there any evidence that people with mild valvular disease having a higher incidence of bubble formation through induced turbulence?

Bove

No there is not. I spent four summers as a medical student studying cavitation in stenotic valves. We were never able to show that it occurred.

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PATENT FORAMEN OVALE IN UNDERWATER MEDICINE

Paul Langton

Abstract

The foramen ovale, between the right and left atria, exists in the foetal heart as a vital physiological communication. Haemodynamic closure occurs in the neonatal period with most people having permanent fusion of the foramen. In up to a third of adults the closure is functional only and a potential right to left atrial communication persists as a patent foramen ovale. Studies in patients with decompression illness after diving suggest a consistent increase in the prevalence of patent foramen ovale, as detected by transthoracic contrast echocardiography. The association is strongest for those patients with early onset of neurological decompression illness, particularly those cases occurring in the absence of other risk factors traditionally associated with decompression illness. However, patent foramen ovale is a common finding in the general population and the absolute risk of decompression illness, even in the presence of a patent foramen ovale, remains very low.

Key Words

Decompression illness, heart conditions, investigations.

Introduction

There has been considerable interest in the potential contributory role of the foramen ovale in the development of decompression illness (DCI) and arterial gas embolism (AGE) in SCUBA divers. Venous bubble formation is known to occur during hyperbaric gas exposures well within the recommended limits of recreational diving.[1] The relative absence of clinical decompression sickness is thought to be related to the filtering of venous bubbles as they pass through the pulmonary circulation, thus preventing systemic arterial exposure. It is proposed that the presence of a patent foramen ovale (PFO) allows venous bubbles to pass across the interatrial septum into the left heart and then into the arterial circulation, with the potential to cause AGE.

Background

The foramen ovale exists as a vital physiological communication between the right and left atria during foetal life. Atrial division (Fig 1) initially occurs with the formation of the septum primum, a crescentic structure grows from the top of the common atria and fuses with the endocardial cushions that demarcate the atrioventricular junction. As it develops some of the central tissue of the septum primum beaks down to create the foramen secundum, maintaining interatrial communication. The septum secundum then grows from the right superior margin of the septum primum to incompletely divide the atria; it remains deficient inferiorly, against the endocardial cushions. The combined atrial septum (primum and secundum) thus forms the foramen ovale and allows oxygenated inferior vena caval blood (returning from the placenta) to be directed across the atrial septum to the left heart and thenceforth to the developing brain (Fig 2). In contrast, deoxygenated (superior vena caval) blood streams preferentially from the right atrium through the right ventricle to the pulmonary circulation and then via the ductus arteriosus back to the placenta. The foramen ovale remains open in the foetus because of the existence of significantly higher pressure in the right atrium as compared with the left.

The physiological changes that occur at birth include a profound lowering of pulmonary vascular resistance secondary to lung aeration, and a fall in right atrial pressure. At the same time systemic pressures increases, with a rise in left atrial pressure and hence the functional closure of the foramen ovale (Fig 3). In most infants this functional closure is followed by fusion of the flap like membrane, forming the fossa ovalis. In about a third of individuals fusion does not occur and a potential inter-atrial shunt persists as a PFO. For shunt flow to occur however the right atrial pressure must exceed that in the left atrium. The phasic nature and right to left flow of a PFO help distinguish this anatomical variant from an atrial septal

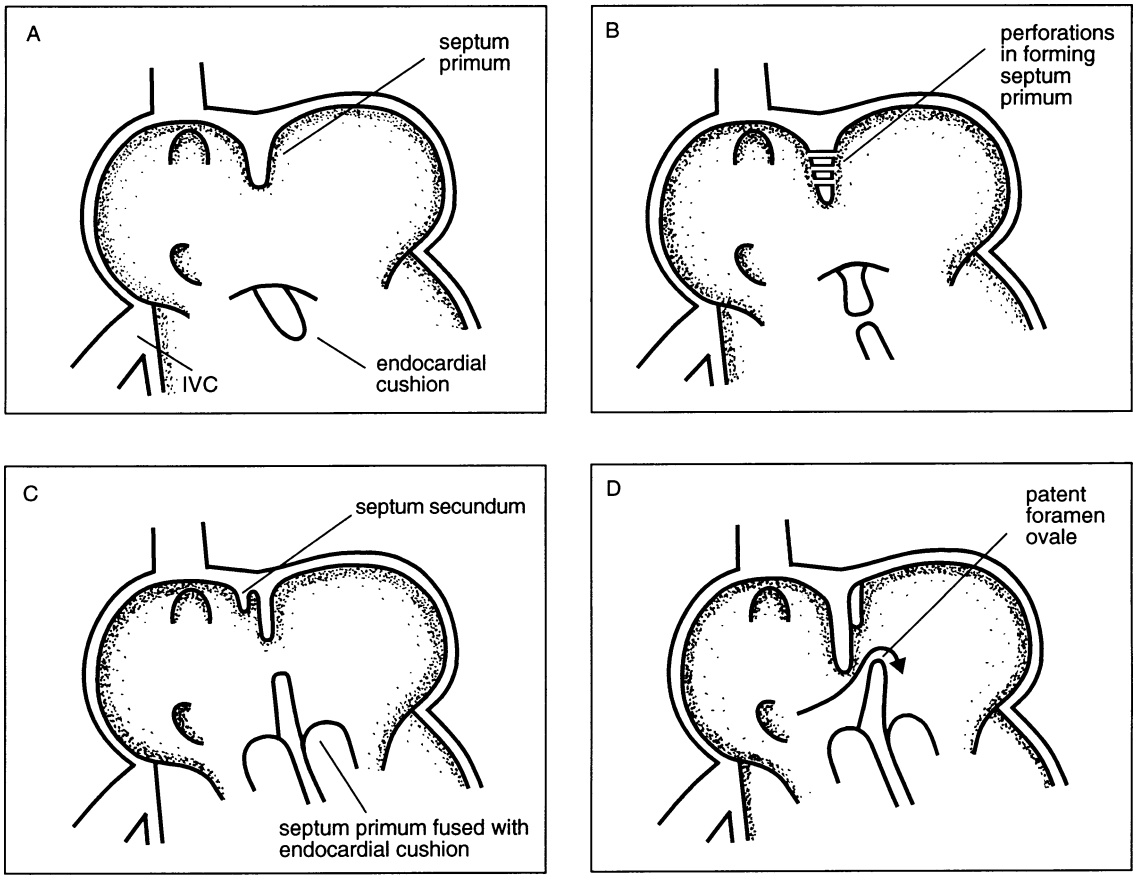

Figure 1. Diagrammatic representation of the formation of the interatrial septum in the primitive common atria. Diagram 1D demonstrates flow from the inferior vena cava across the foramen ovale.

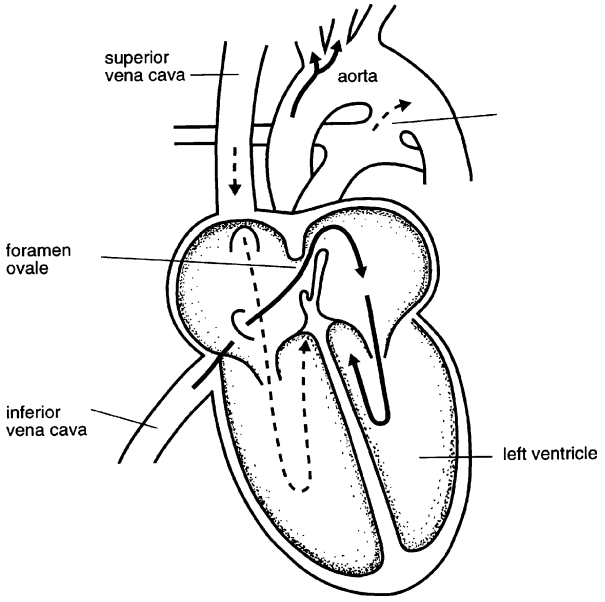

Figure 2. In the foetal circulation oxygenated blood from the placenta is directed via the inferior vena cava and foramen ovale to the developing brain (solid lines). Deoxygenated blood (broken lines) from the superior vena cava passes to the pulmonary artery and then crosses the ductus arteriosus to the aorta and is returned to the placenta.

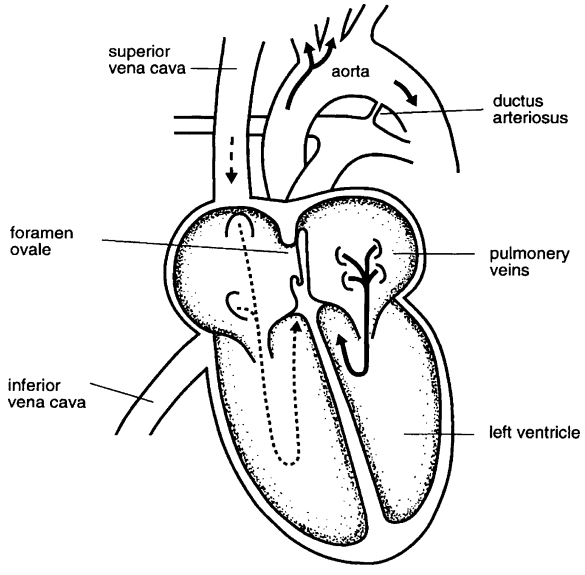

Figure 3. In the neonate the lowering of right atrial pressure and increase in left atrial pressure cause the functional closure of the foramen ovale.

defect, where there is a predominantly left to right (bidirectional) shunt across the septum and associated dilation of the right heart and left atrium.

Post mortem studies have attempted to define the frequency of PFO by detection of probe patency from right to left through the fossa ovalis. Hagen et al at the Mayo Clinic have studied 965 "normal" hearts in an autopsy series, using calibrated probes to define the maximum potential size of the PFO.[2] By this method most PFOs were small (mean 4.9 mm), however communications of up to 19 mm were found. The incidence of probe defined PFO was approximately one third in subjects below 30 years of age, falling to approximately one quarter for those above this age. There was a tendency for the average size of PFO to increase with age, suggesting that smaller PFOs may spontaneously close in early adult life.

Anatomical presence of PFO does not imply interatrial shunting. Although mean right atrial pressure is lower than left atrial pressure, there can be transient phasic inversions in this gradient in early atrial systole with the potential for right to left shunting in the absence of any unusual physiological circumstances. Situations that elevate right atrial relative to left atrial pressure would increase the tendency for any right to left shunting,[3] such as breath holding, coughing and the Valsalva manoeuvre (all common during scuba diving). Immersion itself may cause an elevation of right atrial pressure, but has not been shown to affect the interatrial pressure differential.

Echocardiography

PFO during life has most commonly been detected by transthoracic echocardiography, and more recently by transoesophageal (TOE) echocardiography, through either the detection of interatrial shunting with colour flow Doppler, or by the observance of ECHO contrast transit from the right to the left heart after injection of contrast agents such as agitated saline In most cases the degree of the shunt is very small and occurs as left atrial pressure rises above the right. Less commonly right to left flow is seen. The colour Doppler technique is limited by the need for good views of the interatrial septum which are most readily obtained from the subcostal approach, but adequate views are often difficult to obtain.

Agitated saline is most commonly used as the contrast agent for the detection of PFOs. It has many microbubbles suspended in solution, which form an effective contrast medium for ultrasonic detection.[4] In practice agitated saline is injected into a large peripheral vein and this bolus can be visualised within several cardiac cycles as it passes through the right heart. The microbubbles are then normally filtered out by the pulmonary microcirculation.[5] In the case of some subjects with a PFO, ECHO contrast material can be seen to pass spontaneously from the right to left heart, usually distinguishable at the atrial level. Detection of right to left shunting can be increased by the performance of provocative manoeuvres that elevate relative right atrial pressure, such as after a cough or in the release phase of the Valsalva manoeuvre, at the time of ECHO contrast injection.[6,7] Left to right shunting is occasionally seen as a negative contrast effect (which has to be distinguished from inferior vena cava (IVC) flow), although this would be more typical of an atrial septal defect. There is considerable variability in the ability of contrast injections to detect shunting from moment to moment and at least 2-3 boluses are usually injected (with up to 6 being reported), in routine practice.

Because of the technical considerations involved in the detection of PFO, routine transthoracic ECHO studies looking for PFO need to be performed by an experienced sonographer using a validated protocol of baseline and repeated contrast ECHO views before and after provocative manoeuvres. With such a protocol the incidence of PFO in a normal control population can be defined, with most studies identifying functional PFOs in about 15 - 31 % of people.[7-9]

Transoesophageal echocardiography detection of PFO has been widely used in both unexplained stroke in younger patients and in all patients with stroke. While there is clear evidence that TOE has greater sensitivity over transthoracic ECHO for the detection of PFO, this applies equally to control subjects and patients with stroke.[10] When comparing the prevalence of PFO in young stroke patients to that of true control subjects, studies show somewhat conflicting results as to whether PFO is more common in the patient group.[10,11] TOE seems able to detect a greater number of (possibly) smaller PFOs their relevance to disease states remains to be proven.

Importantly the detection of PFO by any contrast ECHO relies on the passage of microbubbles into the arterial circulation, in a similar way to the proposed mechanism for DCI (although in DCI the bubble size is likely to be greater). There is a published incidence of predominantly transient neurological side-effects after contrast ECHO (about 1 in 6,000).[12] It would seem prudent to defer investigation of a possible PFO in a patient with DCI until after the episode has completely resolved.

PFO and decompression sickness

Several case reports from the early 1980s identified the association of PFO with decompression illness (DCI) after diving.[13,14] Moon's group found that PFO was detectable on transthoracic ECHO in 11 of 30 patients with DCI (37%), and that the subgroup of patients with severe signs and symptoms (weakness, vertigo, cognitive impairment) had PFO in 11 of 18 (61%).[14] Interestingly, all these patients had a PFO evident during spontaneous

breathing. The authors did not study a control group of normal subjects, relying on reports from other studies which may not have been directly comparable. Cross et al. have subsequently attempted to define the incidence of PFO by transthoracic ECHO in a population of 'normal' control divers.[9] They examined 78 divers who had no history of DCI, by contrast ECHO before and after Valsalva manoeuvre. Twenty four divers were found to have PFO, with the incidence of 31 % being similar to the rate of PFO detected in a similar age group (their mean was 34 years) in Hagen's autopsy series.[2] This suggests that previous lower estimates of background rates[7,8] may have underestimated the prevalence of PFO in the diving population, possibly by the study of relatively older patients and/or methodological issues. That the overall incidence of PFOs in Moon's series[14] was similar to rates reported at autopsy and by Cross[9] suggests the primary role of PFO in overall DCI incidence is open to question.

In a second series reported by Wilmshurst et al.[15] 61 patients with DCI were divided into predetermined clinical subgroups. The control subjects were the diving "buddies" of the patients or experienced divers who had never had DCI and were of similar age to the patient group. The incidence of PFO on transthoracic ECHO in this control group was 24% (15/63). The overall incidence in divers with DCI was 41%, however in the subgroup of 29 patients with onset of neurological DCI within 30 minutes of surfacing 19 had PFO (66%, $p<0.001$ cf controls or other patients).

With respect to dive profile associations with DCI, Wilmshurst subdivided all patients into those with and without recognised risk factors for DCI.[15] The patients whose dive profiles would have otherwise been considered "safe" were more likely to have PFO (16/25) than in those who performed dives that would be accepted as having increased risk of DCI (9/36). This finding supports the hypothesis that PFO is probably causally related to these episodes of early onset neurological DCI occurring after otherwise safe dives. However the small numbers and subgroup analysis do limit the validity of this interpretation.

Cross et al. reported 19 cases of neurological DCI and found PFOs on transthoracic ECHO in only 6 of 19 patients (32%).[16] The clinical severity (ie sensory changes only or more severe neurological signs) and time between surfacing and symptom onset were not reported.

Limited prospective data regarding the association exists. In a study by Vik et al. anaesthetised pigs were exposed to air at 5 bar for thirty minutes and then rapidly recompressed. Arterial bubbles were detected by transoesophageal ECHO.[17] Presence of PFO was defined anatomically at subsequent autopsy. The pigs with PFO had a much higher rate of arterial bubble detection (6/6) than the non-PFO group (2/8). This supports the hypothesis that PFO increases the risk of arterial gas embolism and hence presumably the risk of clinical DCI.

PFO and other neurological syndromes.

The frequency of unexplained stroke in relatively young patients greatly exceeds that of divers with DCI and the occurrence of such strokes may be related to PFOs. In 1988 two studies reported an increased incidence of transthoracic contrast ECHO detected PFO in young patients (<55 years) with otherwise unexplained stroke (40% vs 10% and 50% vs 15% respectively).[8,18] It is thought that PFO allows the passage of venous derived thromboemboli into the arterial circulation and hence cause neurological events. This is similar to the proposed mechanism for some cases of early onset neurological DCI (passage of gaseous emboli).

In comparison to PFOs detected incidentally, patients with unexplained stroke generally have larger foramina, with a greater degree of (semi-quantitative) right to left contrast shunt, and are more likely to have an associated atrial septal aneurysm with their PFO.[19-23] Any relation of atrial septal aneurysm with DCI is however uncertain, and this may be a confounding factor when trying to compare data derived from stroke patients with those relating to DCI.

With respect to risk of recurrent neurological events, several studies have followed up young patients (<60 years) with a PFO and an otherwise unexplained stroke for two or more years.[23-25] The risk of further events is up to 2 % per annum if there are associated cerebrovascular risks (e.g. atrial fibrillation), or up to 4.5 % when both PFO and an atrial septal aneurysm exist. In the absence of these added factors, the risk of recurrence is very low (<1 %).

Absolute Risk

On the limited data outlined above, it would seem that the presence of PFO may confer an increase in the relative risk of some types of DCI. This relative risk needs to be interpreted in light of the overall incidence of DCI. It is estimated that there are over 50,000 divers in the United Kingdom, of whom 12-15,000 would have a PFO.[26] The reported incidence of DCI is around 100 cases per year, of which only approximately 50 represent the early onset of more severe neurological DCI that has been most closely linked to PFO. Looking at the relative incidence of PFO in patients with early severed DCI versus controls (66 % versus 24%),[15] PFO is associated with an excess of about 42% or about 21 cases a year. This represents an increased risk in the order of 1 in 600 for subjects with a PFO. Although the confidence intervals for these estimates would be large, the estimates do provide us with a starting point to put the absolute risk associated with PFO in perspective.

Management

Any management of PFO is dependant on the circumstances in which it is detected.

There is no general agreement to support screening for a PFO prior to diver training. If a PFO had been picked up incidentally during an ECHO, the reason why the subject had been having the ECHO in the first place may be more important in assessing future diving risk. It would be reasonable however to explain the potential risk of DCI (in absolute terms) to such a patient. If a subject had an incidental ECHO that did not identify a PFO, unless the technician had been using a protocol to formally look for PFO, one could not assume that PFO was absent.

In a patient who has had early onset of neurological DCI, particularly in the absence of other well recognised risks for DCI, it is reasonable to look for a PFO with a transthoracic contrast ECHO study. It would be prudent to defer this investigation until the episode had completely resolved. A PFO detected may or may not be relevant compared to other risks for DCI. Those with a greater degree of (semi-quantitative) right to left shunting are possibly more important than very small shunts. All patients who have suffered DCI need careful advice about future diving. If a PFO has been demonstrated a detailed discussion of the problem with the patient, including their likely future risk, probably more important than a proscriptive approach banning further diving. Regardless of the presence of a PFO, the diver has to be exposed to a bubble forming dive profile before being at any risk and it may well be possible for them to dive more conservative profiles without forming bubbles. The vast majority of subjects with PFO do not suffer from DCI, despite the fact that many of them will form venous bubbles during recreational diving exposures.[1]

Open heart surgery to close a PFO alone would not be advocated. Cardiac bypass itself exposes the patient to gaseous microemboli, and leaves some scarring in the chest. Transvenous devices can be used to occlude an atrial septal defect, including a PFO.[27] However some trans-septal flow often persists and currently these devices US Food and Drug Administration approval. Their use would be considered experimental.

Conclusions

Patent foramen ovale is a common finding in the normal population. On limited data it appears to confer an increased risk for early onset neurological decompression sickness, particularly if there is a large shunt evident. Although PFO may, under conditions that predispose to DCI, make it more likely to be apparent, overwhelming consideration remains the predisposing diving conditions. Detection of suspected PFO requires a protocol of contrast transthoracic ECHO before and after provocative manoeuvres; the possible role of transoesophageal ECHO is undefined. The absolute risk of DCI in a subject with a PFO remains very low. The risk of recurrent DCI in those patients with PFO who continue to dive is uncertain, but is likely to be minimised by adherence to conservative dive protocols.

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SPUMS ANNUAL SCIENTIFIC MEETING 1996

THE SCOPE OF NON-CONVENTIONAL RECREATIONAL DIVING

R W Bill Hamilton

Key Words
Deep diving, mixed gases, oxygen, recreational diving, safety, tables.

What is "recreational diving"?

Non-conventional recreational diving has to begin with a basic definition of recreational diving, which can be summarised as diving for fun. Recreational diving is well recognised as being scuba diving in the range to 40 msw (msw = metres of sea water; 1 msw ≈ 0.1 bar or 10 kPa), and further it is diving with air as the breathing gas and not involving decompression stops. Realistically, these are not the limits within which all recreational divers operate, but until recently they were the limits to which divers have been trained by the recreational diving training agencies, particularly in the U.S.A. The British Sub-Aqua Club (BSAC) trains divers to 50 msw and allows decompression stops. This zone defined above is also recognised by the U.S. Occupational Safety and Health Administration as being outside commercial diving. Thus instructors can teach diving within these limits without their employers having to comply with the Commercial Diving Standard. In Britain recreational diving instructors at work who breathe non-air mixtures are considered to be commercial divers.

As mentioned and as the name implies, recreational divers are doing it for fun. Implicit in this is that these divers are not employees and they are not at work. Other types of sport diving are "recreational" in not being work, but may involve considerable specialisation and skill. Some of these are cave diving, ice and other types of overhead-

environment diving, decompression diving with scuba, hookah diving, enriched air diving, rebreather diving, "technical diving," and combinations of these. Another "outside the limits" type of diving is deep air diving; this deserves special attention because it was to avoid deep air diving that technical diving was developed. Another category often considered to be "technical" and thus outside recreational limits is diving with oxygen-enriched air ("nitrox"). This has recently been rescued by PADI and BSAC and is now regarded by these agencies and others as being legitimate recreational diving.

One recurring question is, "What about instructors?" When they breathe exotic gas mixtures and perform decompression, are they within the limits of recreational diving? The answer to this one is still out in the U.S.; some discussion on this was done at the 1996 SPUMS meeting and will be reported in the Journal.

Deep recreational dives

Deep is a relative term which involves the diver's own skill and preparedness as much it does the water depth. Even within the mentioned limits special "deep" training is needed to go even as deep as 40 msw.

For some years now some scuba divers have exceeded the 40 msw limit in air scuba dives, using decompression stops when necessary, and under some conditions have even used oxygen for decompression. One clever method was to use the otherwise unreliable USN Exceptional Exposure air decompression tables with oxygen decompression. With oxygen in the last couple of stops the decompressions were not much of a problem, and the tables were "legal" and "in the book." However, these tables allowed divers to go well beyond the depth at which nitrogen narcosis can become seriously debilitating. As depth increases much beyond 60 msw the PO_2 (partial pressure of oxygen) in air also becomes a risk factor due to CNS (central nervous system) toxicity. It was clear to those who thought much about it that there was a need for something better than diving deep with air.

Relevant background experience

Mixed gas surface-supplied diving by navies and particularly commercial diving companies had been developed to a high level of sophistication but within rather narrow, experience-based limits. Air was not used beyond the depths where it is well tolerated, but for the tethered diver with a topside supervisor, narcosis was much less a threat that for the free-swimming scuba diver. The benefits of helium to prevent narcosis have been well established in this community, but in general the procedures were not easily adapted to scuba operations.

Although not generally dedicated to extending diving depth, another substantial experience base had developed in the application of technology to diving; this was the historical development of rebreather diving by navies, especially the clearance divers of the British Royal Navy during and after WW II. While properly thought of as "technical diving," this experience was not really tapped for the development of the initial wave of technical diving as we know it now.

However, rebreathers were involved in the early development of technical diving. In the middle 1980s Stuart Clough of Carmellan Research began using Rexnord CCR-155 rebreathers (now Carleton), focussing on photogrammetry, exploration, and treasure hunting. This was supported by lab trials at Dr. Maurice Cross' Diving Diseases Research Centre (DDRC) at Plymouth in the UK and included dives to 150 msw and also work with neon.[1] This group found that using a PO_2 of 1.4 bar(140 kPa) gave an efficient decompression. The idea had been mentioned by Vann some years earlier,[2] but the US Navy used only 0.7 bar PO_2 in the military versions of these rebreathers, for reasons other than decompression. This level, 1.4 bar, is just below the level of central nervous system toxicity and because of the high oxygen it gives a nearly optimal exposure to inert gas at bottom pressure (that is, it makes the inert component as low as it can safely get). An expedition in 1987 by Rob Palmer to study blue holes on Andros Island in the Bahamas involved Stuart Clough, Bill Stone, Rob Parker and myself, among others; successful dives to nearly 90 msw were done with the rebreathers.[3]

As a direct follow-on to the Andros operation, a group of cave divers making deep penetrations began to add some helium to their bottom mixtures. Cave exploration in North Florida by Parker Turner, Bill Gavin, and others called for long times (over 1 hr) at depths in the 70-80 m range; these divers (quite correctly) regarded such dives as being too dangerous with air, primarily because of the narcosis. The use of special mixes also allowed the oxygen fraction to be reduced, allowing a lower PO_2 to be used at bottom depth and thus making longer bottom times feasible without incurring oxygen toxicity.[4] The use of special mixtures made special decompression tables necessary, and these were developed.[5] Cave divers had already developed highly specialised techniques and equipment, a high level of discipline and a special category of training, so were a good place for this to start. This technology quickly spread to deep wreck divers, who learned to do this same pattern with diver-carried gas.[6,7] This is the technology that became known as "technical diving." It is correct to say that technical diving was invented to avoid having to dive deep with air.

The pattern from the beginning was to use an oxygen-helium-nitrogen trimix with both oxygen and nitrogen appropriate to the depth, then switch to an intermediate enriched air mixture until oxygen could be

breathed (at first at 9 msw, but this was reduced to no deeper than 6 msw as experience built).

Boyle's Law expansion

Like a rising bubble, the concept began to grow. Jim King, who could afford it, began serious deep diving operations, working with Billy Deans of Key West, who became, and still is, the leading technical diving guru. Others learned to do the decompression calculations, based on the published algorithm of Prof. Bühlmann.[8] In due course commercially available do-it-yourself decompression software became available.

Early interest was in cave exploration, but soon open-sea wreck divers began to develop techniques. Early development focussed mainly on gas logistics. At first tanks were over-pressurised, later bigger tanks became available. Cave divers stage their extra gas, but open sea divers have to be self-sufficient, even to the extent of completing their decompression while drifting, possibly having no contact with the boat. Drs Zannini and Magno[8] had earlier helped Italian coral divers develop similar techniques, but this was not known to the American cave divers. There had been other deep open-circuit scuba dives by specialists, not all successful (some of these were real explorers), and the impression was that this was risky (rightly so!), highly specialised, proprietary, and the techniques were not available to others. Some significant explorations have used technical diving techniques.

aquaCorps takes it "out of the closet"

The final step in making technical diving a reality was spreading the information to other divers. This began with the publication of a journal dedicated to it. Michael Menduno was so intrigued by this new development in recreational diving that in 1990 he founded aquaCorps, a "journal" (really a magazine) dedicated to this concept. It not only disseminated technology, but it got people talking about it. Menduno coined the term "technical diving." He also had the idea of running a conference dedicated to technical diving, and "tek" conferences were held just before the DEMA show from 1992 to 1996. aquaCorps ceased publication after a total of 12 issues, closing down after the 1996 tek show and is now out of business. Several other magazines now address technical diving issues.

As the interest in this extended-range diving increased, manufacturers began to provide big tanks, special components of the technical rigs, gas mixing facilities, scooters, etc. Decompression software became available, and enriched air computers. Training courses have proliferated, most of them spun off from enriched air training; several are offered by companies that pretend to be "associations" of technical divers. On the down side, a dozen or so fatalities in 1992 showed the hazards of not doing it right.

Enriched air for the masses

In a somewhat parallel development, the use of oxygen-enriched air (OEA) in recreational diving has reached a high level of refinement. This is the practice of improving the decompression situation by replacing some of the nitrogen in air with oxygen. It was started in the 1970s within the diving program of the US National Oceanic and Atmospheric Administration (NOAA) by Dr Morgan Wells and was picked up by his colleague Dick Rutkowski who began promoting and popularising it (they called it "nitrox") among recreational divers. Promotion of OEA created considerable controversy, largely because of the NIH (not invented here) factor, gross and somewhat unsophisticated over promotion (mainly by Rutkowski), and a general lack of understanding of the practice. Australia was not spared this controversy. Those uncomfortable with the practice stretched their imaginations in finding reasons not to do it as much as its supporters did to promote it. NASA spent these early years finding reasons not to use OEA in their neutral buoyancy training tanks, but when the Hubble telescope repair called for a long mission, and training for it could be done most efficiently with enriched air, NASA jumped right in. The final stroke of acceptance has been with PADI's entry into this practice (see paper by Drew Richardson).

Those inside the technical diving community regard diving with enriched air as not being technical diving (with the exception of technical training organisations, who keep this issue totally muddled with the multiplicity of courses offered). Those outside see OEA as exotic and highly technical, so find it easy to lump it with technical diving.

Although not really "technical" diving, use of enriched air called attention to alternative breathing mixtures. Interestingly, technical diving practice itself did not cause such controversy, perhaps because it did not threaten to invade the recreational diving domain. Some "nitrox" related myths were that it would corrode your tanks and buoyancy compensator (inconsequential), that you could not treat a diver with DCS from "nitrox" diving (standard treatments work the same way they do for air dives), that gases have to be used within two weeks (if they change it is because they were not properly analysed to begin with), that you should use two analysers (if you need think you need two of them, then chances are you do not know how to use either one of them correctly). Other myths from the promoters were that there were some half dozen benefits (there is only one, it improves decompression); that it reduced narcosis (it does not, since oxygen is as narcotic as nitrogen); and that one could benefit from using OEA at 50 msw (this is not at all worth the effort because of oxygen toxicity limitations).

What is "Technical diving"?

Given all this, perhaps it is now possible to define technical diving. At the outset it should be pointed out that the term "technical diving" comprises so many different aspects of diving practice that SPUMS policy does not address this as a single entity.

Technical diving

is recreational;
it has been developed entirely by recreational divers who do it voluntarily, at their own expense and risk;
it does not meet occupational safety standards;
the term is an analogy with technical mountain climbing;
is self-contained recreational diving which may extend beyond the range of traditional recreational diving;
necessarily involves special training, discipline, experience, and commitment beyond ordinary diving;
uses special techniques and equipment, including breathing mixtures, gas management, decompression procedures, decompression stations, thermal protection, buoyancy and ascent control, propulsion, and redundancy;
requires detailed operational preparation and planning.

A technical dive involves a change in breathing mix or use of a rebreather.

The definition excludes some things. Technical diving is not;
diving with oxygen-enriched air ("nitrox");
using rebreathers in the recreational envelope (40 msw, no-stop);
and of course deep air diving.

Operational organization is imperative for all but the mildest technical dive; some good examples of how to do this are now available.[10]

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NITROX

David Elliott

Key Words
Accidents, deaths, injuries, mixed gas, nitrogen, nitrox, oxygen, safety.

"Nitrox" is an easy word to use for the range of oxy-nitrogen mixtures but there are several other terms also in use. For those mixtures in which the oxygen content is greater than 21%, "Enriched Air Nitrox" (EANx) is a common term and "Oxygen-enriched air" (OEA) is another, while others (such as the one which suggests that nitrox is a "safe" version of air) are proprietary. Nitrox has also been a term used in saturation diving procedures by NOAA for mixtures in which the oxygen content is *less than* 21% and it has been suggested that the term nitrox for oxygen-rich mixtures could be ambiguous and that nitrox should be reserved for oxygen-lean mixtures.

But there is a precedent: "heliox" is also an easy word to use. The heliox mixtures used in deep diving are

oxygen-lean but also 50/50 heliox, for example, is used for therapy in the Comex 30 metre table. Yet it has never been suggested that the term heliox should not be used for the oxygen-rich mixtures. So, in this paper, nitrox will refer to *any* oxy-nitrogen mixture other than air and, as this is in the context of recreational diving, nitrox will usually refer to an oxy-nitrogen mixture with more than 21% oxygen.

In fact, adoption of the broader term "nitrox" is particularly appropriate for recreational diving because, as will be shown later, there are some circumstances when, although EANx or OEA has been supplied to the breathing bag of a rebreather, the "oxygen-enriched air" which is then breathed by the diver will contain *less than* 21% oxygen.

Oxygen-enriched air has been used in military semi-closed rebreathers for more than 50 years but that is a special application which will be reviewed later. It is probable that this principle was used first in mine clearance because of the safety advantage of less noise from exhaled bubbles with a rebreather than from an open-circuit underwater breathing apparatus.

The thought occurred to several commercial diving companies some 30 or more years ago that diving with oxygen-enriched air in open-circuit breathing apparatus could shorten obligated decompression times usefully. It had positive financial benefits, particularly in tidal waters and so, in consequence of commercial secrecy, it is not easy to discover where the idea originated or how practical and safe it turned out to be in those early days. Around 1963 Micoperi, an Italian company which, with Shell, owned a North Sea diving company, used high oxygen levels for some nitrogen dives. Andre Galerne's diving company, I.U.C., was doing much the same at around the same time, also as a discrete proprietary technique.[1]

Equivalent air depth

The whole essence of nitrox diving is that one can ignore the oxygen content of a breathing gas for the purposes of the decompression calculation. So, when breathing an oxygen-enriched air mixture, the nitrogen uptake at depth is reduced and the uptake can be considered the same as that when breathing air at some shallower depth, the "equivalent air depth" (EAD).

The EAD can be calculated for any percentage oxygen level:-

Metric formula

$$\text{EAD [in m]} = \frac{(\text{Actual Depth} + 10) \times N_2\%}{79\%} - 10$$

or

Imperial formula

$$\text{EAD [in feet]} = \frac{(\text{Actual Depth} + 33) \times N_2\%}{79\%} - 33$$

Strictly speaking this principle is not absolutely correct because, for example, increased oxygen tensions can contribute to decompression sickness either by the effect of altering blood perfusion rates to critical tissues or, exceptionally, by contributing directly to the illness.[2] These and other theoretical concerns do not detract from the fact that the principle of EAD has served many divers well for more than 50 years.

The advantages of EAD

The practical advantage of increasing no-stop bottom times at depth is obvious for those recreational divers who base their diving on the no-stop tables. For those planning decompression stops one advantage is that of briefer stops for any given bottom time and depth.

However, the advantage for no-stop diving is limited. At shallow depths where dive duration is limited by the volume of gas that the diver can carry, there may be no advantage of using nitrox, particularly for a single dive. At around 22 m an average diver will carry just sufficient gas to complete a dive with a duration which is around the compressed air no-stop time and so the diver could not stay there longer even if he or she were on nitrox. The benefit may come with the second dive of the day.

PADI quote no-stop times at 18 m of 56 min on air, 95 min on 32% oxy-nitrogen and 125 min on 36% oxy-nitrogen and the BSAC decompression tables have very similar durations. One other training agency quotes no-stop times at 80 feet (26 m) of 30 min on air, 45 min on 32% oxy-nitrogen and 55 min on 36% oxy-nitrogen. The USN tables would be less conservative at 40, 50 and 60 min respectively. This difference is not related to the nitrox theory or oxygen exposure but is a reflection of different underlying decompression models. These examples all illustrate the small increments of increased bottom time available and thus the need to weigh the potential advantages carefully against the costs of training, equipment and gas and the risks from other hazards.

At the deeper depths the use of oxygen-enriched air is constrained by the need to avoid oxygen neurotoxicity. PADI, BSAC and several of the technical diving agencies suggest that exposure to a PO_2 of 1.6 bar should be for only special contingencies, not regular recreation. One of the technical diving training agencies compares the no-stop times of air, 32% and 36% oxygen over a depth range to 40 m. These tables demonstrate the even smaller advantages to be gained in the depths which take the diver beyond the oxygen limit of PO_2 at 1.44 bar.

Another advantage of nitrox quoted by some agencies is that oxygen-enriched air has a potential for increased decompression safety. This is true but it is achieved as an *alternative* to using the principle of longer

bottom times and shorter decompressions. By ignoring the equivalent air depth and using the air decompression tables for the actual depth dived on enriched air, the risks of decompression sickness are reduced. For a large population of divers it would be very difficult to see any improvement when using nitrox over the already very low incidence of decompression sickness when using air.[3] However spinal decompression sickness appears to be more common in, for example, those over 40 years old. At present additional safety can be achieved by using an even more conservative no-stop air table, for instance by using one's decompression computer in the altitude mode when diving at sea-level . But in effect this is a decision to surface early on a regular basis and one which can make it difficult to find buddies! In these circumstances the use of nitrox on a regular air table (or with a regular air-based computer) would provide safety and preserve friendships. I am told by its owner that there is one dive shop in the Caribbean which provides 32% nitrox specifically for increased safety, so it seems that some recreational divers are prepared to pay for this particular advantage.

While in theory a diver could compromise between the two separate advantages, a prolonged bottom time or a safer decompression, one must choose because one cannot have it both ways, as has been suggested by several technical diving training agencies.

Another claim which has been made for the safety of nitrox appears to be unsupported by any evidence. "If a diver has been breathing nitrox during the course of the dive then the chances are that their injuries will be comparatively less serious than if they had been diving on air".[4] Such misconceptions are not uncommon and I am assured that this potentially misleading error will be corrected in the next edition.

Maximum depths with nitrox

Oxygen toxicity theory and the accumulated experience underlying the depth limits imposed on oxygen-enriched gas mixtures have been reviewed at this meeting already.[5] Accordingly only a few special points need to be illustrated and they will be reinforced by using commercial experience of open-circuit nitrox. Professional divers dive in a safer manner than sport divers because they are required to use procedures and equipment that would not be tolerated in recreational diving. By diving with a hose they have an underwater duration not limited by a scuba tank. Hard wire communication is routine and provides an additional safety feature. Pre-dive gas analysis of premix and back-up mixes must be meticulous and, with hose-diving, an on-line gas analyser is another safety factor. Depth is continuously monitored at the surface by an on-line transducer which means that they are unlikely to be allowed to exceed the maximum depth for the particular mix in use. Should a fit occur they will not drown because they are diving with a full face mask or helmet.

Even with such constraints, PO_2 1.5 bar is the maximum permitted in the North Sea. In spite of that, incidents do occur and one oxygen fit has been reported at PO_2 1.52 bar and another at 1.32 bar PO_2. In reviewing a proposal that the PO_2 1.5 bar limit could also be used by other categories of working divers, one should also consider the difference in equipment and procedures. A diving scientist is trained on scuba and, like a recreational diver, would use a half mask and an independent mouthpiece. Using nitrox in these circumstances an underwater fit, though rare, could be lethal.

There is no safe/unsafe cut-off value for oxygen. One is dealing with the probabilities of a seemingly random event. One recreational agency puts PO_2 1.4 bar forward as the maximum advised; BSAC and PADI use PO_2 1.44 bar. Only for exceptional tasks, such as diver rescue, would they go as far as 1.6 bar. One Technical Diving agency in its text implies using 32% oxygen at a maximum of 46 m (PO_2 1.8 bar). This may have been an error and they may have meant when using their other standard mix, "28%". Even this would provide a PO_2 of 1.57 bar and, the greater the PO_2, the greater the hazard. These are examples of the potential for confusion among those who come to nitrox from air diving. And, at depths as deep as 46 m (150 ft), for what advantage? The DCIEM tables provide no-stop time at 46 m on air of 6 min and 7 min at the 28% oxygen EAD of 41 m. On each dive the hazards and costs of using nitrox need to be assessed against the benefits.

Convulsions

Oxygen neurotoxicity has just been well reviewed at this meeting[5] but, in regard to the nitrox training provided by some agencies, perhaps too much emphasis is being placed on a diver being able to recognise the subtle prodromal warnings, if present. Just how useful underwater acronyms, like "VENTID" and "VITBEND", might be in enabling a diver to remember the list of symptoms, then decide to abort the dive and do so safely, is uncertain. The probability is that if one recognises a prodromal symptom, it is already too late.

A commercial nitrox diver on PO_2 1.5 bar heard a sound in his head like an "outboard engine" but he knew that there was no boat there. He felt that he was "going" and had the sense to try and wedge himself into the structure that he was on so that, if he lost consciousness, he would not sink to the bottom. He had a full oxygen fit, confirmed by a his own video camera, and he did sink to the bottom. His depth was being monitored at the surface so and he was quickly rescued, still wearing his full face mask.

But often, there is NO warning.

If one witnesses a fit, what action should one take? Most important underwater is trying to ensure the retention of the mouthpiece, if it is still in. One international agency recommends emergency surfacing but, if the diver is in laryngeal spasm then, theoretically at least, that manoeuvre might cause pulmonary barotrauma. Another major training agency avoids this hazard and recommends waiting until the diver becomes limp before surfacing. "Near drowning is easier to treat."

If the mouthpiece is retained then there is no panic and no need to rush. The diver can be returned to the surface when the fit is over. If the mouthpiece has come out, the rescuer must choose between the possibility of near-drowning of the diver fitting at depth and that of gas embolism of the diver fitting on ascent. This decision must be made in the absence of medical advice. Quite a responsibility.

40/60 or is it 60/40 ?

Another convention which needs to be re-established is that in describing oxy-nitrogen, as introduced by and used by NATO, the oxygen is always first. In a 60/40 oxy-nitrogen mixture one would expect to find 60% oxygen. It is therefore worrying to find a reversal of this by some of the newer training agencies, for instance "PO_2 2.4 bar in 60/40 treatment gas at 6 bar". An alternative, which is always to specify only the oxygen percentage, would seem acceptable for describing mixtures. Of course, whatever the convention may be for written texts or on labels, gas analysis is the ultimate and only acceptable indicator of oxygen content before actual use.

Repetitive diving

The calculations needed to predict the safe duration of a repetitive dive are fairly straightforward even if the shallower second dive is on a richer oxygen mixture. But some divers do get their repetitive calculations wrong even on air and, using nitrox, there are simply more opportunities for error. This alone justifies the need for proper training and occasional retraining of those who decide to dive with nitrox.

Gas mixing

The problems imposed by handling and of mixing oxygen are described elsewhere. "Top-ups" and other home brews can be lethal and the diver must always get the tanks filled by a reputable nitrox agency and *personally* witness the analysis the contents of his or her own tanks and label them accordingly.

Conclusion

The benefits from using equivalent air depths with open-circuit nitrox, as taught by the major recreational training agencies such as PADI, can be achieved safely provided that the diver is meticulous in following the appropriate procedures. Its advantages are however limited. There is only a narrow depth range in which nitrox can be used advantageously and the increased bottom times, which are only a little longer, come at an increased cost. At the shallower depths one cannot always take advantage of the increased bottom times on one tankful and near the maximum depth limits for each mixture there is the possibility, albeit remote, of an underwater convulsion. One wonders if the benefits are worth all the extra training, effort and cost. For some they are, but this must be a personal decision.

One must also be a little concerned about the marketing bias of a few of the agencies. For example, there is no proven advantageous reduction of nitrogen narcosis in the depth ranges used as is claimed by one. In one or two agencies there is a tendency to highlight the deeper maximum depths attainable but attainable only when using a PO_2 of 1.6 bar. In others there is a tendency to play down the hazards of nitrox in relation to compressed air diving. "Nitrox" says one agency "has just the same problems as diving with compressed air.". They both "have a potential for decompression sickness, the potential for trauma from handling pressurised gas and the hazards of oxygen toxicity." This is a misleading statement which is true only in absolute terms, but one which is not quantitatively true:

- when using EAD to determine decompression, the potential for decompression sickness with nitrox will approximate to that with air at that EAD.
- the potential for trauma when handling pressurised air does not include the hazard of handling oxygen and explosive mixtures.
- the hazards of oxygen neurotoxicity do not exist with compressed air diving (unless diving deeper than the recommended limits of the major training agencies).

Compressed air diving, as taught well by the wiser agencies, still has many practical advantages.

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THE PADI ENRICHED AIR DIVER COURSE AND DSAT OXYGEN EXPOSURE LIMITS

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Key Words

Mixed gas, nitrogen, oxygen, recreational diving, safety, training.

Introduction

In January 1996, PADI International released an Enriched Air Nitrox dive training program which is fully supported with educational materials for the student and instructor. This paper will review some of the philosophy, highlights, content and treatment of this topic found in the course. The purpose of the course is to familiarise divers with the procedures, safety protocols, hazards, risks, benefits and theory of no-decompression diving with oxygen enriched air containing 22% to 40% oxygen. The emphasis is on diving with EANx 32 and EANx 36 (also known as NOAA Nitrox I and II). Training emphasises the importance of proper procedures to ensure safety, and realistically balance the pros and cons of enriched air diving. Instructors are encouraged to elaborate beyond the material in the course outline to accommodate individual student interests and aspects of enriched air diving unique to the local environment.

The goals of this program are:

1 To enable a diver to plan and make no-decompression dives using enriched air blends containing 22 to 40% oxygen, remaining within accepted dive table and oxygen exposure limits.

2 To enable a diver to obtain, and care for, equipment used in enriched air diving.

3 To enable a diver to avoid the possible operational hazards and underwater hazards associated with oxygen.

There are two enriched air training dives required for certification. These may not exceed 30 metres (100 ft), or exceed a PO_2 of 1.4 bar, whichever is less.

An overview of enriched air

Enriched air is any nitrogen/oxygen gas mixture with more than 21% oxygen. Enriched air is sometimes called nitrox. However, the term nitrox includes nitrogen/oxygen mixes with less than 21% oxygen, which are used by commercial divers to reduce oxygen exposure when remaining under pressure for days at a time (saturation diving). These types of nitrox are made by mixing pure nitrogen and pure oxygen, rather than by adding oxygen to air. For clarity, the terms “enriched air” or “enriched air nitrox” are preferred by PADI.

Most of the special training one needs to dive safely with and handle enriched air relates to its higher oxygen content. The primary application of enriched air is to extend no-decompression limits beyond those of normal air. Based on US National Oceanic and Atmospheric Administration (NOAA) tests, Navy tests dating back more than 50 years, 20 years field experience by scientific divers and field experience in thousands of recreational dives, the no-decompression limits for enriched air are generally considered as reliable as those for normal air tables and computers. However, there is a trade off. As one reduces nitrogen exposure, one increases oxygen exposure. Therefore, much of what needs to be taught to students deals with keeping oxygen exposure within safe limits. Practically speaking, depending upon the dive depth and breathing rate, dives may be limited by enriched air supply rather than no-decompression limits. Therefore, in some cases, planned dive profiles and planned repetitive dives may not be able to take advantage of the additional time enriched air offers.

Decompression limits for EANx are calculated using the equivalent air depth (EAD) which is the shallower depth at which an air breathing diver would be exposed to the same partial pressure of nitrogen. The diver using EANx uses the time available at the EAD for calculating the time available at the deeper depth.

Because one absorbs less nitrogen using enriched air, one might expect that using enriched air within normal air no-decompression limits would substantially improve safety. This is probably *not* true. The decompression illness (DCI) incidence rate is already so low that it is unlikely that

simply reducing nitrogen can produce a *meaningful* risk reduction. Although there has been no study of this, statistical estimates suggest that using enriched air within normal air limits only reduces mathematical risk a fraction of a percent. The DCI incident rate is estimated as 0.004% (one in 25,000 dives) to 0.001% (one in 100,000 dives); if one cuts that by half (which is very unlikely), the best one could do is reduce incidence by 0.002 percent or 1 case in 50,000 dives. Therefore, it is inaccurate to suggest that enriched air is "safer" than air in any meaningful way. Used properly, both are safe and have impressive safety records. Used improperly, enriched air has more potential risk due to oxygen toxicity. Safety stops, avoiding factors that predispose one to DCI (such as dehydration), avoiding sawtooth profiles and other safe diving practices probably reduce one's risk far more significantly than using enriched air within normal air limits. Admittedly, some divers feel any mathematical DCI incidence risk reduction, even though tiny, still makes it worth using enriched air for dives than can be expected to be made safely with normal air. This is a personal choice without any safety concerns, provided enriched air procedures are followed.

Although enriched air reduces nitrogen, many diving physiologists do not believe enriched air significantly reduces narcosis when making deeper dives.[1,2] This is because oxygen under pressure appears to have similar narcotic properties to nitrogen under pressure. Thus, while enriched air has less nitrogen, it has about the same potential for narcosis. Although some divers say they experience less narcosis with enriched air, it is wisest to assume enriched air will not reduce narcosis. Some divers claim they feel better after a dive with enriched air. There is little objective evidence for feeling less tired or better after diving with enriched air, but it has been cited frequently. This may simply be a psychological effect.

Compared with air diving, diving with enriched air offers longer no-decompression times, but it also has five disadvantages and potential hazards.

1 Potential for oxygen toxicity
Much of what is taught is the PADI EANx program deals with staying within oxygen time and depth limits. Exceeding safe oxygen limits can be extremely dangerous. This is the most serious of the potential hazards unique to enriched air diving.

2 Special equipment
Because of the higher oxygen content, enriched air diving requires a dedicated cylinder and may require other equipment exclusively for enriched air diving. It can also be very hazardous to fill an enriched air cylinder from a conventional air source. Special equipment also includes a properly calibrated oxygen analyser to verify cylinder content; enriched air equipment also requires special maintenance.

3 Availability
Enriched air is readily available in some areas, in others one will not find it at all.

4 Proper gas blending and handling
One must ensure that one is diving with the blend of enriched air one intends to use and that no one confuses one enriched air cylinder for another or for normal air. It can be very hazardous for someone to use enriched air accidentally, or the wrong blend accidentally.

5 More complex dive planning
Enriched air diving requires more planning steps, with more potential for error and less tolerance for error if one makes one. One must use care and double check the dive table and oxygen calculations to avoid both DCI and oxygen toxicity. Monitoring one's depth becomes critical.

Equipment for enriched air diving

The primary concern regarding enriched air and dive equipment is the high oxygen content. Pure oxygen and high oxygen mixes cause materials to burn or explode more readily, even at normal temperatures. High oxygen content may also cause equipment to deteriorate rapidly.

A common guideline in diving is that standard scuba regulators, buoyancy compensation devices (BCDs), submersible pressure gauges (SPGs) and alternative air sources may be used for enriched air blends up to 40%. This is based on recommendations, standards and field experience by NOAA, the US Navy and the US National Institute of Safety and Health. In practice, this guideline has a good record. However, local law may require that all equipment used with enriched air be cleaned to oxygen service specifications, and local practice may also require that specific equipment meet oxygen service standards and include specific marketing or tags. Regulators rated for 300 bar (4,500 psi) meet oxygen service standards. Some groups within the dive community advocate oxygen service standards for all equipment used with more than 23% oxygen.

Most scuba equipment manufacturers have recommendations and/or modifications for their equipment when it is used with enriched air. Others state that their equipment should not be used for enriched air. Follow the recommendations of all manufacturers' guidelines, contact the manufacturer for information as necessary, as recommendations may change over time. In all cases, gas mixes with more than 40% oxygen (used outside recreational diving and beyond the scope of PADI's course) require the equipment to meet oxygen service specifications.

Enriched air requires a cylinder dedicated specifically to use with enriched air for two reasons.

1 It is critical for safety that no one accidentally confuses the enriched air cylinder for one containing standard air. Therefore, the cylinder must be clearly marked.

2 One method of blending enriched air requires putting pure oxygen in the cylinder. This is called "partial pressure" blending. If partial pressure blending with pure oxygen will be used, the tank and valve must meet oxygen service standards even though the final enriched air blend will have less than 40% oxygen.

As a result, enriched air cylinders have standardised decals and/or tags and colour coding generally agreed upon by the international dive community. These markings ensure that you can readily identify an enriched air tank, determine its contents and determine whether the cylinder can be used for partial pressure blending.

Yellow cylinders should have a 10 centimetre (4 inch) green band around the tank shoulder with yellow or white lettering reading "Enriched Air", "Enriched Air Nitrox", "Nitrox" or a similar designation.

Non-yellow cylinders should have a 15 centimetre (6 inch) band around the tank shoulder. The top and bottom of this band should be a yellow 2.5 centimetre (1 inch) band, with the centre 10 centimetres (4 inches) green. The green portion should have yellow or white lettering reading "Enriched Air", "Enriched Air Nitrox", "Nitrox" or a similar designation.

Enriched air cylinders should have a dated annual visual inspection decal stating that the cylinder has been serviced and inspected for enriched air use. The decal should also indicate if the cylinder does or does not meet oxygen service standards for partial pressure blending.

Additionally, enriched air cylinders should have a contents decal or permanent tag. This decal or tag should, at a minimum, list the oxygen content of the blend the cylinder currently holds, the fill date, the maximum depth for the blend, and the name of the person who analysed the oxygen content to verify the blender's analysis (this should be the diver who will use the tank). Decals are replaced and tags rewritten when the cylinder is refilled.

Besides these markings above, local laws and regulations may require additional or modified markings on enriched air cylinders. Some areas have recommendations or requirements that an enriched air cylinder be used within a given period, such as within 30 days of filling, and the cylinder may be marked accordingly. In other areas, standard air cylinders are stamped "air only", highlighting the need for a dedicated cylinder.

Problems with filling enriched air cylinders

The first is a fire/explosion hazard. Some substances readily burn or explode in the presence of high oxygen concentrations. This includes trace hydrocarbons (lubricants) that may be found in standard compressed air. These trace lubricants may accumulate over time in a compressed air cylinder, raising the potential for fire or explosion hazard if the cylinder is exposed to high oxygen percentages. Similarly, during the filling process compressed gases can back flow into the filling system from an enriched air cylinder. This also poses a potential fire/explosion hazard in the presence of high oxygen concentrations.

The second problem is getting the correct percentage of oxygen in the blend. The amount of oxygen in an enriched air blend is critical. If the percentage of oxygen varies by more than 1%, oxygen exposure, maximum allowable dive depth and no-decompression limits will be affected.

If partial pressure mixing in the cylinder will be used, air used in filling enriched air cylinders must meet oxygen compatibility requirements, Normal compressed air does not meet these requirements. Oxygen compatible air is produced by using special oil-free compressors, special filtration or a combination of both. This is crucial because even trace oil or contaminants may create an explosion/fire hazard. Other methods of producing enriched air do not require putting pure oxygen in the cylinder. These methods greatly reduce filling hazards, but nonetheless, the cylinder must be dedicated for enriched air use and serviced accordingly.

Enriched air blending and filling requires keeping records of system maintenance and fills beyond those required for a conventional compressed air system. Enriched air cylinders should only be filled by reputable, qualified enriched air blenders. Qualified blenders have the proper equipment for producing oxygen compatible air and minimising contamination of equipment that must remain in oxygen service and/or enriched air service. Qualified blenders have the special training required to produce accurate enriched air blends and confirm the accuracy. Qualified blenders have been trained to follow the operational procedures and to maintain the records necessary.

Qualified enriched air blenders and service are identified by checking the gases and procedures used.

1 Gas verification

The operation should be able to show regular analysis of the air it uses for enriched air blending. This air should meet local standards for oxygen compatible air, such as US Compressed Gas Association (CGA) Grade E air standards modified to have no more than 0.1 mg per cubic

metre of detectable hydrocarbons or 10 parts per million of carbon monoxide (many operations try to limit it to two parts per million), or Grade J standards. In all cases, the air should be filtered to eliminate detectable particles (dust, etc.).

2 Proper procedures

Cylinders should be properly marked. There should be good records of gas analysis, filling dates, machinery maintenance and operators actions. A lack of these may indicate that the operation is not qualified or prepared to properly support enriched air diving.

Oxygen analysis

Enriched air is analysed by the blender after blending. Nevertheless, the diver who will be using a cylinder of enriched air also must personally verify the oxygen analysis of the cylinder. Do not dive with a cylinder of enriched air if you have not personally verified its contents. Failure to verify cylinder contents could lead to DCS or drowning due to oxygen toxicity if the cylinder contains an enriched air blend different from what you believe it to be. This is an important safety principle that avoids problems by double checking the initial analysis, verifying that the cylinder has been correctly marked for that blend, and confirming that the cylinder was not accidentally confused with another.

Enriched air must be within 1% of the desired oxygen content. If the blend is more than 1% off from the desired oxygen content, you must recalculate your equivalent air depths (EADs) and oxygen exposure based on the actual content, or have the cylinder refilled with the desired blend.

Oxygen Toxicity

Exceeding oxygen limits can cause central nervous system oxygen toxicity (CNS toxicity). CNS toxicity may cause a diver to convulse. Convulsions are not usually harmful in themselves, but underwater the diver is almost certain to lose the regulator and drown. A fatal accident is the primary serious hazard of exceeding safe oxygen limits. Warning signs and symptoms may precede a CNS convulsions, but usually, CNS convulsions occur without warning.

Warning signs and symptoms, if they do occur, include:

1 Visual disturbances, including tunnel vision
2 Ears ringing
3 Nausea
4 Twitching or muscle spasms, especially in the face
5 Irritability, restlessness, euphoria or anxiety
6 Dizziness.

PADI teaches divers to remember these symptoms by remembering VENTID, which stands for vision, ears, nausea, twitching, irritability and dizziness.

Divers are taught to end the dive and ascend immediately in the presence of any of these symptoms.

Heavy exercise is the thought to predispose CNS toxicity, and should be avoided if you near, or will near, oxygen exposure limits, especially if your dive accidentally exceeds 1.4 bar of oxygen. Some drugs, including the decongestant pseudoephedrine (found in Sudafed and other products), are CNS exciters believed to predispose to CNS toxicity. It is generally recommended that divers avoid decongestants when diving, because they may wear off during the dive, leading to nasal congestion with its accompanying problems. If taking a prescription, divers in training are instructed to consult with a physician knowledgeable in diving medicine before using the drug while diving with air or with enriched air.

Carbon dioxide accumulation in the body is also believed to predispose oxygen toxicity.[3] It's important to breathe continuously to avoid retaining carbon dioxide. If one experiences headaches after a dive, as a precaution, consult a physician familiar with diving to make sure you do not retain carbon dioxide.

Pulmonary or whole body oxygen toxicity is caused by prolonged exposure to high oxygen partial pressures. Exposures of several hours are necessary to develop whole body oxygen toxicity and are highly unlikely within the oxygen exposure limits in the PADI EANx program. Symptoms include burning in the throat and chest, coughing and shortness of breath. Pulmonary oxygen toxicity is more of a concern in technical and commercial dives that require long decompression stops using pure or high amounts of oxygen, 50% or more. Nonetheless, divers are advised to discontinue diving for a few days if symptoms are experienced that could indicate pulmonary oxygen toxicity.

Managing oxygen exposure

The high oxygen partial pressures experienced with enriched air must be kept within limits or they pose serious hazards to the diver. The higher the partial pressure, the shorter time that one can safely be exposed to it. Therefore, one must track oxygen exposure with tables much as one tracks nitrogen exposure. Oxygen exposure limits are independent of depth; they relate entirely to partial pressure. The oxygen partial pressure (PO_2) is 0.80 bar at 10 m (33 ft) using EANx 40. Using EANx 36, one has the same PO_2 at 12 m (40 ft). The oxygen exposure limits are the same for both dives.

The maximum PO_2 for PADI enriched air diving is 1.4 bar. 1.4 bar is the recommended maximum because it keeps you well within established oxygen limits appropriate for recreational diving. Planning a dive within 1.4 bar PO_2 also provides a margin for error. Some evidence suggests that as the PO_2 exceeds the 1.3 to 1.4 bar range, the EAD concept becomes less reliable. Staying at less than 1.4 bar PO_2 reduces the likelihood of problems with this. If the planned dive depth would exceed 1.4 bar, either switch to an enriched air blend with less oxygen, or plan a shallower dive.

The contingency PO_2 limit is 1.6 bar. PADI discourages planning dives with a partial pressure this high because there is no room for error. Partial pressures between 1.4 and 1.6 should be considered a margin for error only. Divers at work have had oxygen toxicity convulsions near 1.6 bar while at work. Exceeding safe oxygen limits poses a high risk of oxygen toxicity.

Dive planning tips for PADI EANx

Treat the entire dive as though it were made at the deepest depth/highest partial pressure. Although NOAA limits do not specify minimum surface intervals, and there is no measurable credit for surface interval, it is recommended that one have a surface interval of at least an hour between enriched air dives whenever possible, especially if one exceeds more than 50% of allowable oxygen exposure. This is believed to further reduce the likelihood of oxygen toxicity.

Do not exceed 100% of allowable exposure in 24 hours. Doing so, even at lower oxygen partial pressures, puts one at risk of oxygen toxicity. It is recommended for extra conservatism that one limits one's exposure to 90%. If planned dives would cause one to approach or exceed oxygen exposure limits, switch to an enriched air with less oxygen and/or plan dives to shallower depths. Maximum allowable dive time is always the shorter of no-decompression time or remaining oxygen exposure time. Always check both.

After a dive in which one inadvertenty exceeded the contingency PO_2 limit of 1.6 bar, the oxygen exposure is considered 100%. It is recommended not to dive for at least 12 hours. The Oxygen Exposure Table allows one to track the accumulating oxygen exposure when making repetitive and multilevel dives with differing oxygen partial pressures. This is sometimes called the "oxygen clock". Because people differ in their physiology, no table, computer or other method of measuring oxygen exposure can guarantee that oxygen toxicity will never occur, even within accepted oxygen limits. In rare instances, oxygen toxicity has occurred within the NOAA limits. Stay well within oxygen limits. It is easy to keep one's oxygen partial pressure well within 1.4 bar by using an enriched air with less oxygen and/or by limiting depth.

Analysis of oxygen exposure limits

The PADI Enriched Air Diver course makes use of a new Diving Science and Technology (DSAT) Oxygen Exposure Table, distributed by PADI. This table is based on the commonly accepted NOAA single exposure limits (Table 1).

TABLE 1

NOAA OXYGEN LIMITS FOR SINGLE EXPOSURES

PO_2	Time
0.6 bar	720 minutes
0.7 bar	570 minutes
0.8 bar	450 minutes
0.9 bar	360 minutes
1.0 bar	300 minutes
1.1 bar	240 minutes
1.2 bar	210 minutes
1.3 bar	180 minutes
1.4 bar	150 minutes
1.5 bar	120 minutes
1.6 bar	45 minutes

The DSAT Oxygen Exposure Table allows the user to convert time at particular PO_2s to a percent of allowable exposure. Exposure in 24 hours may not exceed 100%. This methodology makes it practical in the field to track exposure during repetitive dives, multilevel dives and when using more than one blend of enriched air.

NOAA limits extend total exposures for PO_2s from 1.1 to 1.6 with a minimum surface interval of two hours between exposures. The DSAT Oxygen Exposure Table does not allow this additional exposure time and limits 24 hour exposure to the single exposure limits for the following reasons:

1 The PADI course emphasises keeping PO_2s below 1.4 bar, which is an appropriately conservative level for recreational divers. The significantly reduced time at higher PO_2s encourages this and maintains conservatism if 1.4 is exceeded.

2 Within the realm of no-decompression diving with enriched air, there is little need for greater exposure. Divers who stay within 1.4 bar and make progressively shallower dives will not often find themselves limited by oxygen exposure, even with the existing limits.

TABLE 2

US NAVY OXYGEN LIMITS FOR SINGLE EXPOSURES

PO_2	Time
1.0 bar	240 minutes
1.1 bar	120 minutes
1.2 bar	80 minutes
1.3 bar	60 minutes
1.4 bar	50 minutes
1.5 bar	40 minutes
1.6 bar	30 minutes

3 From a training and educational viewpoint, building in the two hour credit would complicate the table and field use. This increases the possibility of error with little real benefit in the majority of diving situations.

Although the NOAA limits have been widely accepted within the technical, scientific and research dive communities, DSAT could not simply accept the limits on that basis alone. Of particular concern were the old U.S. Navy limits (Table 2), which are more conservative.

Another concern was that much of the testing that led to the NOAA limits was conducted using pure oxygen closed circuit scuba. Tests using semi-closed nitrogen/oxygen mixes suggest that the presence of nitrogen might contribute to the onset of oxygen toxicity.

On the advice of Dr Des Gorman, DSAT compared the NOAA limits with the existing data of manned test dives of oxygen exposure and with the published analysis of, and comments on, those tests by Professor Kenneth Donald.[3] Donald is widely regarded as a leading authority on hyperbaric oxygen exposure, having begun ground breaking research into the field in 1942.

Based on Donald's findings, the NOAA limits employed in the Oxygen Exposure Table seem reasonable and well within the limits of manned tests.

1 The limit of 45 minutes at 1.6 bar seems very conservative and appropriate. Most of the published body of testing oxygen exposure involves PO_2s greater than 1.6 bar. This makes extrapolating to lower PO_2s difficult, but there is a significant (approximate) overlap at the range edge that supports the NOAA limits. Tests by Donald using pure oxygen at 25 fsw (7.5 m), where the PO_2 is 1.75 bar, resulted in few cases of oxygen toxicity and, with only one exception, those that did occur involved underwater exercise and durations beyond 45 minutes. Donald reported that "The Admiralty Experimental Diving Unit was unable to demonstrate oxygen poisoning in the range of 0 to 20 fsw." Using pure oxygen, this is the PO_2 range of 1.0 to 1.6 bar.

Against this data set, a shorter time limit of 45 minutes at the higher PO_2 limit of 1.6 bar seems reasonable. With the emphasis on a maximum PO_2 of 1.4 bar, the exposure in the DSAT table seems appropriate. The time limits for PO_2s below 1.4 bar stem more from pulmonary oxygen toxicity concerns than from CNS (acute) toxicity[4] An analysis of the exposure limits on the DSAT table when calculated as Oxygen Tolerance Units (OTUs) shows a maximum of approximately 300 OTUs at a PO_2 of 1.0 bar, which conforms with the daily OTU dose recommended by the Repex oxygen exposure limits for repeated daily exposure to oxygen.[6]

2 Donald discounted the old Navy limits as unrealistically conservative. In *Oxygen and the Diver*, he says "Time limits were also given from 30 minutes at 1.6 bar to 240 minutes at 1.0 bar. These time limits appear to have been quite arbitrary and unrelated to acute or pulmonary oxygen poisoning. These restrictions cause a considerable limitation in the scope of mixture diving." According to Donald, the US Navy limits may have resulted because tests by Lanphier seemed to show a possible reduction in oxygen tolerance when breathing nitrogen/oxygen mixtures.

3 Donald did not believe the evidence supported the idea that nitrogen/oxygen mixtures increase the probability of oxygen toxicity. He cites the limited data that Lanphier based his conclusion on, and cites experiments that show the nitrogen has neither a positive nor negative effect. Donald stated "Thus [Lanphier's] total evidence that, contrary to the Royal Naval findings (Donald, 1943 (i) & (ii)) and experience, oxygen was more toxic when breathed in oxygen-nitrogen mixtures is of little formal significance".[3]

4 A more recent question involves the role of carbon dioxide retention in causing oxygen toxicity. Although few individuals retain carbon dioxide, especially when using conventional open circuit scuba equipment, recent tests support the limit of 1.4 bar of oxygen as appropriate when an individual's carbon dioxide retention is not known.[6]

Field data support both the DSAT limits and Donald's research and experience. The NOAA limits have been in use more than a decade, with virtually no incidence of oxygen toxicity reported within the proposed range. With the additional conservatism built in, the DSAT Oxygen Exposure Table appears to be well suited for use by recreational divers.

Computer dives using enriched air

The optimum method for diving enriched air with a computer is to use an enriched air computer. Following the manufacturer's instructions, one programs these computers with the blend one is going to use, the computer tracks no-decompression status and oxygen exposure. If one already owns an air dive computer, one can use enriched air with it. The simplest way is to plan the dive as a standard single depth enriched air dive using EADs and tables. Then, dive with the air computer. During the dive, one can use whichever gives more no-decompression time, the computer or the EADs and tables. However, remember that repetitive dives must be calculated based on what one followed in the first dive.

It is important to plan and track oxygen exposure, especially for long multilevel dives. Plan dives to ascend in levels, calculate each level's oxygen exposure separately and add them together. Start at the deepest point of the dive and progressively work shallower, stay at or above the depth levels on which one based the oxygen exposure. If one does not track oxygen levels, one must base one's oxygen exposure on the deepest depth and total dive time. If only making one or two dives, one may find this much simpler.

Diving emergencies and enriched air

If a diver convulses underwater (possibly due to oxygen toxicity), the generally recommended action is to handle the emergency as one would for an unconscious diver underwater. This recommendation is based on US Navy procedures, which the Divers Alert Network (DAN) defers to in this situation because there has been little study of this in recreational diving.

a Hold the diver's mouthpiece in (if still retained). Do not attempt to replace it if it is out of the mouth.

b Immediately bring the diver to the surface and check for breathing.

c Establish ample positive buoyancy for both rescuer and victim.

d Call for assistance as needed and available and begin in-water rescue breaths if the victim is not breathing. Take the diver to the boat or shore and help remove the diver from the water.

e Once out of the water, check for a pulse and breathing. If they are absent, begin or continue rescue breaths and/or CPR. In any case, contact emergency medical care. If the diver is breathing, begin first aid for DCI as a precaution.

f Even if the diver appears fully recovered, the patient should be examined by a physician.

Some experts recommend that if a diver's mouthpiece is in place, one should hold it there and not begin the ascent until the convulsion subsides. After the convulsion ends, bring the diver immediately to the surface. This recommendation is based on the fact that a convulsing diver may hold his breath. In any case, the primary concern is getting the diver to the surface to prevent drowning, so one can begin first aid and get help.

If a diver is suspected of having decompression illness after an enriched air dive, administer oxygen first aid and obtain emergency help exactly as one would if the diver had been diving using air. If possible, inform emergency personnel and the recompression facility that what the diver's time and depth was, that the diver was using enriched air, and what the blend was. In a DCI emergency, if you run out of emergency oxygen before you can get a breathing patient into emergency medical care, have the patient breathe any enriched air available. While not as beneficial as 100% oxygen, enriched air has more oxygen than air.

For any questions or comments regarding the PADI EANx course, contact the authors.

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DEEP WATER BLACKOUT

David Elliott

Key Words

Accidents, carbon dioxide, deep diving, nitrogen narcosis, oxygen, unconsciousness.

This review is the first of several to assess some of the physiological hazards and associated risks of what has been termed "advanced recreational diving." This includes nitrox diving, extreme air diving, technical diving and the use of rebreathers. It is instructive to look first at the interactions of carbon dioxide and hyperbaric oxygen in "shallow water blackout" as an introduction to the potentially more complex synergisms that may occur in the presence of nitrogen narcosis, as in "deep water blackout". Shallow water blackout remains a hazard for those, such as movie cameramen, who use closed-circuit oxygen breathing apparatus. Deep water blackout, as described, is however associated not with oxy-nitrogen rebreathers, but with the use of open-circuit compressed air breathing apparatus.

Loss of consciousness underwater is a serious event, particularly for a diver wearing a half-mask and using a mouthpiece, because the most likely outcome is drowning. Of the many causes of impaired consciousness at depth, the concept of deep water blackout is distinct from the more obvious possibilities such as carbon monoxide poisoning and myocardial infarction. Deep water blackout is part of an ill-defined and fortunately rare group of incidents which are best titled "loss of consciousness of unknown aetiology" and this phenomenon appears to be a hazard for only those compressed air divers who swim deeper than the limits recommended by most recreational training agencies.

In essence, the circumstantial evidence is that under certain conditions the swimming diver on open-circuit compressed air can lapse into unconsciousness at depths below 50 m (165 ft) without a primary cause being obvious. The importance of these considerations relates to the risks that are undertaken by the so-called "Extreme Air" divers. "How deep do you dive?" is a siren call to the novice. In 1990 Gillam achieved the depth of 452 feet (133 m) with compressed air scuba (attaining, as he did so, a PO_2 of 2.9 bar) and, since then, Marion has reached 513 feet (156 m). A number of those wishing to take an even deeper place in a book of diving records have died at depth, maybe deeper than the holders, maybe not. Perhaps these records and deaths are merely a reflection of wide biological variation but the well-informed deep diver needs to be aware that there are a number of relatively unquantifiable risks and also needs to know that, to get a world record recognised, the diver must make it back to the surface.

Within this general category of underwater loss of consciousness falls another longstanding concept, that of the shallow water blackout. Historically the term is firmly associated with the use of closed circuit breathing apparatus using 100% oxygen. It is important to put aside the subsequent adoption of this term for hypoxic incidents associated with prolonged breath-hold diving. Shallow water blackout was first investigated more than 50 years ago and was well described by Donald.[1]

Shallow water blackout

A number of unexplained cases of impairment or loss of consciousness were reported some 55 years ago[1,2] among those swimming with pure oxygen rebreathers at depths less than 25 feet (7.5 m, 1.8 bar PO_2). There were no convulsions or other signs or symptoms of oxygen toxicity and recovery was rapid, once out of the water. Confusion and disorientation were common, headache, nausea and respiratory distress less so. Barlow and McIntosh[3] were able to exclude as causes of this the effects of pulmonary overpressure while immersed and also "dilution hypoxia". Dilution hypoxia, a problem unique to rebreathers, is a situation which occurs when the available oxygen in the counter lung is consumed leaving the diver to breathe only some of the nitrogen which has been excreted from the tissues into the counter lung. This hazard of hypoxia is "silent" because of the absence of any CO_2 build-up, as would usually be associated with hypoxia, because the CO_2 is constantly removed by the scrubber in the circuit. The risk of fatal hypoxia in these circumstances is minimised by a meticulous two minute "nitrogen wash-out" procedure of breathing oxygen and then emptying the breathing bag before descent.

The effects of high concentrations of carbon dioxide in the absence of oxygen lack were examined[3] and showed impairment or loss of consciousness when exercising hard on pure oxygen breathed through 800 ml external dead space. As a result of these and other studies, the CO_2 scrubber was improved and the number of incidents diminished. Nevertheless unexpected impairment or loss of consciousness was still encountered and, besides the specific circumstances of hyperoxia and hypercarbia, Donald looked towards a synergism between oxygen poisoning, CO_2

intoxication and hypoxia due to procedural errors, as being among other causes.[1]

Carbon dioxide retainers

Some divers might be more susceptible to underwater incidents of impaired consciousness because they do not hyperventilate in response to CO_2. The existence of a proportion of "CO_2 retainers" among a population of regular naval divers was proposed by Lanphier.[4] Such persons appear to have acquired CO_2 hyporeactivity and, once acquired, it is not temporary but appears to persist. This aspect of underwater respiratory physiology has been reviewed recently by Lanphier and Camporesi.[5]

Nitrogen

Although the intoxicating effects of breathing compressed air at depth had been noted for some years it was not until 100 years later that this effect was attributed to the specific narcotic action of a raised partial pressure of nitrogen.[6] The manifestations of nitrogen narcosis are proportionate to the partial pressure of the inspired nitrogen and, subject to individual variability, begin to be noticed during descent from around 30 m (4 bar). Nevertheless, mental impairment due to nitrogen certainly occurs at less than 3 bar and effects have been reported at 2 bar.[8] Narcosis increases to the extent that early reports described a "semi-loss of consciousness" on air at depths as great 350 ft (105 m, 11.6 bar). The signs and symptoms are similar to those of alcoholic intoxication and there may be an impairment of a diver's ability to recognise and cope with a diving emergency when it occurs. Down to some 50 m (6 bar) the individual may find only that concentration is difficult and that there may be a slightly impaired degree of neuromuscular co-ordination. Before codes of safe diving practice advised against compressed-air diving deeper than some 50 m, experience demonstrated that only very few divers could accomplish useful work at depths greater than 90 m (10 bar). Although compressed air has been breathed at depths as great as 600 ft (180 m, 19 bar) in submarine escape procedures, the duration of this exposure was deliberately kept short and thus was within the latent period of onset of narcosis. One of the deepest recorded experience is that of Goodman[8] who describes the glassy appearance of the diver's eyes at 462 ft (144 m; 15 bar) as suggesting those of the "firmly plastered drinker" and adds that after some 45 seconds the simple task of assembling pegs had deteriorated to mere fumbling. "Bending forward ever more closely over his 'precious' pegboard, with intermittent bursts of inappropriate laughter and hearty, self-satisfied chuckling, the subject has, after 90 seconds of air breathing, effectively retreated into a private world."

The mechanism of narcosis is the same as that of the gaseous anaesthetics and that of alcohol intoxication. The individual passes through similar stages in each, from excitation to sleep. The biophysical basis for narcosis is well reviewed elsewhere.[9] Of interest in the practical situation is the interaction and possible potentiation at depth of narcosis by the effects of the individual respiratory gases, oxygen and carbon dioxide.

Oxygen

The pulmonary and neurological features of oxygen toxicity are reasonably well known but the behaviour of oxygen as an "inert" (i.e. narcotic) gas has received less attention. The greater the depth of the oxygen exposure the greater its relevance.

> In 1970 a 15-minute oxy-helium bounce dive in a bell with an air atmosphere to 400 feet (120 m) was done by 2 divers. On completion, they were hoisted in the bell for in-water stops until they reached 180 feet (55 m) where the stop duration was long enough to shut the bottom door. At this stop there was a switch on BIBS (built in breathing system) from 10/90 oxy-helium to 20/80 prior to transfer-under-pressure to the deck chamber. A medical officer was watching the video monitor of the bell interior during the transfer and within a few minutes of the gas switch was alerted by a "squark" noise from one diver. First one and then the other was seen to lapse, over a period of several seconds, into unconsciousness. There was no fit, they each just slowly slid down the side of the bell to the floor. This was not a foreseen emergency and, even when locked on to the deck chamber, they were still inaccessible. It seemed a long time before the first began to recover. In fact it was the one who had been seen talking and taking his BIBS out of his mouth to do so. It was two and a half hours before the second began to recover by which time a decompression schedule had begun from an initial emergency recompression.

It was later found that at 55 m they had, in error, switched from 10/90 to pure oxygen.

At that depth the PO_2 was 6.5 bar. The civilian consultant to the Royal Navy, Professor Bill Paton, said later "Didn't you realise that oxygen could be an anaesthetic?" and quoted an earlier paper of his to support this.[10] Certainly it was oxygen and certainly no obvious fit was seen. They both were deeply unconscious for a long time and, as also reported by Donald,[1] oxygen neurotoxicity is not confined to epileptiform fits.

Oxygen, not surprisingly, also potentiates narcosis. Fenn[11] suggested that this was a direct action, rather than one associated with the known potentiation associated with carbon dioxide retention.

Carbon dioxide

It was proposed that nitrogen narcosis is enhanced by carbon dioxide retention by Case and Haldane.[12] This was demonstrated by Hesser et al.[13] who, while holding both PN_2 and PO_2 constant, had subjects inhale various CO_2 mixtures to increase alveolar PCO_2. The interaction caused tracking task errors to be significantly increased.

However carbon dioxide retention is not the cause of depth narcosis[14] and, indeed, the symptoms of depth narcosis and of carbon dioxide at depth are quite different.[15]

Deep water blackout

After recognising the potential effects of CO_2, oxygen and nitrogen at raised pressure, the concept of deep water blackout began to take shape. It was based on a few well-observed cases. These may be relatively anecdotal, but where else to begin?

The current depth limit for working divers on air for most countries is around 50 m (165 ft) and this is a sensible limit. However, in the days when the Navy limit was set at 180 feet (55 m), some deeper interventions were allowed for selected tasks such as aircraft salvage.

A ditched helicopter which sinks must be retrieved rapidly before corrosion sets in if the cause of the accident is to be found and possible additional aircraft losses are to be prevented. On call for this purpose the Royal Navy had a special team that would dive to around 250 ft (76 m) on the Deep Air Oxygen Table 13 when needed. Typically this would be for a 16-minuted bottom time at 250 ft (76 m) which requires 35 minutes of decompression with in-water oxygen stops from 60 ft (18 m). This might appear to be reckless diving but not so. The essential difference between military diving and "extreme recreational diving" lies in the assessment of hazards and in maintaining control of the risks. Some sport divers will go solo to deeper than 90 m (300 ft) on a single scuba tank of compressed air, carrying no spare regulator or tank.

In contrast, many navy divers did the deep air dive, but only as a member of a team. The team, as many as 8 in all, would support *only one diver* in the water at any time. The dive would be on a *surface supplied hose* leading via a demand valve to a *full face mask*. At the surface control gas panel the diver's supply would be switched to oxygen when he reached 18 m (60 ft) but, as he was at the end of a 300 ft (90 m) hose, to breathe down the volume of air would take him approximately until he reached his 30 ft (9 m) stop. A challenge for a decompression mathematician, but a diving procedure that proved to be safe operationally for a well equipped team.

Because of the need for the "Helicopter Team" to keep in practice between their fortunately rare emergency call-outs, they used to come to the Deep Trials Unit periodically for a deep air diving experience in the controlled environment of the wet pot of a large chamber complex. It was here that one could watch on television screens how well divers performed on air at deep depths. The wet pot was about 2 metres deep and pressurised to 8.4 bar. It was fitted with a "trapeze" against which the diver had to push very hard while swimming to keep a particular mark in place on the wall in front of him. On several occasions individuals were obviously narcotic, attempting to dissemble their equipment or responding to commands in an aimless manner. On surfacing, "That was a good dive", they had no memory of this and when shown the video of their errors, they would assert that "It must be somebody else". Thus the team became well informed of the hazards of deep air.

More worrying were the occasional lapses of several divers into unconsciousness. The divers were, of course, wearing a full face mask, so there was never any risk of drowning, and the unconsciousness was over in less than a minute but, for anyone using a half-mask and a mouth-piece, that would have been long enough to get into serious trouble. My certain recollection is that it was those who exercised hardest who got into trouble. The possible factors for deep water blackout appear to be:-

carbon dioxide: associated with a short burst of motivated hard exercise. Some members of the group might also have been in the category of CO_2 retainers

oxygen : greater than PO_2 1.6 bar;

nitrogen : at these depths narcotic at rest and potentiated by carbon dioxide.

It remains a phenomenon that may deserve a full investigation but, regardless of any scientific study, it will always be there waiting to catch just some of the uninformed.

Those who have attempted to break the depth record for compressed air scuba have probably not been fully informed. They have done so in ignorance of the risks and some have paid the ultimate price. Among the survivors of deep air diving one can expect an attitude, common towards doctors in early experimental deep diving, "We don't want you telling us not to do it. Just let us get on with it. Then you can tell us later how we did it." The answer is that they were fortunate, unlike those who have not yet resurfaced.

Conclusions

Using one definition of "Technical Diving", which is that at some point in a technical dive there is a change of breathing mixture, deep air diving is **not** technical diving. Extreme scuba diving on air is just stupid.

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ARTICLES OF INTEREST REPRINTED FROM OTHER JOURNALS

BUOYANCY

Key words
Buoyancy, training, reprints.

Buoyancy control is one of the most important skills to master if your diving is to be safe, let alone comfortable or good for the environment. You may have been diving for quite a long time but are you diving at your correct buoyancy?

First you should plan your buoyancy and the extent to which you can change your buoyancy according to the type of dive. If you need to work on the bottom, carry out photography, dive with trainees or carry out decompression stops you may need to be able to adjust your buoyancy so that you are a kilogram or two negative near the surface at the end of the dive as well as at any other time. But if you are carrying out a very standard "look and see" dive with no decompression then you need to be close to neutral buoyancy throughout the dive.

If you are negative you run the risks of
1 Uncontrolled descent into unintended depths
2 Stirring up the sea bed, which is bad for marine life and for visibility.
3 A reduced margin of safety if you need to ascend relatively rapidly, if you need to help another diver surface, or if you meet descending currents.

How do you achieve neutral buoyancy ? The very first piece of training comes in the pool when you demonstrate neutral buoyancy. Both instructors and

trainees often get this wrong. They imagine that if your torso and head rise when you breathe in then you must be neutral. All that may be happening is that you pivot on your fins which remain firmly on the bottom and you are still negative but did not realise this. The instructor may need to hold the trainee off the bottom and let the trainee adjust buoyancy until the instructor has a very slight weight to hold. So learning to appreciate when you are really neutral is an important part of the training.

You should consider your gear once again when you get into the sea. Wet-suited divers are often fairly sensibly weighted but dry suited divers often go about the matter the wrong way. The wrong idea is to wear bulky underclothing, dive with a lot of air in the suit and an enormously heavy weight belt.

If you are wearing more than 13.5 kg (30 lbs) you are probably overweight because should your suit flood or lose most of its air your buoyancy aid (BC, ABLJ or stab jacket) may have insufficient lift to raise you unless you drop your weight belt.

In any event you will carry so much air in your suit that any change in level is likely to mean appreciable additions of air or dumping of air from your suit to keep neutral buoyancy You may not be able to do this rapidly. Draeger dump valves often do not dump air particularly rapidly. If you dive with a Zeagle or similar stab jacket you may see merit in wearing a weight belt that carries insufficient weight to hold you down but enough to prevent a very fast ascent.

The remaining weight is placed in the pockets of the jacket where it can either be dumped quickly by pulling the release or more slowly by taking it out by hand. You can choose to dump either set of weights or both in serious situations. So if you dive with a dry suit and more than 13.5 kg (30 lbs) of weight take thought or advice. Some membrane dry suits with appropriate undersuits are rated at 8 kg (17-181b) of weight (for a large man) by the suit makers and compressed neoprene dry suits at only slightly more.

It is interesting and relevant to note that in quite a number of recent fatal diving accidents the victims had been wearing more than 16 kg (35 lbs) of weight on their belts!

But above all learn to sense changes in your buoyancy as you dive.

Reprinted, with minor editing, by kind permission of the Editors, from SCOTTISH DIVER 1995; 34 (1): 21.-22 The address of SCOTTISH DIVER is Scottish Sub-Aqua Club, The Cockburn Centre, 40 Bogmoor Place, Glasgow G51 4TQ, United Kingdom.

GLEANINGS FROM MEDICAL JOURNALS

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

VALSALVA MANOEUVRE AND VITREOUS HAEMORRHAGE

Valsalva maneuver induced vitreous hemorrhage
Jones WL. *J Am Optometric Assoc* 1995; 66 (5); 301-304

Abstract

Background: A 43 year old male who performed a severe and sustained Valsalva manoeuvre developed a vitreous haemorrhage that was suspected as originating from an optic nerve vessel. Valsalva haemorrhagic retinopathy is not a common ocular finding and a breakthrough into the vitreous is even more uncommon. The vitreous haemorrhage cleared with recovery of previous acuity levels and a residual of small areas of fibrosis in the inferior vitreous. A discussion of the physiology of the Valsalva manoeuvre and of possible ocular and non-ocular complications is presented. There is also a discussion of how this manoeuvre is used in the diagnosis of orbital diseases.

Conclusion: In this case a vitreous haemorrhage was induced by a marked and sustained Valsalva manoeuvre which resolved without significant sequelae. The Valsalva manoeuvre is capable of causing many ocular complications; it is important to advise patients about the potential side effects of this manoeuvre.

Key Words
Reprint, Valsalva manoeuvre, vitreous haemorrhage.

ANXIETY AND PANIC IN DIVERS

Anxiety and Panic in Recreational Scuba Divers
Morgan WP. *Sports Med* 1995; 20 (6); 398-421.

Abstract

In the early 1980s it was estimated that 1 to 1.5 million Americans were involved in scuba diving as a form of recreation. While the number of active divers in the US remains a matter of controversy, the most recent estimate falls between 2.7 and 3.1 million.

Scuba diving is a high-risk sport; it is estimated that 3 to 9 deaths per 100 000 divers occur annually in the US alone, in addition to increasing numbers of cases of decompression illness each year. However, there has been a tendency within the diving community to de-emphasise the risks associated with scuba diving. While there are numerous factors responsible for the injuries and fatalities occurring in this sport, there is general consensus that many of these cases are caused by panic.

There has been a tendency to focus on variables such as age, diving experience, use of alcohol and drugs, and type of dive (e.g. cave, deep water, wreck) when looking at causes of diving fatalities. While the cause of death is listed as drowning in more than 60% of scuba diving fatalities, drowning is usually caused by specific problems such as: lack of air; entanglement in fishing nets, rope, or kelp; air embolism; narcosis and panic.

While 60% of diving fatalities can be attributed to medical, environmental or equipment problems, the remaining 40% are usually classified as unexplained or due to "undetermined" causes. However, there have been anecdotal reports of divers removing their regulators when exposed to stressors provoking anxiety and panic responses and reports of rapid ascents as a consequence of panic.

There is also evidence that individuals who are characterised by elevated levels of trait anxiety are more likely to have greater state anxiety responses when exposed to a stressor, and hence, this sub-group of the diving population is at an increased level of risk.

Panic behaviour represents one of the most significant problems confronting divers of all ages and both sexes. Panic is not restricted to beginning divers; competent divers with many years of experience report that they have experienced panic. Despite the fact that panic can be a significant problem in scuba diving, there has been a tendency for diving organisations to ignore this problem. Also, editors and authors of introductory scuba books have typically not included discussions of anxiety and panic. Indeed, there is a conspicuous absence of terms such as anxiety, panic, stress and fatalities in most of these publications.

Key Words
Panic, recreational diving, reprint.

TOURIST HEALTH

Tourist health services at tropical island resorts.
Wilks J, Walker S, Wood M, Nicol J and Oldenburg B. *Aust Health Rev* 1995; 18 (3): 45-62

Abstract

An unfamiliar holiday environment may give rise to a range of common and unique medical conditions and injuries. Based on a six-month retrospective analysis of clinic records, the present study reports a detailed profile of health services provided for guests at three tropical island tourist resorts. A total of 1183 clinic visits were analysed; 735 (62 per cent) concerned medical conditions, while 448 (38 per cent) were related to injuries. The most common medical conditions treated were respiratory, digestive, skin problems, eye disorders and genito-urinary complaints. Injuries included lacerations, bites and stings, sprains and fractures. Health services consisted mainly of medication, specialist nursing care and first aid. Study findings highlight the advantages of using an internationally accepted classification system for primary health research, and the critical role of the nurse in tourist health services.

Key Words
Health, tourism, reprint.

MARINE TOURISM

Remote nursing services at island tourist resorts.
Wilks J, Walker S, Wood M, Nicol J and Oldenberg B. *Aust J Rural Health* 1995; 3: 179-185

Abstract

Based on a 6 month retrospective analysis of clinic records, the present study reports a detailed profile of nursing services provided for guests at three tropical island tourist resorts. A total of 1,183 clinic visits were analysed, with presentations coded according to the International Classification of Primary Care (ICPC). The main reasons for presentation were skin problems, ear disorders, respiratory, digestive and musculoskeletal complaints. Health services consisted mainly of education, specialist nursing care and first aid. Study findings highlight the importance of using an internationally accepted classification system for primary health research and the critical role of the nurse in tourist health services.

Key words
Health, tourism, reprint.

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SAFE LIMITS: AN INTERNATIONAL DIVE SYMPOSIUM
(Continued from SPUMS J 1995; 25 (3): pages 153-170 and SPUMS J 1996; 26 (2):pages 108-140)

SEVERAL CASE STUDIES FROM GENERAL PRACTICE

Peter Chapman-Smith

A common but misleading case of decompression illness

This 29 year old clerk presented in my general practice surgery on a Tuesday morning. Two days earlier, he had been scuba diving for crayfish off the Hen and Chicken Islands near Whangarei. A regular diver, although not following any named tables, he utilised and had faith in an Aladin dive computer. The conditions at sea were surgy, his buddy being seasick. He had dived 3 or 4 times from midday, into decompression times for the first time. Following diving to 25 m for 38 minutes, he ascended. 20 minutes at 3 m at the end of this time, with 10 minutes on the surface during this shallow period. After a 2 hour surface interval, a second dive to 12 m for 40 minutes with a 3 m stop for 10 minutes followed.

He had experienced nausea, and chest tightness on that day. The next morning he was lethargic, just not himself, and was anxious.

In the past he had had a significant motor vehicle accident (MVA) 10 years before, sustaining multiple fractures, was on no medications and he was evidently allergic to Penicillin. Both shoulders had been reconstructed".

His detailed examination findings were quite normal, and he was reassured and sent home with the offer to return if not settling.

He came back 3 days later, with persistent symptoms. Still nauseated, with lethargy, tightness in his chest, now unsteadiness, and a niggly pain in his right shoulder. He had been to the chiropractor the day before for treatment on his own initiative.

Full neurological examination was still normal, including a sharpened Romberg test. His peak flow was 610 l/min, and there were no chest or cardiac signs.

A provisional diagnosis of decompression illness was made, and he was referred to Auckland for a trial of recompression therapy. Treated with a single treatment, on oxygen at 10 m for 60 minutes then a 30 minutes ascent, all signs and symptoms were relieved immediately according to the Naval Hospital report. His 100-7 test improved from 60 to 40 seconds, and some degree of left hip weakness of extension and flexion disappeared (previously ascribed to his old MVA). This was considered to be multifocal neurological non-progressive decompression illness.

After 24 hours he was well and was discharged to go home, with a 1 month ban on scuba diving. Reviewed at my rooms 1 week later with his wife, he reported some ongoing chest tightness, had tried Voltaren but felt this had not helped.

At follow up 1 month after the event, he was having difficulty coping with stress, was having mood swings, tending to cry and was labile in his mood. Memory was fine and he had not been diving.

His case was chosen to highlight a very common description of decompression illness, that is persistent tiredness, with patient insight that all is not well. "I am just not right!" is a common description. Examination can be often be quite unfruitful. A clear history, the ongoing nature of the problems, and complete resolution of symptoms with a trial of recompression treatment often confirms what is suspected.

Multiple presentations of decompression illness

A 19 year old male suffered from musculoskeletal decompression illness in July 1993 in New Zealand, presenting with a 3 day history of fleeting joint discomfort and paraesthesiae, and a one day history of upper limb weakness and lethargy. The previous day he had been scuba diving with a conservative profile, to 12 m for 35 minutes, then making a rapid ascent at the end of the first dive to assist his buddy experiencing loss of buoyancy control. After 1 hour and a quarter on the surface, he dived again to 8.5 m for 35 minutes chasing those elusive crayfish, but made 3 ascents during the dive. After diving he was well.

One day later, he became aware of some slight fleeting joint discomfort and tingling affecting his right wrist, knee and hip. Unconcerned, he went fishing for the day. That night his arms were much less strong, tiredness was more so, with increased paraesthesiae.

A PADI diver, with 110 logged dives to this date, and no other medical or diving history of note, he had been welding and using a power saw the month before this.

Examination findings were remarkably normal, with a soft flow murmur noted, but no neurocognitive deficit. The murmur disappeared before his hospital discharge.

With typically vague symptoms, and affected joints 24 hours after diving, decompression illness was presumed. A single recompression and IV fluids were given. There were 2 extensions to the table at 18m before decompression. All his symptoms completely resolved by the next morning. He was advised not to fly or dive for 1 month.

4 months later he came for a commercial diver's medical. A trained scuba diver for 3 years, he had a past history of tonsillectomy and grommets. When examined, a systolic murmur and early diastolic murmur were noted. He admitted to very long standing ongoing enuresis 2-3 times weekly. His mid-stream urine was normal, as was an echocardiogram. A 2D colour doppler echocardiogram was recommended but not done. He was reviewed by a physician who diagnosed a trivial flow murmur only, with complete right bundle branch block noted, as well as posterior fascicular block on an ECG.

He came back 2 months later unwell, with myalgia, photosensitivity, and feeling just not right. He felt lethargic, forgetful, unwell and was aware of poor mental concentration. Over Christmas he had been diving a lot, well within accepted times. Mostly these were single daily dives at the Poor Knights Islands, between depths of 10 and 25 m. 20-25 minute bottom times were average, with the longest exposure to nitrogen being a dive 1 month earlier to 23.5 m for 38 minutes.

Now he was 10 days post dive, with aching shoulders, knees and near the joints he felt some pain. Short term memory was reduced, and blood tests were expectedly normal. A lean and reserved young man, his detailed neurological examination was normal. After discussion he was transferred to the Royal New Zealand Navy Hospital (RNZNH) for recompression. On arrival he was noted to have poor attention, but no abnormal signs were detected in his cardiovascular and neurological systems.

Presuming chronic static constitutional decompression illness, a trial of pressure (Table 62) was conducted. The following day his feelings of malaise and lethargy were gone. 24 hours later he went home well.

Reviewed 1 week later, all of his symptoms were gone subjectively. Mentally more acute, and well coordinated, he was advised against flying and diving for one month. A further month passed, he reported no symptoms at all, and was encouraged not to pursue his planned career of commercial diving.

Interestingly, his mother came to advise me that he was born at 38 weeks, with low oestriols, and his childhood milestones were normal. Recurrent tonsillitis and otitis media were childhood illnesses, but he continued to do well at school. At the age of 11 years, he was labelled as a minor dyslexic, although with a reading age of 7 and a half years. His short term memory failed him, and his mother insisted that his work efficiency improved when under pressure. Over the years she reported purchasing many tubes of "Deep Heat", a heat rub, for frequent complaints of vague musculoskeletal pains.

Interestingly, when in Vanuatu, diving with his father 5 months earlier, decompression profiles were chosen on 5 days with a maximum depth of 58 m. There were 75 hours without diving before flying out. His first episode of diving illness was to present 2 weeks later. His father had made similar dives, and was well at this time.

When he became unwell 5 months later, his father also became quite feverish and unwell within hours. They both experienced chills, rigors, and malaise for about 18 hours.

Clearly he was advised not to undertake a career of commercial diving at this point. A full recovery was noted but an informed consent approach was taken about further recreational diving.

Five months after his second treatment at RNZNH, he reported daily hip and knee pain, with onset one hour after rising. At times severe, he stated he could not stand for more than an hour without the need to sit or lie down. A depressed mood prevailed, with interest shown to exclude tropical diseases such as dengue fever. Antibodies for this were negative, and other bloods were normal.

He demonstrated 2 apparent separate episodes of decompression illness 6 months apart, the first after more provocative profiles, and an excellent response to recompression first aid with relapsing ongoing musculoskeletal pain within months. Depression colours the picture, a not uncommon scenario.

Recurrent constitutional decompression illness

This 47 year old self employed mechanic presented with his third episode of decompression illness requiring recompression in January 1993. He had been successfully treated with recompression in 1977 and 1989. At this time he was advised not to dive again, but had done many dives since, always completing a 3 m stop.

He had been chasing crayfish at 5 p.m. two days earlier, his maximum depth was 16 m with a bottom time of 41 minutes. In good, calm sea conditions, he has sat on a rock at 6 m to de
compress until his gauge showed 150 psi. A slow ascent followed to the surface as usual.

He returned to work to complete a job, and felt well. Two hours after the dive he noted an earache on the left. There were no other ear symptoms, and he took an aspirin. The next morning he awoke with earache still, but in both ears and with pain in both legs also. All over his skin, he was aware of altered sensation. His ears felt blocked, and he knew he was "not right". A complete physical examination was normal, and mild exostoses were noted in both ears. He was referred for recompression treatment in Auckland, and he required multiple treatments for recalcitrant symptoms. Forty eight hours after leaving RNZNH to return home, he relapsed and complained of numbness in both feet, and a fuzzy sensation in his head. He returned to RNZNH for a further 5 treatments in the chamber. Three days later he felt "100%". He celebrated with 2 cans of beer that night.

Two days later his head felt pressurised, with frequent headaches and fortunately no balance problems or inner ear symptoms other than some tinnitus. A further detailed physical examination was normal. His audiogram remained symmetrical with a minor high frequency loss, probably occupational in nature, with 60-70 db loss above 6000 Hz.

He was reviewed one month later with his wife. Both ears felt blocked 60% of the time, with earache, and subjectively some increase in his hearing loss. Tinnitus was a constant and harrowing problem. Lethargy was a feature, with a desire to go home to bed for a snooze about 2 p.m. A trial of low dose amitryptiline and diazepam was just sedative. Simple analgesics were helpful.

Over the next 2 months, occipital headaches persisted daily. Some features of depression emerged, namely insomnia, low mood, tiredness, and poor energy. He was aware of a staggering gait after drinking minimal alcohol, and his frustration was ongoing as several antidepressants were tried without success. A short term memory deficit now affected his usual job as a mechanic. To stop during a job to answer a phone call or an enquiry would mean he had to restart the entire job again, not knowing what had been done to that point. Insight was quite clear. He estimated his work output as being 40% of his usual. His attention span was poor, with poor libido, problems starting micturition, less frequent bowel motions and insomnia, awake for 4 to 5 hours each night. Minimal motion now made him seasick. His physical examination now demonstrated poor balance, being unable to stand on either leg alone with eyes closed, and a sharpened Romberg test of 3 seconds! He was referred to the local Rehabilitation Unit and an MRI was considered but not done. The orthopaedic consultant in charge replied suggesting from my letter perhaps he had "a reversible organic brain syndrome"!

A reassessment was done at RNZNH on the next obvious referral for assessment, now 6 months after treatment. A little improvement had occurred, but the earache, insomnia, buzzing tinnitus and memory problems were ongoing.

An ENT assessment 2 months later was to reveal nothing new. A formal neurological consultation was arranged a further 2 months later, now 10 months after presentation. The neurologist declared there was no other underlying illness at all, thought he was depressed, and suggested a psychiatric referral . At this time his left sole of his foot was numb, especially the heel which also ached, more so in the cold. His recall for simple names, of good friends and details of vehicles that he had attended recent expensive update courses on maintaining were often blank. A new ache in the groin was worrying him.

Four months later, now 15 months after his illness, he reported feeling moody, irritable, with frequent headaches, worse bladder dysfunction, not incontinence but a poor sense of bladder volume. Still forgetful, but a little less so, he estimated his work output as now 60%. Duodenal ulcer symptoms appeared. A course of Famotidine was helpful, as was a trial of Prozac. He continued to make silly expensive mistakes in his business.

He was then referred for a head injury assessment with a local speech therapist, which confirmed his cognitive deficit, which is ongoing. Auditory short term memory deficit, especially at paragraph and information levels were his main dysfunction. Less so was the inability with written material. Errors in word finding and associative thinking and in name recall were obvious. Some compensation had occurred, but real problems remain in his running a business.

In short, a poor result to his third therapeutic recompression and a further example that the current level of treatment is but rudimentary. As is often the case, opinions from specialist colleagues are blinkered by conventional thought, are frustrating and depressing for the individual with decompression illness.

Key Words

DCI, sequelae, treatment.

Dr Peter Chapman-Smith is a General Medical Practitioner in Northland, New Zealand, near the best recreational diving patch on the East Coast of the North Island. World class diving is readily accessed 14 miles off-shore at the nearby "Poor Knights Islands". Peter is an active diver and underwater photographer. He was the previous overall winner of NZ "Oceans" Intemational Underwater Photographic Competition, and has an ongoing interest in diving and hyperbaric medicine. Peter is the past Chairman of the New Zealand Chapter of SPUMS,

and past president of several diving clubs in NZ. He is currently president of the Northland Branch of the NZ Sports Medicine Federation and an examiner of commercial divers and sports divers. His address is Te Hape Road, RD 9 Maunu, Whangarei, New Zealand.

The above paper was presented at the Safe Limits Symposium held in Cairns, October 21st to 23rd 1994. It is reprinted by kind permission of the Division of Workplace Health and Safety of the Department of Employment, Vocational Education, Training and Industrial Relations of the Queensland Government, and of the author, from the symposium proceedings pages 108-112.

DIVE ACCIDENT MANAGEMENT IN CENTRAL NEW ZEALAND

Tony Hochberg

Preamble

This paper deals with the management of compressed air diving accidents in sport and commercial divers.[1,2]

Decompression illness (DCI) must be considered in the differential diagnosis of anyone becoming ill after a dive. The presentation of DCI may be obvious, delayed or unusual and present a diagnostic dilemma.

Advice and referral to a hyperbaric facility should be considered, even if there is a low probability of DCI, as frequently the severity is underestimated and the prevention of long term sequelae is of prime importance.

The key components of treatment are stabilisation, time to recompression and consideration of adjuvant therapy.

Assessment

The successful management of DCI involves effective communication between the referring agency (diver, dive shop, doctor, hospital etc.) and the physician trained in hyperbaric medicine.

Key questions in the assessment of any diver include the following:

1 Name and age of diver.
2 Status includes the level of consciousness, bleeding and visible injuries.
3 Dive profile, including any dives in the last 7 days, whether in fresh water or salt water, what dive tables or dive computer was used and the stage of the dive symptoms developed etc.
4 Symptoms.
5 Air travel and/or ascent to altitude over 300 m following the dive.
6 Relevant medical conditions, e.g. migraine, high blood pressure, asthma, epilepsy.
7 Medications.
8 Emergency treatment instituted.

Transportation

For referrals from a peripheral location, an intimate knowledge of the available emergency services and the level of skill of the operators is crucial to the successful evacuation of an acutely injured diver.

The modes of transportation for an injured diver include motor vehicle, train and air transport, either helicopter or fixed wing.

The first two modes of transportation are used if the probability of DCI is very low, the presentation is very late (with no serious complications such as neurological DCI that would necessitate the high cost of aeromedical evacuation) or there are adverse weather conditions.

By road the journey from Wellington to Devonport Naval Base, in Auckland. can be accomplished comfortably in 8 hours (a distance of 700 km).

Emergency air evacuation is not without its risks. Air ambulance standards are defined by the Civil Aviation Authority in an information circular Gen A dated July 1983.[3] Unfortunately deregulation of authorised air ambulances over the last few years has resulted in fatal aircraft accidents during aeromedical evacuation.

In New South Wales, Australia, Donaldson[4] provided some figures for comparison on helicopter aeromedical evacuation fatalities: a crude ambulance mortality rate of 3 deaths per 20,000 hours flown, which compares to a NSW road ambulance mortality of 0.015 deaths per 100,000 vehicle hours logged. In the USA the figure is under 6/ 100,000 hours for helicopter flights.

Contraindications to air travel in a diver include, shock, pneumothorax, myocardial infarction, vomiting and aerocele.

Equipment

Aeromedical equipment fits into two categories. The first is equipment integral to the aircraft e.g. stretcher, mountings, oxygen cylinders. The second is equipment carried onto the aircraft to accompany the diver.

The following are essential, intubation kit, chest drainage kit and medical kit. Table 1 lists a suitable medical kit.

Aircraft Type

The ideal aircraft is at least twin-engined, capable of being pressurised to sea level or greater, fast, long range, noise and vibration free with short take off and landing capability, large loading doors, adequate space, lighting and temperature and with toilet and hand washing facilities.

Helicopters can never meet all of these ideals and compromises therefore are made.

In Wellington there are two air ambulances dedicated to aeromedical work. These are a Piper Cheyenne II, a twin engined pressurised turbo-prop aircraft, and a BK-117 Messrschmitt Kawasaki twin turbine helicopter.

The Piper Cheyenne II (commissioned April 1994) is capable of being pressurised to sea level at an altitude of 10,000 feet. At an air speed of 230-240 knots the flight time from Wellington to Auckland International Airport is 70 to 75 minutes.

The Cheyenne is capable of carrying one stretcher patient or six seated patients in addition to two flight crew. It has a rate of climb of up to 4,000 ft/minute, which in turbulent weather is very important for patient comfort. Oxygen stores will typically last 3 hours at a flow rate of 10 litres/minute.

Standard equipment includes a Propak Life Monitor, Ivent ventilator, pulse oximeter, suction unit and a defibrillator with options for pacing when required. A unique stretcher bridge has been designed so that no equipment needs to be unplugged or transferred when moving the diver into or out of the aircraft.

The BK-117 Helicopter (commissioned April 1993) must be flown at an altitude not exceeding 300 m which is very fuel inefficient. At an airspeed of 120 Knots the journey from Wellington to Devonport Naval Base is two and a half hours. This includes two stops for fuel.

The BK-117 has the same equipment as the Piper Cheyenne with the added ability of being able to carry two stretcher patients or up to nine seated patients with the two pilots.

Cost

The Piper Cheyenne is billed, to Area Health Boards or the Accident Compensation Commission or the diver, at

TABLE 1
EQUIPMENT LIST
(TO BE CARRIED IN A CONVENIENT CASE).

Airway equipment
Guedel airways
Laryngoscope and selection of blades
Spare laryngoscope batteries and bulbs
Endotracheal tubes
Cricothyroidotomy set
Suction catheters - endotracheal and oral

Breathing support
Stethoscope
Self-inflating bag and range of masks
Oxygen supply, portable and aircraft-mounted

Circulatory support
Aneroid sphygmomanometer
Intravenous cannulae
Fluids and giving set
Intravenous cutdown set
Central venous catheter

Specific Items
Intercostal drain set with Heimlich valve
Cervical support collars
Urinary catheters
Nasogastric tube
Thermometer, non-mercury
Drugs, needles, and syringes

General items
Scissors
Sticking plaster
Dressings and eye pads
Crepe bandages
Torch
String, plastic bags
Alcohol swabs
Sterile gloves
Tissues
Vomit containers

Separate items of equipment to be kept with above
Suction apparatus
Portable ventilator
Monitor/defibrillator
Syringe pump
Pulse oximeter
Vacuum mattress
Traction leg splint
General limb splints
Pillows and blankets
Urinal and bedpan - where to empty?

An end tidal CO_2 monitor and oesophageal stethoscope are optional.

$850 per hour (actual running costs are $1,400 /hour). The typical cost of evacuation is $3,400.

The BK-117 is charged at $1,600 per hour (actual running costs are $2,200/hour). Typical cost of evacuation is $8,000.

Geography

The importance of geography in the management of diving accidents includes the location of hazardous dive sites, the location of hyperbaric facilities and the peculiarities of the terrain in between

Peculiarities of the Terrain

The central volcanic plateau of the North Island is composed of 3 active volcanoes, Mount Ruapehu at 2,797 m (9,175 ft), Mount Ngauruhoe at 2,291 m (7,515 ft) and Mount Tongariro at 1,968 m (6,458 ft). State Highway One runs alongside and peaks at an altitude of 1,600 m.

Divers travelling to Devonport by train or car must therefore avoid this route and go the longer and more complicated way through New Plymouth. Two divers have reported an aggravation of symptoms while passing through the central volcanic plateau which was alleviated by descent.

For fixed wing evacuation across the central volcanic plateau icing on wings can be a problem along with cloud with a particularly high ceiling. A coastal route is often favoured by pilots.

Hazardous Dive Sites

It is very clear that certain dive sites pose a greater risk than others. The Mikhail Lermontov which sank in 1986 in the Marlborough Sounds is a prime example with 3 recent dive related deaths and probably as many as 20 cases of DCI.

Hyperbaric Facilities

New Zealand had three Hyperbaric Chambers in operation 3 years ago (Auckland, Wellington and Christchurch) but now (October 1994) we have only one based at the Naval Hospital in Devonport, Auckland.

This has significant and potentially serious implications given that a large amount of scientific and recreational diving is undertaken in Wellington, Marlborough Sounds, Fiordland and Foveaux Strait. Some of these locations are over 1,575 km (900 miles) from Auckland.

With a population base of 3.25 million there is reasonable justification for another hospital based chamber.

Commercial recompression chambers can be found at Maui Oil Rig near New Plymouth and on The Little Mermaid in Wellington.

The importance of an understanding of geography to dive accident management is well summarised by the following case:

A 20 year old experienced diver (550+ dives) presented with a paralysed left arm following a provocative dive profile (41.4 m for 23 minutes, surface interval of 3 hours then 28.2 m for 30 minutes) combined with heavy alcohol consumption after the dive. His symptoms were of progressive weakness and numbness of the left arm. He was evacuated to Auckland International Airport in the Piper Cheyenne pressurised to sea level while on 100% oxygen. The estimated time of arrival at Auckland Airport coincided with peak rush hour traffic (1700). This would have added at least 2 hours to his arrival at Devonport by road ambulance. A helicopter was therefore standing by and there was only a further 15 minute delay. Six and a half hours elapsed between assessment and treatment.

Differential Diagnosis

In the assessment of any symptoms following a dive one must consider non-diving related illness.

Rarely a combination of diving related pathology will co-exist with non-diving related pathology eg acute appendicitis.

A short list to consider includes the following:

Hypoglycaemia
Migraine
Multiple sclerosis
Epilepsy
Gastro-oesophageal reflux
Aerophagy
Myocardial infarction
Porphyria
Ciguatera poisoning
Shellfish poisoning
Round window rupture
Alternobaric vertigo
CO intoxication
Salt water aspiration
Shallow water blackout
Viral illness

Management

The initial management of DCI can be summarised as follows:

FIND THE DIVER

Ancillary aids such as safety sausage, flares, torches, fluorescent dyes, whistles and radio transmitters all have their respective merits.

REMOVE FROM DANGEROUS ENVIRONMENT

At depth

If unconscious:

Purge regulator before placing it in the diver's mouth.
Gas should not be purged into an unconscious diver.
Surface with the victim's neck extended to enhance upper airway patency.

If conscious:

Controlled ascent where possible.

On surface

Unconscious or conscious:

Remove weight belt.
Inflate buoyancy compensating device (BCD) for positive buoyancy and ensure that the diver does not float face down.
Maintain an open airway.
Expired air resuscitation (EAR) only if immediate exit from the water is not possible.
In water external cardiac compression (ECC) is ineffective and should not be used.
Avoid obstacles such as kelp, rips and jagged rocks.

RESUSCITATION

EAR and ECC where necessary
Control bleeding and seek assistance

POSTURE

Horizontal for most divers
Left lateral position if unconscious or vomiting
30° head down for cerebral arterial gas embolism (CAGE). Conventional wisdom suggests that a short period, less than 10 minutes, of head down may result in enhanced clearance of bubbles from the cerebral circulation followed by a horizontal position.[5,6]

The value of posture is well demonstrated by the following case. A call was received from the skipper of a dive boat concerning a 28 year old untrained but experienced diver who was dizzy and vomiting after a provocative series of dives. The skipper was advised to place the diver horizontal and on the left side. This resulted in a marked resolution of symptoms. The eventual diagnosis was vestibular, cerebellar, cerebral and musculoskeletal DCI.

Divers with neurological symptoms must not be allowed to sit up until inside a recompression chamber.

The sitting position may be adopted for isolated inner ear barotrauma as this may reduce perilymph leakage.

100% OXYGEN

Device

Close fitting oronasal mask, or mouthpiece and nose clip, supplied by demand valve or oxygen reservoir bag.
Standard disposable medical oxygen masks or nasal prongs are unsuitable for treating diving accidents.

Stores

Calculate sufficient oxygen stores to retrieve at least two injured divers to a recompression facility at a flow rate of 20 litres/minute.

Air breaks

For the first 4 hours on oxygen no air breaks are required.
From 4-12 hours on oxygen 5 minutes on air every 25 minutes and after 24 hours on oxygen alternate hours air and oxygen.
Record and observe for symptoms of oxygen toxicity e.g. central chest pain, dyspnoea, hiccups, tinnitus, tunnel vision.
Calculate the risk of oxygen toxicity e.g. by units of pulmonary toxicity dose (UPTD).

FLUID BALANCE

Dehydration is known to aggravate DCI through influences on blood rheology and compromise of the microcirculation by haemoconcentration. Therefore liberal oral or intravenous fluids are encouraged and an accurate record should be kept. Colloid or crystalloid IV fluids may be used.

Oliguria or anuria despite liberal fluid administration may indicate bladder paralysis due to spinal cord DCI. Urinary catheterisation is mandatory in this setting.

Contraindications to aggressive fluid management are brain oedema secondary to brain injury, pulmonary oedema secondary to near drowning, valvular or ischaemic heart disease and renal failure secondary

to pre-existing renal disease eg IgA glomerulonephritis.

URINARY CATHERISATION

Essential in spinal cord DCI with bladder dysfunction.

HYPOTHERMIA

Hypothermia is classified as mild when the rectal temperature is between 35-32°C, moderate between 32-28°C and severe below 28°C.

Treatment modes include, passive e.g. space blanket, active e.g. heated blanket or warming in a bath and central e.g. warmed IV fluids, perhaps warmed peritoneal fluids, humidified oxygen at 40-42°C via an endotracheal tube.

ADJUVANT TREATMENT

There are no drugs of proven benefit in the treatment of DCI (this includes aspirin, corticosteroids and alcohol).

Lignocaine may hold promise for peripheral centres referring divers and warrants urgent consideration by researchers.

Diazepam is effective in controlling or preventing oxygen toxic convulsions and also in controlling vestibular symptoms. Diazepam should only be used to control vestibular symptoms on the advice of a Hyperbaric Physician as it may make titration of treatment in recompression chambers difficult.

RECOMPRESS AS SOON AS POSSIBLE.

Perhaps one should use a transportable chamber e.g. Duocom.

Air or road transport at an altitude of less than 300 m.

Care by trained personnel.

I see no role for in-water oxygen therapy.

At the moment you are aware that an injured diver is on his way to you for assessment you must begin the process of evaluating the availability of your emergency services and be prepared to meet the diver at the local airport, time permitting, or if they require stabilisation at the local hospital.

Not infrequently the BK-117 Helicopter is in Nelson and the Cheyenne is in Palmerston North and one must therefore calculate and co-ordinate with ambulance control priorities. For example one routine transfer of a ventilated paraplegic patient to Christchurch Spinal Unit was delayed, after discussion with the Intensive Care Consultant, to enable urgent transport to Devonport of a seriously ill diver.

Contact Numbers

New Zealand Diver Emergency Service	09-445-8454
Australia Diver Emergency Service	
within Australia	1-800 088 200
from outside Australia	61-8-373-5312
USA Diver Alert Network (DAN)	1-919-684-8111

Conclusion

Effective and well rehearsed communication between the referring agency, the hyperbaric physician or doctor in a peripheral centre and the specialist hyperbaric unit are crucial to successful management of an ill diver.

Time is of the essence.

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