The Editor's Offering

The South Pacific Underwater Medicine Society Journal, we must get accustomed to using the full title so that our Society becomes better known, wishes the infant European Journal of Underwater and Hyperbaric Medicine (EJUHM) a long and fruitful life after its transformation from the EUBS (European Underwater and Baromedical Society) Newsletter. Dr Peter Mueller, the Editor of both EUBS Journals, who is a member of SPUMS, sorry, the South Pacific Underwater Medicine Society, has modelled the new Journal on our Journal, which is high praise for the Society. Members will be pleased to know that the Instructions to Authors in EJUHM are almost exactly the same as those for the SPUMS Journal, which will help those who wish to submit papers to both journals.

It is not a typographical error that there are two book reviews of Reverse Dive Profiles listed on the cover. The review of the Reverse Dive Profiles Workshop by Hamilton and Baker, which appeared in Pressure and was reprinted in the last issue, led to the Journal obtaining two review copies. Book reviews are supposed to be done by those with special knowledge so the Editor approached such people. When one of these flatteringly suggested that the Editor should provide a review the bait was swallowed as the Editor was already half way through the book and the reviewer gets to keep the book.

Much of this issue is devoted to the debate conducted in Layang Layang about treatment of decompression illness (DCI) in an attempt to find a treatment regime, or regimes, suitable for DCI of differing onset and symptomatology . The two guest speakers, Drs Alf Brubakk and Richard Moon, presented the cases for extremes of acceptable practice in the treatment of DCI. Richard Moon presented the "Doctor knows best" case, treatment in hospital for everyone with symptoms of DCI regardless of the delays to treatment brought on by distance and evacuation. Alf Brubakk presented the "Treat as soon as possible because the best results come with treatment within 30 minutes" case, which is well supported by the experience of Navies and the commercial oil diving companies. The opening presentations covering the pathophysiology, natural history of untreated DCI, the ideal treatment dose of oxygen and adjunctive therapy have appeared in previous Journals.[1-4]

In this issue we wrap up the discussion (pages 149-179). **The comments that follow here are the Editor's personal opinion and should be interpreted as such and not as official SPUMS thoughts.** Many people are evacuated from popular diving areas, often for long distances, to a chamber for treatment, arriving many hours after the onset of symptoms. The Diver Alert Network (DAN), which insures divers, spends a large amount of money evacuating divers every year. Even when the divers reach hospital, anecdotal evidence has it that the journey from ambulance to being at pressure in the chamber often takes hours. There is a prevalent myth that only doctors can recognise DCI. There are no doctors on oil rigs with the divers, but there are DMTs (diving medical technicians) who are lay people trained to recognise DCI, and, equally importantly, taught how to treat DCI. Figures about the incidence of DCI in recreational divers are inaccurate because the denominator is unknown and an unknown number of sufferers do not present at a chamber. But whatever the accuracy the figure is known to be somewhere between 1 in 1,000 and 1 in 100,000, which in the Editor's opinion is low. It is known that technical divers, defined as those who change their breathing gas during a dive, often use oxygen for some of their decompression and anecdotally treat themselves with in-water oxygen if they develop symptoms while at sea. Some are unsuccessful and end up in a chamber, but others are successful and do not get recorded in the statistics. One of the confusing factors comparing naval and oil company statistics, which are often commercial secrets so not available, with recreational statistics is that in the first group there is a disciplined approach to the management of DCI while recreational divers have no supervisor to force them rapidly into a chamber. There many obstacles to changing recreational DCI treatment, not least the American liking for suing everyone involved when outcomes are less than perfect. It is clear that few people will be willing to risk bankruptcy by treating DCI cases in less than "ideal circumstances" which would be presented by the plaintiff's lawyers as a chamber, fitted for treating intensive care cases, in tertiary hospital. Even though there is evidence that rapid treatment is highly effective most doctors in hyperbaric facilities are against on-site treatment with less than a multi-lock, multi-place steel chamber. It is quite possible that better education, both in diving practice and DCI symptom recognition would have a larger effect on the incidence of DCI than providing earlier treatment for stricken divers. As the London paper-boys used to say in the 1950s "Read All About It".

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ORIGINAL PAPERS

EVIDENCE-BASED MEDICINE AND HYPERBARIC PRACTICE

Mike Bennett

Key Words
Evidence, hyperbaric oxygen, treatment.

Introduction

Evidence-based medicine (EBM) has been defined as "the conscientious, explicit, and judicious use of the current best evidence in making decisions about the care of individual patients".[1] Despite recent enthusiasm expressed for the concept by many health care professionals, there has been a degree of criticism. There are those who feel the reference to evidence erodes clinical freedom and is designed by bean-counters to control medical expenditure. There are fears that EBM is "cookbook" medicine, requiring all individuals to receive the same diagnostic and therapeutic measures, regardless of individual needs. This is a grave misunderstanding. EBM requires the synthesis of best evidence and clinical expertise/experience in order to arrive at the best diagnostic and therapeutic approaches for each individual. Medical practitioners should see EBM as empowering and I hope this article will convey some of the sense of clinical enrichment.

The practice of EBM cannot spring into existence without effort. We need to train ourselves to ask appropriate questions, execute efficient searching techniques (in order to discover evidence and be sure we have the best), develop skills at critical appraisal of this evidence, grasp some basic clinical statistical methods (OH NO! Perhaps we should call this "rules of evidence") and relate our findings to individual patients. This paper is designed to introduce the concepts central to the practice of EBM and to use examples to show their relevance to hyperbaric practice. An excellent review of what constitutes EBM and why it is relevant to all of us was published in the Journal of the American Medical Association in 1992,[2] while another major resource of practical benefit is a pocket guide to teaching and practice of EBM by Sackett and others.[3] There are also a number of internet resources available. A short list of these appears in Table 1.

Asking good questions

The process of EBM begins with the identification of a clinical (or diagnostic, prognostic etc.) problem for which a practitioner feels there is no clearly defined and validated answer. From this realisation, often arrived at in the course of patient care, the practitioner must accurately define the problem before taking steps to discover an answer. One approach is to begin by asking structured clinical questions.

Clinical questioning is an important skill in itself. Sackett has defined a schema for building an "evidence-based" question, that is, one to which a focussed search is most easily applied. Bennett has discussed the application of this approach to facilitate critical appraisal within an anaesthetic journal club.[3,4] There are four major elements to such questions, all of which need careful consideration in order that the clinical problem, alternative therapies and outcomes of interest are clear to the searcher. Once a sufficiently focussed question is designed, it becomes much clearer to the searcher which citations represent possible

TABLE 1

SOME EBM RESOURCE SITES ON THE INTERNET

	Resource type		Address
1	Searching	PubMed gateway	http://www.ncbi.nlm.nih.gov/entrez/query.fcgi
		Ovid gateway	http://medline.unsw.edu/ovidweb/login.htm
		DORCTIHM *	http://sesinfo/powweb/hyperbar.htm
		Cochrane	http://som.flinders.edu.au/fusa/cochrane/default.html
2	Critical appraisal	JAMA	http://www.acponline.org/journals/acpjc/
3	Rules of evidence	McMaster University	http://hiru.hirunet.mcmaster.ca/ebm/default.htm
		Stats gateway	http://uni.koeln.de/themen/Statistik/onlinebooks.html
4	General EBM	Oxford Centre for EBM	http://cebm.jr2.ox.ac.uk/
		Netting the evidence	http://www.shef.ac.uk/uni/academic/scharr/ir/netting.html

*Database of Randomised Controlled Trials in Hyperbaric Medicine- not yet active at time of writing (April 2000).

answers to the question and which are distractions from the clinical problem. An example question is worked through in Table 2, beginning, perhaps, from a discussion in any hyperbaric unit about whether HBO_2T works for carbon monoxide poisoning.

on internal and external validity. The most appropriate methodology will depend on the type of question asked. Most of the discussion which follows is primarily aimed at questions concerning a therapeutic intervention (does HBO_2T work for…?). Different methodologies are more

TABLE 2

BUILDING AN EVIDENCE-BASED QUESTION
(modified from Sackett et al.[3])

	1 Patient problem	**2 The intervention of interest (or cause/ prognostic factor etc)**	**3 Compared to…. (not always required)**	**4 Outcomes**
Tips	Need to define the patient of most interest	Be exact about the intervention	Often simply the main alternative	Focus on important outcomes of interest that seem relevant to the intervention
Example	"In adult patients with moderate to severe carbon monoxide poisoning…	...does the administration of hyperbaric oxygen (>1.5ATA for at least 1hr)	…compared to a regimen of normobaric oxygen for at least 2 hours…	...result in any demonstrable reduction in neurological or cardiovascular mortality or morbidity?

Searching for evidence

Once a question has been designed to the satisfaction of those interested in the outcome, the next step is an attempt to discover the evidence. It is important to develop a structured and practised approach to seeking evidence. While there is much scope for different approaches from individual searchers, there are some important elements that should not be overlooked. Table 3 (p 123) shows one search strategy that might be suitable in attempting to find evidence concerning an indication for HBO_2T. It is a modification of the protocol suggested by Andrew Booth from the School of Health and Related Research and available from the *Netting the Evidence* web site. [5]

Critical appraisal

Once a clinical problem has been defined and an efficient search conducted, the next requirement is for a means to determine which evidence is likely to be most reliable. Critical appraisal is the term given to the process of selecting the best articles of those retrieved and applying the rules of evidence to determine their applicability to an individual clinical situation.

Table 4 (page 125) is a methodological hierarchy suggested by the author. While there are many such schemes available from a variety of sources, most are very similar as there is broad agreement about the effect of methodology

appropriate for questions of diagnostic test evaluation (what does the $PtcO_2$ mean…?) or the definition of the magnitude of a health problem (how common are diabetic ulcers?, for example). For a detailed discussion of the role of trial design in the minimisation of bias in clinical trials, see Sackett et al.[3]

In general, the best available evidence of therapeutic efficacy is to be found through well conducted, large, multi-centre randomised controlled trials (RCTs) or meta-analysis of a number of smaller RCTs. The randomised and blinded trials so familiar to us now remain the only sure way of eliminating systematic bias from clinical inquiry. They do not, of course, eliminate the chance variations that may mislead us. Avoiding misinterpretation of random events as clinically meaningful is the purpose of statistical analysis and appropriate empowerment of well-designed trials.

Our search having identified a number of relevant articles, and the basic methodology of each identified, the most promising should be selected for further review. Each remaining article needs to be examined in more detail to identify any serious threats to internal or external validity. [Internal validity: are there any flaws in construction or execution of this trial that reduce the confidence we have in the results? External validity: are there elements in the patients studied or the trial execution that reduce our confidence that the results apply to our patient(s)?]. This can be a complex process and at the Prince of Wales, we have developed a critical appraisal sheet (Table 5 page 125)

TABLE 3

SEEKING THE EVIDENCE

ONE POSSIBLE PROTOCOL FOR DIVING AND HYBERBARIC MEDICINE

Step 1 MEDLINE Search

MEDLINE is still the best starting point for EBM queries in general. For therapy questions, however, the Cochrane Library has edged ahead as it now contains more controlled trials than MEDLINE.

There are two alternative methods of filtering the evidence from MEDLINE:

1 Conduct a search using two or three terms relevant to the question and then limit the retrieval set to **Review* in PT** (for reviews); **Clinical-Trial in PT** (for clinical trials); **/economics** subheading or **explode costs-and-cost-analysis** (for economic studies); **explode attitudes (**for patient, staff or carer perspectives). (**PT** is publication type)

2 Use the PubMed version of MEDLINE (the **Clinical queries** interface). Select the type of question that you require (e.g. diagnosis or therapy). Then indicate whether you wish to cast the methodological net wide (sensitivity) or to have a narrow focus (specificity).

If you retrieve little in the way of high quality evidence choose the most relevant looking reference and select **"See Related Articles" PubMed.**

Do not forget EMBASE, particularly for European Literature or articles on pharmaceuticals and CINAHL for the nursing literature and Consensus statements.

Step 2 Cochrane Library

This library gives access to all completed and proposed meta-analyses in a growing range of medical specialties. There are reviews of carbon monoxide poisoning and multiple sclerosis, for example. Perhaps even more useful, there is a searchable list of controlled trials and the Database of Abstracts of Reviews of Effectiveness [DARE], all searchable on the World Wide Web.

Step 3 Database of Randomised Controlled Trials in Hyperbaric Medicine (DORCTIHM)

This specifically diving and hyperbaric database is searchable and each trial included is summarised on a single page using the Critically Appraised Topic (CAT) software designed by Douglas Badenock in Oxford. The database is available from the authors and will soon be on the POWH departmental web site .[6]

Step 4 UHMS Committee Report

This regular publication appraises the evidence for the use of HBO_2T across a broad range of indications. It is becoming increasingly evidence-based rather than anecdotal.[7]

Step 5 Direct search of on-line or hard copy specialist journals

The key specialist journal, Undersea and Hyperbaric Medicine, is not available on-line, and so requires hand searching. The South Pacific Underwater Medicine Society (SPUMS) Journal has an on-line and downloadable index to over 2,400 articles published back to 1971 and can be found at: <http://www.spums.org.au/spums_journal_articles_database_.htm .>

Step 6 Pearling

This term refers to the practice of trawling the references of previously located articles for further relevant material.

TABLE 4

DESIGNATION OF LEVELS OF EVIDENCE

Evidence level	Description
I	Evidence obtained from a systematic review of all relevant randomised controlled trials or a single, well-designed, large, multi-centre randomised controlled trial.
II	Evidence obtained from at least one properly designed randomised controlled trial.
III-1	Evidence obtained from well-designed pseudo-randomised controlled trials (alternate allocation or some other method).
III-2	Evidence obtained from comparative studies with concurrent controls and allocation not randomised (cohort studies), case-control studies or interrupted time series with control group.
III-3	Evidence obtained from comparative studies with historical control, two or more single-arm studies or interrupted time series without a parallel contol group.
IV	Evidence obtained from case series, either post-treatment or pre- and post-treatment.
V	Evidence obtained from a single case report.
VI	Evidence based on expert opinion or qualitative review

to ensure we always examine the most important aspects of each paper.

One increasingly popular method of summarising the critical appraisal of an article is the use of the CATmaker software developed by Douglas Badenoch in Oxford.[8] Using this simple program, a one-page summary of the article is presented with a concise presentation of the important clinical findings. This summary constitutes a Critically Appraised Topic (CAT) and an example appears in Table 6 (page 127-128). With a little practice, these summaries can be produced in about 15 to 20 minutes. Once completed, such CATs can be reviewed when required in the light of new evidence. The Oxford Centre for Evidence-Based Medicine web site maintains a collection of these CATs in a 'CATbank'.[8]

More specifically, the Prince of Wales Hospital Hyperbaric Unit has developed a database as described above (DORCTHIM). In this searchable database, all trials are accompanied by a CAT. Any contributions to this collection are welcome.

Basic statistics or "rules of evidence"

Biostatistics are daunting for most clinicians. While we do not all have to achieve a detailed understanding of the subtleties of such mathematical gymnastics, it is not possible to take advantage of the evidence available without some general appreciation of basic statistical concepts. Trisha Greenhalgh has written a well-constructed summary in her two papers in the *How to read a paper* series in the BMJ in 1997.[9,10] This summary is designed specifically for those who feel totally at sea with statistical concepts. For those with a little more experience, she recommends the *Basic Statistics for Clinicians* series (4 papers) in the Canadian Medical Association Journal.[11]

Clinicians are most often interested in the impact on their patients of a proposed intervention. Three methods of measuring the effectiveness of interventions are in common use by EBM practitioners. Referring to the results of a 1996 study by Bouachour[12] on the treatment of crush injuries with HBO_2, Table 7 (p 128) shows three outcome columns:

1 Relative risk reduction (RRR).

The reduction in the incidence of an outcome relative to the incidence in the control group. This gives the reader a sense of the proportion of those who would have suffered an outcome, but will not now because of the new intervention. In this example, we estimate that 86% of those who suffer the outcome of failed wound healing would not have done if HBO_2T had been used. This is important, but without an estimate of absolute risk reduction (or increase), the total impact of the intervention cannot be gauged.

2 Absolute risk reduction (ARR).

The difference between the incidence of an outcome in the two groups. This gives the reader a direct sense of the absolute improvement likely. Here, the absolute increase in the risk of failed healing without HBO_2T is estimated at 38%, that is there will be 38% more cases of failed wound healing without HBO_2T. On its own, this information may not be useful, however. The importance of a 38% risk reduction may be very different if the incidence in the control group is 100% as opposed to the actual rate of 44.4%. In this example, the problem is all but eliminated by the institution of HBO_2T.

3 Number needed to treat (NNT).

The NNT is the reciprocal of the RRR. It is an estimate of the number of individuals who need to be treated with HBO_2T before one more person will achieve a good outcome. In this example, we only need to treat three cases of crush injury before we avoid a non-healing wound in one

TABLE 5

Prince of Wales Hospital Critical Appraisal Sheet

Important information that should be in the paper	**Potential related problems**	**Threats to the internal and external validity of the study**
	1.2 Is this question relevant to the clinical problem?	
2.1 What is the study type?	**2.2** Is the study type appropriate to the research question?	**2.3** If not, how useful are the results likely to be?
3.1a Define the population in which the authors nal are interested. Are the study subjects representative of this population?	**3.2a** Are there selection biases?	**3.3a** Any threat to external validity? Any threat to inter- validity?
3.1b If assigned to groups, how was this accomplished?	**3.2b** Was allocation random? Was allocation made after a decision to enter the trial?	**3.3b** Any threat to internal validity?
3.1c How many reached final follow-up?		**3.3c** Does this proportion threaten internal validity?
4.1 What is being studied (study factor)? How is it measured?	**4.2** Is there any likely measurement error (differential or non-differential)?	**4.3** Is there any likely important cause of bias? (Beware differential error with Case-Control Studies).
5.1 What outcomes are being assessed (outcome factors)?	**5.2** Any important outcomes missed? Any likely measurement error (differential or non-differential)?	**5.3** Do missed outcomes reduce the applicability of this study? Is there any likely source of bias?
6.1a What potential confounders are considered?	**6.2a** Any important confounders missing?	**6.3** How likely is confounding to be a significant source of bias?
6.1b How were they dealt with?	**6.2b** Were they dealt with adequately, or subject to measurement error?	
7.1a Is a point estimate of effect given?	**7.2a** Is it reasonable to accept these results are not due to chance?	**7.3** Is this study useful or inconclusive in answering the research question?
7.1b Are confidence intervals given? If not in a study with statistically non-significant findings, is power given?	**7.2b** Are the differences reported clinically significant? Was the sample size sufficient to detect a clinically significant difference?	
8.1 What are the authors conclusions?	**8.2** Have the authors correctly interpreted the results?	**8.3** Have the authors considered study limitations in their conclusions?

TABLE 6
EXAMPLE CAT (Critically Appraised Topic)

Hyperbaric oxygen did not reduce the number of patients with persistent deficit following carbon monoxide poisoning and was associated with a higher rate of delayed neurological sequelae.

Clinical Bottom Line

1 There was no benefit evident for hyperbaric oxygen in the prevention of persistent neurologic abnormality.
2 There were significantly fewer patients with delayed neurologic abnormality in the normobaric group.

Appraised by Mike Bennett, Department of Diving and Hyperbaric Medicine, Prince of Wales Hospital, Sydney; Monday, 1 March 1999.

Clinical Scenario. A patient presented with acute carbon monoxide intoxication and we wondered if there was any demonstrable benefit in the administration of hyperbaric oxygen.

Three-part question. In patients with carbon monoxide poisoning, does the administration of hyperbaric oxygen, compared to normobaric oxygen, result in any improvement in the acute neurological state or the avoidance of late neurological deterioration?

Search Terms. Hyperbaric oxygenation, carbon monoxide

The Study. Double-blinded concealed randomised controlled trial with intention-to-treat.

Patients referred to a hyperbaric facility for the treatment of carbon monoxide poisoning- all grades of severity.

Control group (N = 87; 87 analysed): Normobaric oxygen at 1ATA for 72 hour with three periods of sham hyperbaric oxygen. Those with persistent symptoms or signs received three further daily sham treatments and a further 72 hours on oxygen.

Experimental group (N = 104; 104 analysed): Daily hyperbaric oxygen at 2.8 ATA for 60 minutes (total chamber time 100 minutes) for three days with normobaric oxygen between treatments. Treatment repeated for another three days if symptoms or signs persisted.

THE EVIDENCE

Outcome	Time to Outcome	Normobaric group	HBO group	Relative risk reduction	Absolute risk reduction	Number needed to treat
Persistent neurological sequelae	Discharge	0.68	0.74	-9%	-0.060	-17
95% CI:				-28% to 10%	-0.189 to 0.069	14 to INF 5 to INF
Delayed neurological sequelae	Unknown	0	0.048	INF	-0.048	-21
95% CI:					-0.089 to -0.007	-145 to -11
Complications of treatment	Discharge	0.01	0.09	-800%	-0.08	-13
95% CI:				-100% to -212%	-0.139 to -0.021	-47 to -7

Non-event outcomes	Time to outcome	Normobaric group	HBO group	P-value
Average number of neuropsychiatric tests abnormal	Discharge	2.7	3.4	0.02

Comments

1 Oxygen doses high in comparison to those generally administered.
2 Cluster randomisation accounted for differences in the final numbers and may introduce some bias.
3 Average delay to treatment was over 7 hours.
4 Minimal improvement in mini-mental state assessment before and after treatment in either group is puzzling.
5 No functional outcome other than mortality.
6 Follow-up at one month only 46%.

Expiry date. March 2000

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TABLE 7

RESULTS OF HBO_2T FOR CRUSH INJURIES
(from Bouachour et al.[12])

Outcome outcome	Time to group	Air group	HBO	RRR	ARR	NNR
Wound not healed	60 days	0.444	0.06	86%	0.384	3
95% CI:			29% to 100%	0.130 to 0.638	2 to 8	
Repeat surgical procedure	60 days	0.333	0.06	82%	0.273	4
95% CI:				9% to 100%	0.029 to 0.517	2 to 34

RRR = Relative risk reduction ARR = Absolute risk reduction NNT = Number needed to treat

person. Many clinicians find the NNT of most relevance when trying to assess the direct clinical impact of a therapy on their patients.

We might conclude, therefore, that the addition of HBO_2T in the treatment of lower limb crush injuries is justified by the impressive reduction in the incidence of non-healing wounds (86% reduction). We can expect to eliminate 38% of non-healing wounds following such injuries and this means we prevent one non-healing wound for every three patients we treat with HBOT.

Implementation of the conclusions

Without a doubt, implementation is the most difficult aspect in the practice of EBM. Appropriate strategies will vary with the individual situation, however it can be difficult to engage colleagues who have not participated in the process outlined above. It is our anecdotal experience that successful strategies arise from active participation by a significant proportion of clinicians. This is often relatively easy to achieve in a small area like a hyperbaric service. It has proved far more difficult in a large practice, such as a busy anaesthetic service, where it is difficult to marshal the majority of the faculty into one meeting.

There is no doubt that the pursuit of EBM is an active one. Colleagues will be engaged with the process when their own clinical questions are under discussion. At the Prince of Wales Hospital, we find it works best in a formal meeting, held regularly, with clinical problems working their way through the system described above, over a series of meetings. A suggested clinical problem will be worked into a formal question in one meeting, the search in answer to that question at the next, the critical appraisal of the chosen reference at the next and finally the CAT reviewed at the next. At each meeting, several different topics will be under discussion in order to maintain interest.

This process is outlined in more detail by both Sackett and Bennett.[3,4]

The most appropriate outcome is of course, better practice with improved outcomes for patients. The process described here is not foolproof and does not guarantee best practice. Each finding will require careful synthesis by the clinician into the overall situation of the individual patient. EBM provides systematic advice on existing evidence, only the clinician can actually treat the patient.

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A POSSIBLE CASE OF CEREBRAL ARTERIAL GAS EMBOLISM IN A BREATH-HOLD DIVER

David Williams

Key Words

Breath-hold diving, case report, decompression illness, cerebral arterial gas embolism

Introduction

Cerebral arterial gas embolism (CAGE) is second only to drowning as the most common cause of death in recreational SCUBA divers;[1] however, it is extremely rare in breath-hold divers unexposed to a compressed air source. The history of a possible case of CAGE in a previously healthy breath-hold diver is described here; and the differential diagnoses are discussed.

Clinical history

A fifteen year old male, from Munda in the Solomon Islands, made frequent repetitive breath-hold dives over a period of three and a half hours to spear fish. His maximum depth was approximately 8 m. On surfacing from his last dive, he developed a sudden severe headache, dizziness, blurred vision, and numbness and weakness of all four limbs. He was unable to stand or walk and had to be carried from the water by his father.

The symptoms persisted, and he was admitted to the Helena Goldie Hospital, Munda, the following day. He had no previous history of medical problems (specifically, no history of pulmonary or neurological illness), and had been completely well prior to and during his breath-hold dives. There was no history of exposure to a compressed air source, and he was the only person in the water at the time that the

incident occurred. There was no history of marine envenomation.

On examination, the patient had a well built muscular physique. He was apyrexial with a respiratory rate of 20, heart rate 64 bpm and blood pressure of 100/70 mmHg. There were no rigors, neck stiffness or depression of conscious level. There were no skin lesions, rash, or signs of envenomation. Hearing was normal, but there was vertigo and blurred vision bilaterally. Visual fields were grossly normal, and there was no evidence of nystagmus. There were no clinical signs of pulmonary barotrauma (pneumothorax, pneumopericardium or subcutaneous emphysema). There was movement of all four limbs, but with profound weakness and numbness.

X-Ray equipment was available, but its use was limited due to practical and financial considerations. In view of these restrictions and the absence of clinical evidence of pneumothorax, a chest X-Ray was not performed.

Following discussion between the doctors at the base hospital and the Divers Emergency Service in Adelaide, the findings were consistent with a diagnosis of cerebral arterial gas embolism (CAGE). For financial and logistical reasons, it was impossible to arrange retrieval from the remote location to a hospital with a hyperbaric facility. He was, therefore, kept supine with continuous surface oxygen via a Hudson mask at a flow rate of 4 l/min, and given 3 litres of 0.9% saline intravenously over 8 hours. Due to the lack of adequate cardiac monitoring, it was not possible to administer an intravenous lignocaine infusion.

The headache, blurred vision and neurological changes resolved progressively and completely within 24 hours of commencing surface oxygen therapy. A blood film showed the presence of falciparum malaria, which was treated with a 2 day course of quinidine and Fansidar (sulfadoxine/pyrimethamine). There were no sequelae, and the patient made a full recovery.

Discussion

The presentation of a catastrophic neurological event of sudden onset and equally sudden resolution in a previously healthy young male breath-hold diver is highly unusual. The differential diagnoses to consider in this case were: cerebral malaria, unrecognised marine envenomation, decompression sickness (DCS), cerebral arterial gas embolism (CAGE) and some other unrecognised neurological or psychiatric illness.

CEREBRAL MALARIA

The finding of malarial parasites on the patient's blood film is not remarkable as chronic infection with *Plasmodium falciparum* is endemic in the population of the Solomon islands, with a prevalence of around 30%. Cerebral malaria is associated with several days of non-specific feverish symptoms, followed by impairment of consciousness, generalised convulsions, and coma which persists for 24 to 72 hours.[2] Neurological examination usually reveals symmetrical upper neuron dysfunction with generalised extensor spasms and decorticate or decerebrate rigidity or opisthotonus. Supportive management and administration of intravenous quinine dihydrochloride or quinidine gluconate are required. The clinical presentation of our subject and rapid recovery with surface oxygen would suggest that cerebral malaria was not the cause of the symptoms in this case.

ENVENOMATION

Venomous species found in the vicinity of the Solomon Islands include sea snakes, cone shells, stone fish, blue ringed octopus and jellyfish (including *Chironex* and *Irukandji* species). Although muscle weakness and peripheral numbness may be a feature of envenomation by these organisms; they all cause skin lesions which may be seen on careful examination, and envenomation by cone shells, stonefish, scorpion fish and *Chironex* is associated with severe pain. Painless envenomation may occur from sea snakes, blue ringed octopus, or *Irukandji*; however, autonomic nervous system involvement is a prominent feature of both blue ringed octopus and *Irukandji* envenomation, and the myotoxins in sea snake venom cause characteristic myalgia and myoglobinuria.[3]

A number of cases of severe prolonged neurological deficit of sudden onset ("sea stroke") have recently been described in divers off the coast of North Carolina, USA , and are thought to be due to brainstem infarction as the result of envenomation by an as-yet-unknown marine organism.[4]

None of the above explain the course of the symptoms observed in our subject.

DCS AND CAGE

The presentation of a severe neurological deficit in a previously healthy subject which is related to diving, is of sudden onset, and resolves on treatment with oxygen, is strongly suggestive of decompression sickness (DCS) or cerebral arterial gas embolism (CAGE). Typically, the neurological deficit associated with CAGE is profound, resembling an embolic stroke, occurs immediately on surfacing and is often associated with loss of consciousness. The neurological symptoms of DCS are less severe, with a latent period of several minutes to 48 hours after surfacing, and with gradual progressive onset of pain or paraesthesia. DCS frequently also involves joints, skin

and lungs. CAGE may resolve after several hours, due to spontaneous passage of bubbles through the cerebral circulation, and tends to improve or resolve completely with surface oxygen. DCS is more persistent, migratory, and has variable and poor response to surface oxygen alone. The clinical course in this case was more consistent with CAGE than DCS; however it may be difficult to distinguish clinically between CAGE and DCS and the two pathologies may co-exist. However the management is similar, being oxygen and early recompression. Consequently, a unified diagnosis of acute neurological decompression illness (DCI) has been proposed.[5]

DCS IN BREATH-HOLD DIVERS

The occurrence of DCS in repetitive breath hold divers unexposed to a compressed gas source has been well documented.[6-11] The clinical manifestations range from intellectual impairment to vertigo and nausea, paralysis, unconsciousness and death.[6]

Nitrogen becomes dissolved in tissues as a function of time and depth as dictated by Dalton's Law of partial pressures and Henry's Law of gas solubility. Rapid ascent results in bubble nucleation and growth in the tissues and vessels. On a single breath-hold dive, it is virtually impossible to acquire sufficient tissue nitrogen loading to cause significant bubble formation on ascent. However, deep repetitive breath-hold dives with insufficient surface interval between dives may result in cumulative nitrogen loading of tissues, with consequent DCS.[8,12]

No-decompression limit tables have been calculated for repetitive breath-hold divers which predict that repeated breath-hold dives to a depth of 66 feet (20 m) can be made safely, provided that the surface interval is greater than or equal to the bottom time for each dive.[13] However, surface intervals of half the bottom time result in a high risk of DCS after only 2 hours of repetitive breath-hold diving.

Although the maximum depth and total duration of the series of dives was known in the case described above, information about the duration of individual dives and surface intervals was unfortunately not available. With a history of repetitive dives over a 3 hour period, it is possible that the subject had acquired a significant inert gas load. However, his maximum depth was only 8 m which makes it difficult to explain the severity and rapidity of onset of symptoms on the grounds of venous and tissue bubble formation alone.

PATHOPHYSIOLOGY OF CAGE

If compressed gas is breathed at depth and the diver ascends, the gas in the lungs will expand by Boyle's law as the ambient pressure falls. If it is unable to escape due to breath holding or bronchospasm, the increase in volume and pressure as the gas expands may cause the lungs to rupture. Gas can then escape into the pleural cavity, causing pneumothorax; into the mediastinum and soft tissues of the neck, causing surgical emphysema; into the pericardial cavity, causing pneumopericardium; and into the pulmonary arterioles, causing arterial gas embolism. The term pulmonary over-pressurisation syndrome (POPS) has been used to describe this sequence of pulmonary over-inflation, rupture, and escape of gas into extra-alveolar locations.[1]

It is not known whether disruption of the alveolar membrane occurs due to barotrauma, volutrauma (shearing forces between adjacent tissues of heterogeneous compliance), or a combination of both.[14,15] It has been suggested that the alveoli rupture when the transthoracic pressure gradient exceeds 10 kPa;[16] however studies on human cadavers have demonstrated a " bursting threshold " of as little as 73 mmHg (9.7 kPa).[17] It is believed that mechanical disruption of the alveolar-arterial barrier allows pressurised gas to enter the arterial circulation; however, this does not explain the observation that CAGE and pneumothorax are found together in less than 5% of divers.[18,19]

CAGE may also occur when bubbles of venous or tissue origin enter the arterial circulation via a right-to-left shunt due to an atrial or ventricular septal defect, patent foramen ovale or pulmonary arteriovenous fistula. The term "paradoxical embolism", which has been used to describe this mechanism, is a misnomer as the mechanisms involved are exactly what would be predicted from a basic understanding of the physiological principles involved.[20]

Approximately 25% of the population have a "probe-patent" foramen ovale which is functionally closed under normal conditions, but may open if right atrial pressure is raised, permitting right-to-left shunting to occur, with arterialisation of venous emboli and subsequent CAGE.[15] This may occur in divers on release of a forced Valsalva manoeuvre when clearing the ears, or straining to lift a heavy object,[21] or due to hydrostatic pressure on the thorax when a diver is partially immersed in the vertical position.[22]

No heart murmurs suggestive of a gross right-to-left shunt were found in our subject. However it is possible that he may have had a small shunt or probe-patent foramen ovale, which could only be diagnosed with the aid of echocardiography, and could have led to arterial embolisation of bubbles of venous or tissue origin.

CAGE IN BREATH-HOLD DIVERS

CAGE and POPS may occur in breath-hold divers who perform a breath hold ascent after taking a breath from

a SCUBA diver's alternative air source at depth.[23] However they may also occur in breath-hold divers who have not been exposed to a compressed air source.[9,24,25]

The mechanism is uncertain, as in theory, the diver should return to the surface with no more air in the lungs than when the dive began. It is hypothesised that local air trapping may occur during the dive with subsequent expansion and pulmonary rupture on ascent.[26,27] Healthy lungs are close to their elastic limit when held at total lung capacity (TLC) and it has been suggested that lung rupture may actually occur before the dive due to the shearing forces caused by maximal inhalation.[28] It is also possible that the displacement of blood into the thorax on submersion causes a reduction in compliance which makes the lung more likely to rupture. Diseased lungs are probably more prone to rupture, as they may have poorly communicating gas-filled spaces, weakened areas due to subpleural bullae or blebs, or areas of heterogeneous compliance due to global or focal fibrosis, which may generate and focus shearing forces.[28]

A number of techniques employed to extend the depth or duration of breath-hold dives result in increased intrathoracic pressure, which may predispose to POPS, CAGE, "paradoxical" embolism and syncope due to decreased venous return to the heart.

The increasing ambient pressure on descent causes blood to be redistributed from the periphery to the intrathoracic vascular bed, reducing vital capacity (VC) and limiting maximum diving depth. To prevent this, breath hold divers often voluntarily increase intrathoracic pressure by performing a maximal inspiration to TLC prior to descent. They may then further increase intrathoracic pressure by a technique known as "buccal pumping" or "lung packing" in which additional mouthfuls of air are forced into the lung by swallowing whilst maintaining an open glottis. This technique may increase the VC by up to 39% by forcing intrathoracic blood into the periphery; however the technique is extremely dangerous, resulting in a dramatic rise in airway pressures of up to 5.72 kPa.[29]

To prolong the duration of the dive, some divers swallow repeatedly or perform a Valsalva manoeuvre while at depth which can delay the conventional breaking point by modifying chest wall mechanoreceptor activity. However this may also result in a potentially dangerous increase in intrathoracic pressure.

Our subject had no history of pre-existing lung disease and did not report the use of pre-dive maximal inspiration, lung-packing, or Valsalva manoeuvre at depth during his series of dives.

OTHER CAUSES

It is possible that the patient may have had a transient ischaemic attack or some other catastrophic spontaneously resolving neurological event; however there is no reason for this to occur in a healthy young subject. Psychiatric illness or malingering could present in this way, however there was no preceding history of psychiatric illness nor secondary gain to be derived.

Conclusion

II would appear that the most likely explanation for his symptoms was that the breath-hold diver experienced CAGE due to pulmonary barotrauma or systemic "paradoxical" embolisation of bubbles formed as a consequence of nitrogen loading in the tissues and venous circulation from repeated breath hold dives.

Acknowledgments

The author would like to thank Dr Stafford A Bourke, MB, ChB (NZ), former Resident Medical Officer at the Helena Goldie Hospital, Munda, Solomon Islands, for his assistance in reporting this case.

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THE WORLD AS IT IS

WELCOME EUROPEAN JOURNAL OF UNDERWATER AND HYPERBARIC MEDICINE

John Knight

Key Words

Diving theory, general interest, hyperbaric research, physiology, diving medicine.

The SPUMS Journal congratulates the European Underwater and Baromedical Society (EUBS) for producing the first issue of the European Journal of Underwater and Hyperbaric Medicine (EJUHM) and looks forward to many years of international co-operation. The EUBS Newsletter, edited by Dr Peter Mueller, has undergone a transformation into a quarterly Journal, published in English, with an International Editorial Board of 32 drawn from 21 countries. SPUMS members can take pride in the fact that Dr Mueller, who is a member of SPUMS, when he was appointed Editor of the EUBS Newsletter last year, told the Editor that his aim was to turn the EUBS Newsletter into a Journal like the South Pacific Underwater Medicine Society Journal. Dr Mueller is to be congratulated on persuading the Committee of EUBS to back his ideas and on the content of the first issue.

Included in this issue are well worth reading papers by David Elliott (Medical Assessment of Fitness to Dive) and Valerie Flook (The Physics and Physiology of Decompression). The thrust of her paper is that off gassing and bubbles are controlled by physics rather than by supersaturation, and that the basic physics of off gassing were published in 1963.[1] The Editor has always been amazed that so little attention has been paid, in the underwater medical literature, to the asymmetry between gas uptake and gas excretion which has been known to anaesthetists as far back as 1956, when he started his anaesthetic education, as the explanation of why induction of anaesthesia with nitrous oxide was faster than the recovery time. Dr Flook's paper, part of a *Back to Fundamentals* series, should be read by all divers interested in the risks of bubble formation. Dr Mueller deserves congratulation on the standard of production of the European Journal of Underwater and Hyperbaric Medicine. There appears to be only one typographical error, a standard that the SPUMS Journal has hardly ever achieved!

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OZTEK 2000 AUSTRALIAN DIVING TECHNOLOGIES CONFERENCE

Lynn Taylor

Key Words

Decompression illness, equipment, meeting, mixed gas, rebreathers.

Richard Taylor again organised a successful OZTek conference in Melbourne, with the help of David Strike and Barry Heard. The topics for the general meeting were of interest to all divers, even if they had no intention of using high tech equipment. Those interested in rebreathers had displays and workshops as another part of the conference while the general subjects were being presented.

On the Friday night the decks of the *Polly Woodside* were covered with drinkers celebrating the start of OZTek 2000. A wonderful place to meet old friends and new speakers.

Developments in decompression theory

In his introduction Chris Parrett, creator of the Abyss decompression software, referred to the 4 goals of deep decompression recreational diving. To dive deeper, for longer, with shorter deco stops and to experience no decompression illness (DCI). A daunting task.

Chris took us through an explanation of the Reduced Gradient Bubble Model (RGBM) theory which is used for his latest version of Abyss. Much of the physics is complex, but the focus is on micronuclei, bubbles, and the surfactants that stabilise them. In utilising this theory to calculate decompression algorithms, the goal is to keep the bubbles in their tiny stable micro-nuclei state. The decompression profiles produced by RGBM calculations incorporate deeper decompression stops than most other algorithms. These attempt to maximise the rate of inert gas elimination by incorporating a maximal ascent to the first stop, establishing the greatest possible tolerable inert gas pressure gradient in tissues, which may well cause bubbles to form before the first stop. The RGBM is designed to minimise the chance of "exciting" bubble micronuclei into growth; hence, the focus is on a reduced gradient. The deeper early stops, perhaps surprisingly, produce reduced time at the shallower stops, and an overall reduction in decompression time.

The first of Chris' algorithms, the Abyss 100 required 524 minutes of decompression for a dive to 84 m for 250

minutes. To improve safety Abyss 120 required 705 minutes of decompression for the same dive. The last of his tissue supersaturation algorithm, the Abyss 150 required 1,426 minutes of decompression for the same same. However using the new Abyss RGBM model only requires 413 minutes of the same level of, presumed, safety. The RGBM theory is incorporated in the algorithm used in both the SUUNTO and ABYSS dive computers.

An interesting theory discussed by Chris involves the concept that micronuclei can be "crushed". The theory is that surfactant molecules coat the surface of micronuclei, otherwise they would disappear because of surface tension effects. Pressure at depth is thought to squash these molecules together so that they compete with each other for space and eventually 'pop-off'. The micronuclei bubble is now de-nucleated and the bubble seed is eliminated. This is the (unsubstantiated) theory supporting a short deep 'bounce' or 'crush' dive at the start of a day's diving activities.

Developments in cave diver rescue and training

Lamar Hires told us that between 1950-1998 there have been over 450 diving fatalities in the overhead environment (439 male + 21 female) with 401 being in the USA and 19 in Australia. In the 1990s, most of these divers have been trained to cave diver level or above. Fatalities have largely been due to errors in switching to the wrong gas mix at the wrong depth. But losing the line in a blackout situation still causes deaths. Lamar Hires is a volunteer cave rescuer and he presented details of an underwater cave rescue and recovery clinic he has developed where cave divers can gain practical experience in rescue and recovery techniques. His description of one rescue where two buddies, expecting to probe deep into the cave, lost contact in nil visibility within minutes of entering the cave was rivetting. The rescue was due to the lost diver's sitting quietly for over three hours waiting to be rescued. Her confidence was misplaced in the first attempt to find her as the rescuers did not ask her buddy the right questions and assumed that they had separated well inside the cave. Lamar Hines reinterviewed him and established the fact that they had not found the line into the cave, so were close to the entrance. When she heard the divers enter the water she started tapping her tank and led her rescuer to her position in a cave off and above the entry.

Recompression chambers in the South Pacific

Divers Alert Network (DAN) are undertaking an international project to register all recompression facilities and gather information on location, contact number, staff experience and chamber capabilities. As part of this process, DAN will be evaluating all chamber standard operating procedures for dealing with an emergency.

Bob Ramsay has been involved in setting up chambers in Papua New Guinea (Por Moresby) and Vanuatu (Santo) in the last few years. Now if you get bent on the *President Coolidge* you no longer have to be evacuated to Australia.

In Australia there are 12 registered facilities and 2 in New Zealand (Devonport Naval Base and Christchurch).

Aspects of the pathophysiology of decompression illness

Dr Simon Mitchell, in a wide ranging review, discussed the classification of decompression illness (DCI), the mechanisms of bubble formation (with examples of their effects in some organ systems) and the major risk factors associated with DCI.

His presentation focussed on the formation of bubbles in the tissues and blood from dissolved inert gas (traditionally called decompression sickness, DCS) and started with a basic explanation of how bubbles form. Inert gas is absorbed into blood and tissues during exposure to increased pressure; bubbles then form during/after ascent when the pressure of the dissolved gas in the blood/tissues exceeds ambient pressure (supersaturation). Quite how bubble formation and growth is initiated is unknown. Surface tension at the gas-fluid interface creates an immense physical force that resists de novo bubble formation. In theory, such force cannot be overcome by the degree of supersaturation achieved in a conventional dive. In reality, venous bubbles have been detected in man after air saturation dives to only 3.5 m. These dissonant observations gave rise to the theory of pre-formed "micronuclei". These minute bubbles, whose source is uncertain and which may be stabilised by surfactants, act as seeds which grow in conditions of inert gas supersaturation. In animal experiments venous bubbles are detectable within minutes of a dive, peak at 25 minutes and are stable for 1-2 hours. A similar time course for bubble detection has been described in humans.

Dr Mitchell mentioned that exercise during pressure exposure hastens the absorption of inert gas in those tissues whose perfusion is increased during work, so increasing decompression requirements and the risk of DCI. However, mild exercise during decompression enhances inert gas elimination, reduces decompression requirements and venous bubble formation, and so reduces the risk of DCI. Gentle finning on a safety/deco stop is advantageous. Strenuous post-dive exercise, such as pulling up an anchor line, has been shown to increase the risk of DCI.

The relationship between cold and DCI is complex and depends upon the timing of the cold exposure. If a diver is cold from the start of a dive, data suggests that bubble formation and the risk of DCI is reduced, probably as a result of decreased blood perfusion and reduced on gassing.

If a diver is warm and peripherally well perfused initially and then becomes cold and poorly perfused during the dive, the risk of DCI is increased. Exposure to excessive cold after a dive is associated with an increase risk of DCI (perhaps due to reduction in perfusion and off gassing). However, active re-warming (such as a hot shower) has been linked with the precipitation of DCI, probably due to a sudden rush of bubbles from a reduction in gas solubility in the rapidly warmed superficial tissue.

Other presentations

Bernie Chowdhury, the publisher of Immersed, discussed the scuttling of the German fleet at Scapa Flow in the Orkney Islands at the end of WW1. The description of how Ernest Cox's determination to salvage some of the ships was carried out successfully, although he knew nothing of diving or salvage, but was able to think latterally. The ships had been scuttled not torpedoed so he reasoned that if all the deck and side openings could be closed the ships could be refloated. Among other things, to provide dry working conditions he had old boilers rivetted together and used them as vertical coffer dams on the decks. In another presentation Bernie recounted his experiences in Icelandic diving where one water filled lava tunnel had been largely closed by the effects of plate tectonics in the year between two visits. His diving had to be done in a Superlite helmet, which made using normal recreational diving gear with a separate mask and mouthpiece a bit difficult. The reason for this was an unhealed perforated eardrum. In the dry in the helmet water could not get into the middle ear. The price was not being able to put his head back very far. What people will do to get into icy water in foreign parts!

Gary Gentile described how the expedition, on which he was the only American among Brits, managed the supply problems, gas supplies and Anglo-Irish race relations (rescue three fishermen from their sinking boat on the first day's diving), using tri-mix, on the *Lusitania*.. He explained the design and use of a floating deco-stop station, which allowed safe decompression while drifting in the strong currents. His fortnight's "holiday", working and diving from 0700 to 2300 every day, only cost him some $US2,000 for travel, accommodation, food, gas supplies etc. His transatlantic air fare was only few hundred dollars extra.

Keith Gordon, from New Zealand, who recently spent days waiting for sea conditions suitable for a dive using tri-mix on the wreck, described the sinking of the *Niagara* in 1941 and the recovery of most of the estimated 590 gold bars on board using grabs directed by a diver in a one-atmosphere bell.

David Applerley described the cave structure of the Pearce Resurgence, in New Zealand, which he dived using an Inspiration closed-circuit rebreather reaching depths of 125 m. There is more to this story as they did not reach the end. The approach to the dive site is by helicopter!

Noel Taylor gave us a guided tour of 8 diveable wrecks at Guadalcanal and briefly reviewed the spectacular diving that the Solomon Islands has to offer. He then described the history behind the sinking of the troop ship *President Coolidge* off Santo and from there went to Bikini and its wrecks. Well off the beaten track nitrox or rebreathers are recommended as the best wrecks are deep.

The OZTek Industry Achievement Award was presented to Kevin Denlay, in recognition of his development and promotion of technical diving within Australia and the South Pacific.

The OZTek Australasian Technical Diver of The Year was awarded to David Apperley, in recognition of having done the most to extend the range of technical diving within the Australian and South Pacific region in the past 12 months.

All in all, an excellent, informative and entertaining two days. Plans are afoot for OZTek 2001.

Lynn Taylor is a PADI IDC Staff Instructor and a DAN O_2 instructor. She came to New Zealand, from England, in 1994 and soon found a passion for diving. Her interests in the technical and medical aspects of diving have stemmed from her science and research background, BSc and PhD, and hence her interest in OzTeK. Her address is 26 Barker Rise, Browns Bay, Auckland, New Zealand. Telephone + 64-9-367-2948. Fax +64-9-367-2500. E-mail <ltt21040@GlaxoWellcome.co.uk>.

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 evidence of satisfactory completion of examined courses in both Basic and Advanced Course in Diving and Hyperbaric Medicine at an approved institution.
3 The candidate must have completed the equivalent (as determined by the Education Officer) of at least six months full time training in an approved Hyperbaric Medicine Unit.
4 The candidate must submit a written research proposal in a standard format for approval by the Education Officer before commencing their research project.
5 The candidate must produce, to the satisfaction of the Education Officer, a written report on the approved research project, in the form of a scientific paper suitable for publication.

Additional information

The candidate must contact the Education Officer to advise of their intended candidacy, seek approval of their courses in Diving and Hyperbaric Medicine and training time in the intended Hyperbaric Medicine Unit, discuss the proposed subject matter of their research proposed, and obtain instructions before submitting any written material or commencing a research project.

All research reports must clearly test a hypothesis. Preference will be given to reports of original basic or clinical research. Case series reports may be acceptable if thoroughly documented, subject to quantitative analysis, and the subject is extensively researched and discussed in detail. Reports of a single case are insufficient. Review articles may be acceptable if the world literature is thoroughly analysed and discussed, and the subject has not recently been similarly reviewed. Previously published material will not be considered.

It is expected that all research will be conducted in accordance with the "Joint NH&MRC/AVCC statement and guidelines on research practice" (available at http://www.health.gov.au/nhmrc/research/nhmrcavc.htm). All research involving humans or animals must be accompanied by documentary evidence of approval by an appropriate research ethics committee. It is expected that research project and the written report will be primarily the work of the candidate.

The Education Officer reserves the right to modify any of these requirements from time to time.

The Education Officer's address is Dr David Doolette, Department of Anaesthesia and Intensive Care, The University of Adelaide, Adelaide, South Australia 5005. Telephone(0)8-8303-6382. Fax (0)8-8303-3909. E-mail <David.Doolette@adelaide.edu.au>

Key Words
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Fax + 61-(0)7-3371-1566
No funded training post.
No objection to someone getting self funded work experience through this unit.

South Australia

Hyperbaric Medicine Unit
Royal Adelaide Hospital
Dr David Wilkinson <dwilkins@mail.rah.sa.gov.au>
North Terrace
Adelaide, South Australia 5000
P +61-(0)8-8222-5116
F +61-(0)88232-4207
One 6 month anaesthetic rotation as part of a Provisional Fellowship year.

Tasmania

Hyperbaric Medicine Unit
Royal Hobart Hospital
Dr Margaret Walker <margaret.walker@dchs.tas.gov.au>
Hobart, TAS 7000
Phone +61-(0)3-6222-8193
Fax +61-(0)3-6222-8322
One anaesthetic registrar post, half time anaesthesia, half-time hyperbaric medicine.

Victoria

The Alfred Hyperbaric Service
Dr Ian Millar <I.Millar@alfred.org.au>
Alfred Hospital
Commercial Road
Prahran, Victoria 3181
Phone +61-(0)3-9276-2269
Fax +61-(0)3-9276 3052
One 6 month full-time registrar position available to those in anaesthesia, intensive care physician or emergency medicine training programs.

Western Australia

Department of Diving and Hyperbaric Medicine
Fremantle Hospital
Dr Robert Wong <Robert.Wong@health.wa.gov.au>
Fremantle Hospital
Fremantle Western Australia 6160
Phone +61-(0)8-9431-2233
Fax +61-(0)8-9431-2819
One full-time registrar, one part-time anaesthetic registrar (6 months part-time rotation) and one emergency medicine registrar (12 months part time).

Key Words
Qualifications.

CONSTITUTIONAL CHANGES

The Annual General Meeting at Castaway Island on May 13th 2000 passed the motions detailed below to amend the Statement of Purposes and Rules of the Society.

That the heading Board of Censors on page 19 be changed to *Academic Board*.

That Rule 42 be changed by replacing the existing wording with *The Committee will appoint an Academic Board headed by the Education Officer*.

That Rule 42 (a) be changed by replacing the existing wording with *The make up of this Board will comprise individuals with proven clinical, scientific and research skills in the fields of diving and hyperbaric medicine. The minimum number of Board Members will be the Education Officer and two others.*

That Rules 42 (b) and 42 (c) be amended by removing the words *of Censors* from both rules.

As no objections have been received it is assumed that the membership has voted in farour of the amendments which now come into force.

Cathy Meehan
Secretary of SPUMS

Key Words
Constitutional amendments

SPUMS

ANNUAL SCIENTIFIC MEETING 2001

will be held from
May 26th to June 2nd 2001
in
Madang, Papua New Guinea

Guest speakers
Dr James Francis and Dr Craig Conoscenti

Convenor Dr Guy Williams

Theme
Diving and the Lung

Workshop
Drowning/Near Drowning

Members wishing to present papers should contact
Dr Guy Williams
PO Box 190 Red Hill South
Victoria 3937, Australia
Tel + 61-(0)3-5981-1555 Fax + 61-(0)3-5981-2213
E-mail <guyw@surf.net.au>

Official Travel Agent is Allways Dive Expeditions
168 High Street
Ashburton, Victoria 3147, Australia
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Toll Free 1800-338-239
Fax + 61-(0)3-9885-1164
E-mail <allways@netlink.com.au>

THE

SPUMS

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WHICH GIVES ACCESS TO THE

SPUMS JOURNAL INDEX 1971-1998
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http://www.SPUMS.org.au

ROYAL ADELAIDE HOSPITAL
HYPERBARIC MEDICINE UNIT

Basic Course in Diving Medicine

Content Concentrates on the assessment of fitness of candidates for diving. HSE-approved course.

October Monday 23/10/00 to Friday 27/10/00
Cost $Aust 750.00

Advanced Course in Diving and Hyperbaric Medicine

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

October/November
Diving Monday 30/10/00 to Wednesday 1/11/00
Hyperbaric Thursday 2/11/00 to Friday 3/11/00
Cost $Aust 750.00

$Aust 1,300.00 for both courses taken back to back

Diving Medical Technicians Course

Unit 1 St John Ambulance Occupational First Aid Course (an essential prerequisite) and medical lectures at RAH. (Cost in 1999 of First Aid course in Adelaide $Aust 545.00 payable to St John Ambulance.)
Unit 2 Diving Medicine Lectures and
Unit 3 Casualty Paramedical Training.

Cost of three unit course $Aust 1, 250.00

October 2000

Unit 1	9/10/00	to	13/10/00
Unit 2	16/10/00	to	20/10/00
Unit 3	23/10/00	to	27/10/00

Diver Medical Technician Refresher Courses
(includes lectures and practical)

October 2000 16/10/00 to 20/10/00

Cost $Aust 500.00

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

Telephone	Australia	(08) 8222 5116
	Overseas	+61 8 8224 5116
Fax	Australia	(08) 8232 4207
	Overseas	+61 8 8232 4207

DIVING MEDICAL CENTRE

SCUBA DIVING MEDICAL EXAMINER'S COURSE

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

Easter Weekend 2001.

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

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

Information and application forms for courses can be obtained from

Dr Bob Thomas
Diving Medical Centre
132 Yallambee Road
Jindalee, Queensland 4047
Telephone (07) 3376 1056
Fax (07) 3376 4171
E-mail <bthomas@eis.net.au>

FREMANTLE HOSPITAL RECREATIONAL SCUBA DIVING MEDICAL EXAMINER COURSE

This course for covers introductory diving physics and physiology, medical assessment of scuba divers (AS4005.1) and an introduction to diving medicine.

Venue
Department of Diving & Hyperbaric Medicine,
Fremantle Hospital, Western Australia

Dates
Friday 17th - Sunday 19th November 2000

Cost
$ 726.00 inclusive of GST

RACGP CME
This course has been submitted to the RACGP for point allocation in the CME category of the QA&CE Program.

For further information or to enrol contact
Dr David Wright
Phone (08)-9431-2233
Fax (08)-9431-2235
E-mail <David.Wright2@health.wa.gov.au>

ROYAL AUSTRALIAN NAVY
MEDICAL OFFICERS' COURSE IN
UNDERWATER MEDICINE

November 27th to December 8th 2000

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

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

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

For information or to enrol contact

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

Tel: (61) 2 99600333
Fax: (61) 2 99604435
E-mail <Robert.Green2@defence.gov.au>

ROYAL NEW ZEALAND NAVY
HEALTH SERVICES
DIVING MEDICINE COURSE 2000

The course introduces candidates to the principles of diving and hyperbaric medicine and focuses on the assessment of an individual's fitness for diving and hyperbaric exposures and the first aid for common diving illnesses.

The course is recognised by the New Zealand Department of Labour, the United Kingdom Health and Safety Executive and the Academic Board of the South Pacific Underwater Medicine Society which gives recognition under AS/NZS 2299.1 1999.

This year the course will be held, at the Naval Base in Auckland, from (a.m.) Saturday 2000/11/18 to (p.m.) Tuesday 2000/11/21.

The fees are $NZ 750.00 (inclusive of GST); this includes a complete set of course notes, and morning and afternoon tea. A maximum of 25 places will be available on the course and early enrolment is advised. This requires payment of $NZ 150.00 deposit. Cheques should be made payable to **NZ Defence Force-Navy.**

For further information, including information about accommodation in the Devonport area, please contact
Angie Smith, PA to the Director of Naval Medicine,
Naval Base, Private Bag 32901, Auckland
Phone +64-(0)9-445-5972. Fax +64-(0)9-445-5973
E-mail <navyhosp@ihug.co.nz>

LETTERS TO THE EDITOR

RECREATIONAL DIVING MEDICALS

Department of Medicine
Cairns Base Hospital
2000/5/22

Dear Editor,

We were grateful to read your editorial comment on our recent paper on the diving medical examination in practice published in the Medical Journal of Australia.[1] Your comments were somewhat at variance from those of the President of SPUMS who wrote an editorial in the same issue of the Medical Journal of Australia[2] which was fairly critical of our paper. It was perhaps unfortunate that Dr Walker did not declare her conflict of interest as President of SPUMS and also that she suggested that our data showed that "most doctors avoided making the "wrong" decision by being more conservative than the Australian standards".

In fact, as you correctly pointed out, our data showed exactly the opposite with most of the responses which differed from AS4005.1-1992 being in favour of allowing the diver to dive. Dr Walker comes out pretty strongly in her editorial in the MJA for continuing the status quo. Again, as you correctly point out in your editorial, the official position of SPUMS adopted in 1995 at the Fiji Annual Scientific Meeting is as we outlined with emphasis on education, risk assessment and informed consent.

The sad fact is that 5 years after having come to this conclusion SPUMS has done nothing whatsoever to make this a reality and the President of SPUMS is still going into print putting forward the contrary view.

Perhaps it is time for SPUMS to make a clear and unequivocal statement as to what the official policy is and preferably publish this somewhere like the Medical Journal of Australia as a first move to implementing the decisions taken in 1995 and improving the current and unsatisfactory situation.

Graham Simpson
Clinical Associate Professor
University of Queensland
Director of Thoracic Medicine

David Roomes
Medical Registrar

References

1 Simpson G and Roomes D. Scuba diving medical examinations in practice: a postal survey. *Med J Aust* 1999; 171: 595-598
2 Walker R. Water hazards. *Med J Aust* 1999; 171: 584-586

Key Words
Fitness to dive, letter, medical standards.

The above letter was forwarded to Dr Robyn Walker for her reply which is printed below.

2000/7/14

Dear Editor

The letter from Drs Simpson and Roomes suggests that a conflict of interest arises if the President of SPUMS comments on matters relating to diving safety. Surely that is an accepted role for the President of a Society which exists to promote and facilitate the study of all aspects of underwater medicine. The Medical Journal of Australia was aware of my role as President of SPUMS. The fact that they did not publish this I can only interpret as acceptance that this did not pose an ethical problem.

Medical standards may be considered by some to be black and white, however, patients come in shades of grey. I believe it is entirely reasonable for practitioners to further investigate, refer, or seek expert advice on individuals fitness to dive. The data[1] shows that on initial assessment most respondents did not pass an individual fit to dive.

To certify that a person is fit to undertake any form of diving requires that individual to be physically fit, medically healthy and psychologically stable. Many doctors limit their consultation to the exclusion of disease rather than undertaking a comprehensive assessment based upon their knowledge of different marine environments, range of diving equipment, diving gas mixtures to be used and the skills required to ensure the diver's safety. I fully support a system where practitioners provide an assessment and risk evaluation based on the above criteria. However, it is unfortunate that the great majority of Australian doctors do not have this training or experience and therefore rely on the proscriptive guidelines as detailed in the Australian Standards.

SPUMS is an advisory and not a regulatory body. The Society will continue to lobby for improved training in diving medicine for medical practitioners, but can not take responsibility for determinations of regulatory agencies.

Robyn Walker
President of SPUMS

Reference

1 Simpson G and Roomes D. Scuba diving medical examinations in practice: a postal survey. *Med J Aust* 1999; 171 (11/12): 595-598

Key Words
Fitness to dive, letter, medical standards.

BOOK REVIEWS

HYPERBARIC MEDICINE PRACTICE. 2nd Edition.
Editors, Eric P Kindwall and Harry T Wheland.
ISBN 0-941332-78-0. 1999.
Best Publishing Company, P.O.Box 30100, Flagstaff, Arizona 86003-0100, U.S.A.
Price from the publishers $US 128.00. Postage and packing extra. Credit card orders may be placed by phone on +1-520-527-1055 or faxed to +1-520-526-0370. E-mail divebooks@bestpub.com .

This second edition of Hyperbaric Medicine Practice by Kindwall and Whelan is a welcome addition to any hyperbaric physician's library. It is clearly written and well organised. The novice reader can obtain a clearer understanding of the basic mechanisms of HBO from the chapters dealing with wound healing, infectious diseases and reperfusion injury. The more experienced reader may refer immediately to chapters concerned with the use of hyperbaric oxygen (HBO_2) in specific conditions such as gas embolism, carbon monoxide poisoning, radiation injury, crush injury and compartment syndrome.

Chapter 3 provides a detailed review of the physiological effects of HBO_2. The book, however, does not really answer the question of what is the optimal dose of oxygen and does this differ with different disease states. We accept different treatment depths for gas gangrene and radiation injury yet the development of an oxygen dose response curve for each treated condition has not yet been elucidated. This is not a criticism of the authors, rather an indication of how much work is still to be done in this field.

The specific indications for the use of HBO_2 follow the approved Undersea and Hyperbaric Medical Society guidelines with additional chapters on what are considered experimental indications. The book reflects its American authors, with the recommendation to treat gas embolism at 6 ATA. Australian practice has evolved with the initiation of treatment at 2.8 ATA with the option of going deeper and changing to a heliox or nitrox mixture as the clinical condition dictates. At times the book is a little confusing with the interchanging of the phrases "decompression sickness" and "decompression illness". An explanation of the different nomenclatures would have been useful.

Different chapter authors reflect differing philosophies in their approach to myringotomy. This should not confuse the reader but indicates that there is a range of opinions, all of which may be appropriate under differing circumstances.

The book provides a comprehensive overview of monoplace chambers, which are popular in the United States, but not used as much in Australasia. Likewise reimbursement issues and health fund subsidies reflect a North American problem. However, these issues are becoming more global and Australian readers are likely to face similar problems in the near future.

Problem wounds are increasingly the realm of the hyperbaric physician and the chapters dealing with this subject are comprehensive and multifaceted, with a focus on total patient management as opposed to just a discussion of HBO_2.

In general the book is well referenced and is an excellent resource source. On page 632 however, the author states that internal cardiac pacemakers are unaffected by the hyperbaric environment which is not entirely correct.[1] Also in chapter 22, references 10, 45 and 46 are listed as not available, preventing the reader from ascertaining the veracity of the reference. The caption for figure 4 , on page 855 in chapter 34, appears to have been transposed and it would be useful if the figures in this chapter were referenced to the text.

The chapters dealing with investigational issues are interesting but reflect the need for randomised trials before accepting HBO_2 as an adjunct in their management. Desperate families seeking advice as to whether HBO_2 will help their loved one with cerebral palsy or hypoxic brain damage approach hyperbaric facilities on a daily basis. It is important, particularly in the era of evidenced-based medicine, that we can justify the use of any intervention or treatment. The reader should be aware that the use of HBO_2 in Hansen's disease (leprosy) or incomplete ileus is not standard practice. Statements such as "the reasons for using two different treatment pressures are not based on gas laws or physiology, but have to do with local time constraints for scheduling and patient preference" (Chapter 40, Hyperbaric Oxygen in adhesive or incomplete ileus associated with abdominal surgery) do little to advance the science of HBO_2.

Overall this book is a well-referenced, state of the art, comprehensive manuscript dealing with the science and clinical applications of HBO_2. I recommend it highly to all interested in the practice of hyperbaric medicine.

Reference

1 Wilmshurst PT. Cardiovascular problems in divers. *Heart* 1998; 80 (6); 537-538

Robyn Walker

Key Words

Book review, hyperbaric oxygen, medical conditions and problems, tables, treatment.

REVERSE DIVE PROFILES
PROCEEDINGS OF THE REVERSE DIVE PROFILE WORKSHOP. OCT 29-30 1999
Editors. Lang MA and Lehner CE.
Washington DC: Smithsonian Institution.
Soft cover, 295 pages. Published January 2000.
Review copy from Smithsonian Institution.
RRP $US 20.00 plus postage and packing

A reverse dive profile, in the context of this workshop, refers to bounce (as opposed to saturation) diving where a repetitive dive is deeper than a preceding dive or a multilevel dive proceeds to a deeper level. This diving pattern is often prohibited in recreational (but not military or commercial) diver training programs and texts, notionally owing to increased risk of decompression illness. Two chapters in the proceedings (Egstrom; Richardson and Shreeves) review training manuals from various sources and trace the first references prohibiting reverse dive profiles to recreational diving manuals of the 1970's, but neither are able to explain the origin.

There has never been specific experimental or field evaluation of the relative decompression risk of forward versus reverse dive profiles. Motivated by the needs of scientific divers, but equally applicable to recreational divers, the workshop assembles and evaluates considerable theoretical and practical evidence for no-decompression stop air or N_2O_2 diving and reaches the conclusion that "We can find no reason for the diving communities to prohibit reverse dive profiles for no-decompression [stop] dives less than 40 msw (130 fsw) and depth differentials less than 12 msw (40 fsw)." They admonish against extrapolation beyond this very specific conclusion.

I found this conclusion somewhat unsatisfying because it is not clear what hypothesis was addressed. Apparently, implicit in the conclusion is that the decompression requirements are calculated using existing algorithms, but despite attempts by some (notably Richard Moon and Tom Neuman) during the consensus discussion, this was not clarified. My confusion arises because many related issues are covered in this interesting book. Amongst these is whether reverse dive profiles are intrinsically more risky than forward dive profiles. The answer is intuitively no, because if you understand the important physiology you should be able calculate acceptable decompression for either profile type. However, the corollary question is whether the important processes are sufficiently understood to do these calculations.

The first half of the book is devoted to description and application of the various decompression algorithms, and represents state of the art of decompression theory. The most modern algorithms are the gas kinetic-bubble dynamic models which include the Duke bubble volume model [BVM(3)], the DCIEM bubble evolution model [BEM], and the USN linear-exponential kinetics [LE] models (USN93D, JAP98-2), all of which have parameters estimated by fit to diving data, and Michael Gernhardt's bubble dynamic model [BDM] and Valerie Flook's physiological model (notable for not having a catchy abbreviation), which are theoretical. Although these models have been developed over the last decade, they are based on the parallel perfusion-limited compartments of Haldane et al. 1908 and the bubble dynamic model developed by Van Liew and Hlastala 1969. The nucleation algorithms (varying permiability model [VPM] and reduced gradient bubble model [RGBM]) have their origins with Yount et al. 1970s, and these also use the Haldane compartmental approach but examine the effects of compression and decompression on the crushing or activation of gas nuclei and the regeneration of nuclei according to statistical mechanical models. The Haldane/Workman/Schreiner approach, pioneered by Haldane and developed by the USN through to the 1957 standard air tables is covered in detail. The diffusion-limited algorithms include the Royal Navy bulk diffusion model of the 1950's, Brian Hills' 1966 Thermodynamic model, and the DCIEM 1984 non-linear model.

There are some thorough technical descriptions, notable the review of the classes of algorithms by Wienke and O'Leary and the chapter by Yount et al. reviewing the development and updating of the VPM model. If you work in the area of decompression theory, you will find little new, but then you were probably at the workshop. If you are a serious student of decompression theory you will find this a very informative review, but do not expect numerical recipes or descriptions of implementations. Unfortunately, despite chapters by the authors, the two models that have not been published in the mainstream scientific literature (Gernhardt; Flook) are not covered in any detail. Flook's model is a hybrid of the Mapleson/Egan standard man model and Van Liew's bubble model and Gernhardt's model, described only with a diagram, is apparently similar. A highlight of the proceedings is the chapter by Hugh Van Liew explaining that all the bubble models are speculative until the existence and nature of gas nuclei are better defined with experimental evidence.

Many of the algorithms are used to compare forward and reverse dive profiles. The different chapters are somewhat standardised, examining predominantly 12 msw/30 msw or 18 msw/30 msw repetitive no-decompression stop dive pairs with surface intervals ranging from 30 minutes to 2 hours, but differ in how they arrive at bottom times. Bubble indices as measures of decompression stress are used to evaluate standard bottom times according to the BDM and Flook's physiological model and real diving data according to the DCIEM BEM (Gernhardt; Flook; Nishi and Tikuisis). Representatives of all the classes of algorithms are used to calculate comparative decompression requirements for standard bottom times (Wienke and O'Leary). No-decompression stop times for the standard dives are compared using both the models and

tables of the DCIEM 1984, USN 1957, and the PADI/DSAT algorithms (Nishi and Tikuisis). An interesting chapter by Gerth and Thalmann uses the BVM(3) and USN93D models to predict the risk of DCI for forward and reverse no-stop profiles generated by many of the algorithms contained in diver carried decompression computers as well as the USN, DCIEM, and PADI/DSAT standard air tables. The general findings of these presentations were that although there were some differences in stress indices, the risk of DCI was not significantly different between forward and reverse dive profiles.

Several chapters examine reverse dive profiles in decompression validation data. The first chapter of the proceedings (Lewis) explains that reverse dive profiles predominate in the original validation data for the USN 1957 tables. This chapter also examines the evolution of repetitive diving, concentrating on the USN perspective and details the Des Granges single compartment algorithm for the USN 1957 repetitive procedures. Evaluation based on the full model compartmental gas loadings suggests this approach is valid for all but very short surface intervals. The N_2-O_2 Primary Data training set used to calibrate the BVM(3) and USN LE models contains some reverse profiles (Weathersby and Gerth) and DAN's Project Dive Exploration database contains some reverse dive profiles from recreational diving (Vann et al.). Brubakk and Eftedal argue in favour of bubble counts rather than treated DCS for evaluating algorithms and illustrate this with pulmonary artery bubble counts in an animal model.

Egstom gives a brief, theoretical account of the principles of risk assessment. Weathersby discusses quantitative risk management illustrated by the introduction of the USN93D model into the US Navy. There is also some basic probability theory and a discussion of the use of maximum likelihood regression in the development of statistically based decompression algorithms (Wienke and O'Leary).

The last third of the book presents practical experience with reverse dive profiles from a variety of diving communities (I apologise for not listing all the authors). An analysis of 209 DCS cases treated at USC Catalina found no difference in the need for prolonged treatment between forward versus reverse dive profiles (Huggins). There are a variety of case reports and statistics of DCS following reverse profiles from recreational diving communities. There is a description of common reverse dive profile practices in the commercial diving industry along with low incidence of DCS (Overland). Most of the reported statistics suffer from unknown denominators and unknown prevalence of reverse dive profiles. Furthermore, in an earlier chapter, Brubakk and Eftedal propose that treated decompression illness is a poor endpoint for decompression studies and provides a nice review of unrecognised/untreated decompression illness in diving populations. Several of the recreational diving instructional organisations as well as the scientific diving community presented their existing policies regarding reverse dive profiles.

The book is a soft cover, black and white format. The quality of the figures is occasional poor; these appear to be slides that have not translated well to the black and white format. Also, figure legends are frequently inadequate. The accuracy of the citations and references is occasionally poor. However, these minor criticisms are easily forgiven in view of the low cost and extremely rapid publication.

David Doolette

Key Words

Book review, decompression illness, diving tables, risk, safety.

REVERSE DIVE PROFILES
PROCEEDINGS OF THE REVERSE DIVE PROFILE WORKSHOP. OCT 29-30 1999
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Review copy from Best Publishing Company, P.O.Box 30100, Flagstaff, Arizona 86003-0100, U.S.A.
Price from Best Publishing Company $US 20.00. Postage and packing extra. Credit card orders may be placed by phone on +1-520-527-1055 or faxed to +1-520-526-0370. E-mail <divebooks@bestpub.com>.

The SPUMS Journal was sent two review copies of this book. One was reviewed above by David Doolette, PhD, the Education Officer of SPUMS, whose work is about decompression theories and calculations. This review is by a non-mathematician anaesthetist, as perhaps more representative of the general run of SPUMS members.

It appears that workshop was assembled to cope with complaints from the scientific diving community that the current recreational teaching, that one should always do one's deeper dive first in order to reduce the risk of decompression sickness (DCS), was affecting their work. In the USA scientific divers are recreationally trained, unlike the UK and Europe where they now have to be trained to professional standards. Perhaps this is why there is so little input from Europe. From this book it is clear that the prohibition is not based on any evidence, except that doing repetitive dives that way allows a longer bottom time for the second dive. The fact that there are so many algorithms successfully avoiding DCS is evidence that they all have errors in their calculations. Read the book and make up your own mind.

Reverse Dive Profiles should be read by all divers who are interested in the practicalities of decompression

tables and computers. However it is a book which requires thought on the reader's part. Many of the graphs appear to have been taken from PowerPoint presentations as it is clear that many of them must have had colours distinguishing the various lines, which lost their distinctions when converted into black and white. Guessing which line was demonstrating what was a struggle at times. But once over that hurdle the book was well worth reading. It is not alway easy reading because many of the presentations have a high equation/text ratio. That said, ignoring the equations still allows the message to come through to the naive reader.

The opening chapter on the evolution of repetitive diving is USA-centric and, to an anaesthetist familiar with the uptake and excretion of anaesthetic gases and the range of responses to given doses of anaesthetic drugs in different people, rather disappointing as, although the reasoning behind the tables is explained, there is no mention of the fact that knowledge of such things, available in the 1950s, was completely ignored in the USA until the early 1980s.

It is quite clear that neither the US Navy (USN) nor the US Commercial Diving industry have rules banning reverses profiles. But both organisations have definite controls on how diving is done, which recreational divers do not. Unfortunately although all these dives are logged, they are not recorded in trustworthy data bases so no denominator can be provided. Although the USN data base includes the decompression details of "all" dives, only those from experimental units are reliable. Why ? Fleet reports are unreliable because an unknown number of divers are decompressed for extra time and depth as a safety measure applied by the supervisors. A dive performed to 60 ft for 60 minutes, and logged as such, may actually have been decompressed as for 70 ft for 70 minutes. Commercial diving decompression data is treated as a commercial secret, so is not available for research.

In fact the only incontroverible evidence about DCS risk of dives comes from the North Sea and reveals that increasing depth and time increase the incidence of DCS regardless of the tables used. A summary of this evidence was published in the Journal in 1998.[1] With the advent of diving computers, with many different algorithms, much computer assisted diving cannot be compared with the various databases on which the modern tables are based because the dive has been recorded without information about the computer's algorithm.

The presentations make it clear that none of the various decompression algorithms accurately describes what happens in the body during decompression. Almost all required adjustments to be made to prevent clinical decompression sickness or bubbles forming as dives go deeper. The bubble models seem to be a bit closer to what really happens as they often offer the deep stops, which have been shown to be needed at deeper than USN stop depths. In many ways the various presentations were comparing oranges, apples and various other fruit as if they were all the same.

One of the most interesting sessions is that on Operational Experience which covers ten different approaches to "reverse dives" none of which have convincing evidence either way about whether there is a problem. The fact that there is no evidence either way largely reflects the fact that most public table testing has been done by governmental agencies while the Commercial sector has largely kept its decompression programs secret. Testing of recreational programs has been accurately recorded only in small numbers and almost certainly not representative of what is actually done in the field.

This review is coloured by the reviewer's lack of faith in the accuracy of computer simulations of drug uptake of intravenous drugs. His belief in computer accuracy was shattered well over 30 years ago. The drug was thiopentone and the experiment showed that the computer simulations were wrong. When thiopentone was first introduced the cessation of its action was attributed, on the basis of its solubility in fat, to uptake by fatty tissues. However new computer simulations of blood flow showed that the thiopentone "was being taken up by muscle, because they received much more blood flow than fat". The experiment was to inject rats with a radioactive relative of thiopentone and then drop the rats into liquid nitrogen at a series of timed intervals after injection, up to and beyond the time that the rats would have woken up. The rats were immediately snap frozen. They were then halved with a bandsaw and the halves placed on X-ray films for about 6 months. When the films were developed it was clear that the heart muscle had been full of radioactivity almost from the moment of injection, but there was none in other muscles until long after wake-up time. Between injection and wake-up time the radioactivity was building up in the fat and then later was slowly redistributed to muscle. Unfortunately your reviewer has been unable to find the reference after some hours of hand searching the likely Journals over the likely years. The search was handicapped by not knowing the correct title nor the name of the author! Any anaesthetist who can remember the paper is asked to pass on the reference to the Editor.

Reading through the book will improve one's understanding of the various algorithms used and bring their limitations and strengths to the reader's attention. The various discussions after the sessions open up insights not available in the papers. The session on the construction of the conference conclusions is an eye opener. Consensus comes as the result of horse trading words. One cannot disagree with the statements that there is not enough data to state that reverse profiles are more dangerous than forward profiles. But no one advanced proposals for the sort of experiments that are needed to assess the safety of the various algorithms. These, in the reviewer's opinion, would be expensive, needing to be of long duration, to get the

necessary number of subjects, and carried out in animals weighing around 70-90 kg to approximate to humans. Mini-pigs come to mind, they figured in some experiments years ago and have the useful advantage that they provide good barbecue, or perhaps spit roast, material at the end of the experiments!

I am quite certain that the majority of educated divers will learn something useful from reading this inexpensive book. It is not always easy reading but it stimulates the grey cells and shows up how little is really known about decompression and how dependant on accurately following the rules for ones tables, or computer, safe decompression really is.

John Knight.

Reference

1 Elliott DH. Decompression theory in thirty minutes. *SPUMS J* 1998; 28 (4): 206-214

Key Words

Book review, decompression illness, diving tables, risk, safety.

PSYCHOLOGICAL AND BEHAVIOURAL ASPECTS OF DIVING

Baruch Nevo and Stephen Breitstein.
ISBN 0-941332-73-X
Best Publishing Company, P.O.Box 30100, Flagstaff, Arizona 86003-0100, U.S.A.
Hardback. Pp 192. Published 1999. Price from the publishers $US 19.95. Postage and packing extra. Credit card orders may be placed by phone on +1-520-527-1055 or faxed to +1-520-526-0370. E-mail <divebooks@bestpub.com>.

This is both an exciting book and a very frustrating one. Let us start with the good news.

Baruch Nevo is a Professor of Psychology at the University of Haifa, Israel, and Stephen Breitstein is a Director of the Recanti Center for Maritime Studies at the same university. Both are highly experienced as divers, as educators and as researchers. They have collaborated to produce a readable book that should open up an area of diving activity that is inherently fascinating and which has often been overlooked in the past when seeking an explanation for the unusual and (occasionally disastrous) activities of the sub-aquatic human being.

Their starting premise is a valid one. Although many authors and researchers have noted the effects of the underwater environment upon psychological performance, this has often been as a side-product of research into essentially "physical" phenomena. There has never been any concerted attempt to correlate, in one specialist volume, our understanding of the effects of total immersion upon such basic human processes as perception, thinking and behaviour. Nevo and Breitstein have set out to review the past 40 years of the world's academic, military and diving group literature dealing with "the diver as a person" and to present the result as "an integrated state-of-the art review".

They begin their book with a conventional, and rather basic, overview of the physical and physiological differences between life for humans above and beneath the surface. Subsequent sections look, in greater detail, at subjects as diverse as colour, sound and distance perception in different water conditions, ability to perform manual and cognitive tasks, social and group activity and the effects of hyperbaric pressure upon human functioning and social interaction. All of this is fairly straightforward and informative, although it is not always clear whether the information is designed for the non-psychologist (who will probably skip a lot of the technical stuff) or the researcher (who will wish that examples and references were a lot more extensive!).

I must admit that I was eager to reach the section of the book that deals with the "personality" of divers and to see what the authors would achieve when they used their psychological expertise to examine the emotional components of dives that had gone horribly wrong. Unfortunately, with little more than a few brief accounts and solely retrospective examination to rely on, their "guesses" turn out to be as limited as yours or mine. Whilst few would quibble with the authors' assertion that "emotional stress and panic are major factors in diving accidents", neither their own research nor the reports of others, really confirm this belief or give rise to ways of predicting or pre-empting such a potentially disastrous response to diving mishaps.

In all fairness to the authors, they fully acknowledge their own restriction in this regard, and stress the importance of a good deal of further work in this, and many other, areas of study into the emotional, personal, mental and cognitive aspects of diver behaviour.

Indeed, "More Work Required" would probably be a good sub-title for this book. Each chapter critically reviews the results of past experiments and research that were frequently uncontrolled, unreproducible or simply anecdotal in nature. At the end of each chapter is a section called "Questions for Future Research". These will surely be a godsend for any PhD student seeking clever ways to combine academic study with their love of diving. It is also a good reminder of just how much more work, much of it very basic, remains to be done.

In terms of providing a basic overview of the relevant literature, Nevo and Breitstein have generally succeeded in their intention. However, given their professional backgrounds, it is unfortunate that the authors have generally avoided any attempt to critically analyse the published data that is available. For example, a 30-year-old study comparing the wagering patterns of diver and non-diver naval officers gains credibility as the unqualified assertion that "divers tend to gamble". By the same token, it is a little disingenuous to present data gathered from "hundreds of documents from ten countries published over a period of 40 years" without at least attempting to place that information within its cultural or chronological content. Although it is nice to see that Edmonds et al. rate a regular mention, the work referred to is the 1983 Second edition of "Diving and Subaquatic Medicine" and not the later revision. Similarly, although many old texts deserve to be preserved, it is disappointing to see that relatively few recent articles, conference papers and publications seem to have made it into this book. The book leaves one with the sense that its authors started out to write a comprehensive psychological textbook, but later decided to prune it for sale as a volume in a series on "popular psychology".

However, this is still a book that will be of considerable interest to all who feel there is more to safer diving than better equipment and more physiological research. If, for example, we all agree that "panic" is a major factor in diving disasters, it is long overdue for extensive exploration of the combinations of events and individuals that may lead to tragedy. Nevo and Breitstein's review of the extensive but muddled current literature is a good starting point. Perhaps their publisher can now be persuaded to send the authors back to their desks to write the second, (revised and greatly expanded) edition of this primer. It could well evolve into the essential and definitive textbook on a fascinating aspect of human psychology.

John Couper-Smartt

Key Words

Book review, diving safety, medical conditions and problems, panic.

DEADLY ENEMIES: TOBACCO AND ITS OPPONENTS IN AUSTRALIA

Ian Tyrrell
ISBN 0 86840 745 3
UNSW Press, University of New South Wales, Sydney, New South Wales 2052, Australia.
RRP $Aust 29.95.

Associate Professor Ian Tyrrell, the Head of the School of History at the University of New South Wales, provides a review of the history of tobacco use over the last 200 years. Fashions come and go and smoking has been in and out of fashion many times in that period. In the early years of white settlement most men in Australia smoked, but very few women did in the days before cigarettes. There have always been movements against tobacco and it is surprising how early the reason for attacking the habit was to improve the smoker's health. This occurred long before most doctors were willing to take sides. That had to wait until the 1970s.

Australia was a long slow journey from either Europe or America until the steam ship became common, so it was not surprising that enterprising farmers started to grow tobacco to take advantage of the various periods of shortage when imports were late. Unfortunately the tobacco was of poor quality so had to be cheap. State governments encouraged tobacco farming, as a method of improving the small farmers' profits and as a source of income from taxation. The introduction of cigarettes, which did not require the smoker to own a pipe, led to increased consumption, especially among young men and boys. The anti-tobacco advertising compared the undersized, perhaps consumptive, fag smoking boy with the healthy, fit and larger non-smoker of the same age.

Sometimes social pressures reduced smoking, as during the depression when many could not afford to smoke if they wanted to eat. Other times social change, the increasing number of women who started smoking in the 1920s and 1930s, led to increases. All along there were voices crying of the dangers of smoking. The evidence of chronic cough and shortness of breath associated with long term tobacco use was accepted by the thinking lay public long before most doctors accepted that tobacco was bad for human bodies.

Doctors do not come out very favourably from the past. Although there were some completely convinced of the dangers of tobacco, most doctors imitated their contemporaries and smoked as much as the rest of the population. Even when it became clear that lung cancer was largely caused by smoking many doctors continued to smoke. Now very few doctors smoke and most of our population is composed of non-smokers, many of them ex-smokers. The worry now is the high rate of smoking among young women.

For those who want information about why people smoke and why they give up this is the book to read. There are 30 pages of references, three of further reading and a ten page index. This evidence of wide reading and research has produced an interesting, informative and at times enthralling book.

John Knight

Key Words

Book review, drugs, general interest, history, medical conditions and problems.

SPUMS ANNUAL SCIENTIFIC MEETING 1999

The 1999 ASM had two speakers, Alf Brubakk and Richard Moon, who presented papers on the pathology, natural history and treatment decompression illness, largely limited to decompression sickness, some of which have appeared in previous issues.[1-4] In this issue are the papers presenting two radically opposed views on what is required for the successful treatment of decompression illness (DCI). Also printed in this issue is the edited transcript of the panel discussion to discuss, and perhaps formulate, guidelines for the treatment of decompression illness under different circumstances, which was held on the last day of the meeting.

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ALL DIVERS WITH DECOMPRESSION ILLNESS REQUIRE RECOMPRESSION

Richard E Moon

Key Words
Decompression illness, hyperbaric facilities, hyperbaric oxygen, treatment

Untreated decompression illness

The course of untreated decompression illness (DCI) or "the bends" has been documented, by several authors, in large numbers of men exposed to compressed air while excavating tunnels and bridge piers in the 19th century.[1-3] In 1854, Pol and Wattelle reported 16 cases of bends and two deaths in a cohort of 64 compressed air workers.[1] In 1881 Woodward reported 119 cases of bends in caisson workers during the construction of the St. Louis Bridge.[2] There were 14 deaths and two men were permanently crippled. Snell noted that bends pain may take several weeks to resolve.[3]

There is also excellent documentation of the abysmal course of neurological bends in divers.[4,5] In reporting bends in the pearl divers of Broome, Western Australia, Blick observed 200 cases of "divers palsy", of whom 60 died before a doctor could be reached. Of the remaining 140, 11 died, 8 from septicemia due to decubitus ulcers and cystitis and 3 from meningitis. He reported that approximately 10% of the remaining cases were permanently affected with slight paresis. The combined mortality and morbidity in this series was over 40%.

Recompression therapy reduces symptoms and improves outcome

The efficacy of recompression therapy dates back to the 19th century, when anecdotal observation indicated that symptoms of decompression illness in divers who are mildly affected would often resolve when they were recompressed during the following shift.[1,6] The scientific rationale for this was provided by Paul Bert, who showed that decompression caused the formation of bubbles in the blood of experimental animals.[6]

Systematic application of recompression therapy was reported by Moir and Keays.[7-9] Moir did not report a reduction in morbidity, but his report convincingly demonstrated that recompression of compressed air tunnel workers prevented death. In one of the largest case series ever published, Keays demonstrated that recompression would resolve even minor symptoms.[8] Although Keays' series was not randomised, he observed that recompression therapy was more successful than non-recompression treatment.

TABLE 1

SINGLE RECOMPRESSION SUCCESS RATE OF USN OXYGEN TREATMENT TABLES
(from Thalmann).[16]

Source	Cases	Complete Relief	Substantial Relief	Comments
Workman[17]	150	85 %	95.3 % after 2nd treatment	
Erde & Edmonds[18]	106	81 %		
Davis[19]	145	98 %		Altitude DCS
Bayne[20]	50	98 %		
Pearson & Leitch[21]	28	67 %	83 %	
Kizer[22]	157	58 %	83 %	Long delays
Yap[23]	58	50 %	84 %	Mean delay 48 hours
Gray[24]	812	81 %	94 %	
Green[25]	208	96 %		All pain only, USN Table 5
Ball[26]	14	93 % (mild cases)		
	11	36 % (moderate cases)		Many cases with long delays
	24	8 % (severe cases)		
TOTALS	**1,763**	**81 %**		

Since Yarbrough and Behnke demonstrated superior results from the use of recompression therapy while breathing 100% O_2,[10] a number of publications in the modern medical literature have provided evidence of the beneficial effects of recompression therapy (Table 1).[11] An exhaustive retrospective review of published series of cerebral arterial gas embolism revealed that full recovery was significantly more likely with hyperbaric treatment than without (Table 2).[12]

TABLE 2

OUTCOME AFTER TREATMENT OF CEREBRAL ARTERIAL GAS EMBOLISM
Data from Dutka.[12]

Outcome	Hyperbaric treatment		No recompression	
Full Recovery	346	84.2%	74	24.7%
Residual	45	10.9%	63	21.9%
Death	20	4.9%	151	52.4%
TOTALS	**411**	**100.0%**	**288**	**100.0%**

Hyperbaric treatment is associated with a statistically significant improvement in outcome ($\chi 2=266.1$, df = 2, $P < 10^{-50}$).

The long term effects of failure to recompress

In compressed air workers, a group notoriously reluctant to report symptoms of DCI, there has been a high prevalence of bone necrosis.[13,14] It has been suggested that bone necrosis is due to some process other than the long term effects of untreated symptomatic decompression illness. On the other hand, while reported series of compressed air exposure often have a low rate of DCI, anonymous reporting systems indicate that the percentage of workers experiencing symptoms is several fold greater. Kindwall reported a tunnelling project in which the official bends incidence was 1.4%, but during some work shifts 26% of workers anonymously reported symptoms.[14] Long term effects of treated decompression illness in divers is rare,[15] and bone necrosis is considerably less common than in compressed air workers. Thus, it is conceivable that failure to treat clinical cases of DCI might predispose to long-term consequences.

The theory that recompression therapy may not be necessary for some cases of DCI is untested and unproven

It has been suggested that vigorous adjunctive therapy, including fluid resuscitation, surface oxygen and possibly adjunctive medications such as lignocaine, may achieve as good an outcome for some mild cases of decompression illness as recompression therapy. However, this is an untested hypothesis and to date there are no clinical data to support the notion that non-recompression therapy is as good as recompression with oxygen.

Conclusion

Recompression therapy is currently the "gold standard" for treatment of decompression illness. There is thus far no evidence to refute the traditional view, that provided a symptomatic diver presents for therapy in a timely fashion after an episode of decompression illness, recompression therapy should be initiated.

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RECOMPRESSION TREATMENTS SHOULD BE TO A PRESSURE EQUIVALENT TO 18 m DEPTH

Richard E Moon

Key Words
Bubbles, decompression illness, hyperbaric oxygen, treatment

Bubble volume reduction

One therapeutic goal of recompression treatment of decompression illness (DCI) is a reduction of bubble volume. The higher the pressure the smaller will be the bubble volume, but high pressure is accompanied by practical problems of increased complexity and a higher probability of a treatment complication. Complications may include inert gas narcosis, DCI during the decompression phase or oxygen toxicity. Ultimately, the maximum treatment depth is governed by the chamber design. Thus, the choice of an initial treatment pressure must be a trade off between the opposing goals of maximising bubble compression and minimising the risk of treatment. Clinical experience has shown that recompression to a pressure equivalent to 18 m breathing 100% O_2 or to 50 m breathing 20-50% O_2 are safe.

Clinical observation

Until the 1940s a hodge-podge of empirical treatment tables were used. Recompression pressures were initially based arbitrarily upon either the depth of the dive or the pressure at which relief of symptoms occurred. Yarbrough and Behnke observed that in human cases, symptoms usually resolved at treatment pressures of 30 psig (2.04 atmospheres gauge, 20 m, 67.5 ft of sea water or 3 bar) or less, but that this treatment pressure was insufficient to prevent neurological damage in experimental animals.[1] They suggested using a short period of recompression to 165 ft (50 m, 6 bar) followed by the administration of 100% O_2 at 60 ft (18 m, 2.8 bar) and observed that this protocol effected complete relief of symptoms in 49 of 50 divers with bends.[1]

The use of O_2 in the treatment of bends was systematised within the US Navy in the 1960s. The method of administration of O_2 recompression was detailed in three reports.[2-4] Initially, it had been suggested that 100% O_2 should be administered at 33 ft (10 m, 2 bar), with further compression to 60 ft (18 m, 2.8 bar) if relief of symptoms did not occur within 10 minutes. However, based upon initial results, a prescribed trial of therapy at 10 m (2 bar) was abandoned in favour of immediate recompression to 60 ft (18 m, 2.8 bar).[2] The new O_2 tables produced a high rate of success. In his 1965 report, Goodman reported that treatment of bends using 100% O_2 at 60 ft (18 m, 2.8 bar) resolved 72 of 79 cases of DCS. Of those cases receiving a minimum of 30 minutes of oxygen breathing at 60 ft (18 m, 2.8 bar), for a minimum treatment time of 90 minutes, symptoms were relieved in 49 of 50 cases.[2] This led to adoption by the US Navy of Tables 5 and 6. These treatment tables, sometimes with minor variations, remain the standard in most hyperbaric facilities today.[5] Since their design and implementation there has been a large experience and a high degree of clinical success (see Table 1 in Moon[6]).

On the basis of historical experience, for the initial treatment of DCI almost all clinicians now use an initial compression to 18 m (2.8 bar) or deeper. To date, comparative studies of 18 m (2.8 bar) vs shallower initial compression have not been published. The only published data compared 14 m (2.4 bar) and 18 m (2.8 bar) for follow up treatment after initial compression to 18 m (2.8 bar). In this retrospective review, Wilson and colleagues from Melbourne, reported that of 50 divers who received 18 m (2.8 bar) follow up tables, 8 (16%) relapsed, compared with 6 of 15 (40%) whose follow up treatment was at 14 m (2.4 bar) ($P = 0.03$).[7]

Animal studies

Direct observation of intravascular bubbles during recompression therapy for cerebral arterial gas embolism was reported in 1967 by Waite, who observed bubbles in the cerebral circulation via a cranial window, following intracarotid injection of 1-7 ml of air in anaesthetised dogs. During recompression to 165 ft (50 m, 6 bar) he observed that of 6 animals, one had resolution of air at 60 ft (18 m, 2.8 bar), three had resolution at 80 ft (24 m, 3.4 bar) and two had resolution at 100 ft (30 m, 4 bar).[8] Gorman and colleagues observed that after injection of small volumes of air, if pial bubbles did not redistribute spontaneously, they could remain visible even after recompression to 11 bar (100 m).[9] While compression to 11 bar (100 m) or greater is usually impractical these particular data do not support the use of shallow recompression depths.

Measures other than bubble volume may be more appropriate, and to that end a series of experiments were performed in the 1980s at the US Naval Medical Research Institute on anaesthetised dogs with decompression sickness, using somatosensory evoked potential amplitude as the end point of treatment. In one study, the effect of PO_2 on outcome at 120 minutes after treatment was tested by recompressing dogs to 4 atmospheres gauge (5 bar) while breathing either one of a range of gas mixtures (see Fig. 1).[10] Using this short-term end point, the optimum PO_2 appeared to be between 2 and 3 bar (10-18 m).[10] A follow up comparison of two therapeutic PO_2 values, but at different depths, 60 ft (18 m, 2.8 bar) on 100%

oxygen and 66 ft (20 m, 3 bar) on 66% oxygen, failed to find a short term difference in outcome between 2.0 and 2.8 bar (10-18 m),[11] supporting the use of an initial recompression depth of 10-20 m.

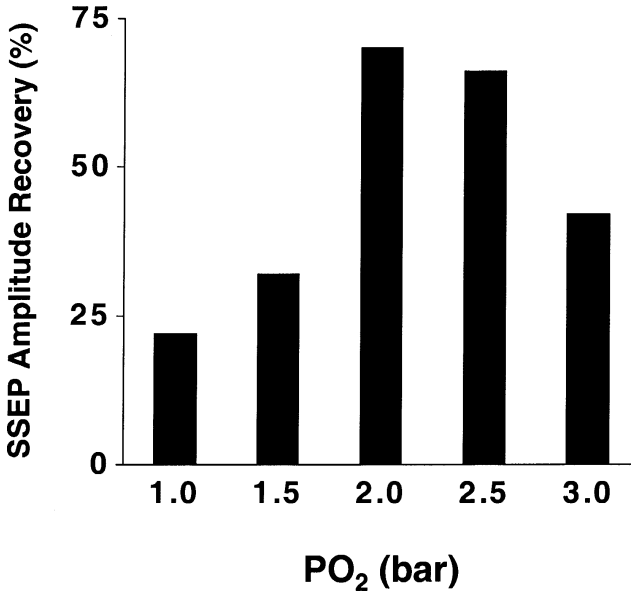

Figure 1. Recovery of somatosensory evoked potential amplitude after treatment of spinal cord decompression sickness in anaesthetised dogs. Therapy was administered at 5 bar, beginning 15 minutes after the onset of impaired neural conduction. The end point was 120 minutes after recompression. The optimum treatment PO_2 according to this model is between 2 and 3 bar. Data from Leitch and Hallenbeck.[10]

Other effects of bubbles

Bubbles have other effects besides mechanical obstruction or distortion. Most theoretical arguments regarding the appropriate pressure and gas composition to use for the treatment of gas bubble disease rest upon analysis of factors that augment gas volume reduction and absorption. This is based upon the thesis that bubbles cause tissue damage by their physical presence, either by occluding blood vessels and inducing ischaemia or, when they occur within the substance of tissue (autochthonous bubbles), by compression and distortion. A powerful argument in favour of this concept is the success of hyperbaric oxygen (HBO_2) in treating these conditions. Evidence has emerged within the last 20 years that bubbles can cause damage via a third mechanism.

Steve Helps and Des Gorman, at the University of Adelaide, embolised anaesthetised rabbits and examined the fate of injected air (25-400 microlitres, compared with 1-7 ml injected in Waite's study[8]) through a cranial window. Surprisingly, the bubbles did not permanently occlude vessels, and usually remained visible for only a few minutes. Despite re-establishment of flow after the bubbles had moved distally, brain blood flow progressively decreased.[12,13] The passage of bubbles appeared to have caused a change in vascular physiology. Later experiments by the same investigators implicated neutrophils, as the blood flow reduction did not occur in animals made neutropenic before the experiment.[14]

These observations and insights provided by experiments on a model of myocutaneous flap ischaemia suggest an additional mechanism for the effect of HBO in gas bubble disease.[15-17] In these studies, blood flow after reperfusion was greater in animals treated with HBO_2 even when it was administered during total ischaemia, when blood flow to the flap was zero. Neutrophils have also been implicated in this process as, in control animals, neutrophils were observed to adhere to the endothelium in the microcirculation of the previously ischaemic flap, but not in animals treated with HBO_2. It has been hypothesised that these neutrophils may cause a reduction in blood flow either by mechanical obstruction to blood flow or by releasing mediators. In the flap model, HBO_2 appeared to inhibit neutrophil-endothelial adherence.

Whether neutrophil-endothelial interaction is important in human DCI is not known. However, studies

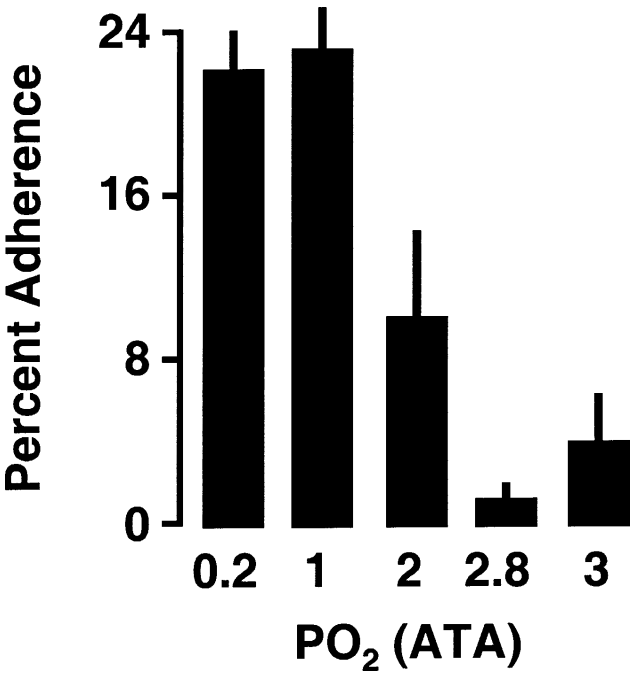

Figure 2. Human neutrophil adherence to nylon columns after 45 minute exposure to different partial pressures of oxygen ranging from 0.2 to 3 atmospheres absolute (ATA) (redrawn from Thom,[18] with permission). Maximum depression of neutrophil adhesion appears to occur at 2.8 ATA (18 breathing 100% O_2).

by Steve Thom in Philadelphia have demonstrated that after volunteers are exposed to elevated PO_2, their neutrophils are less adherent to nylon columns (see Fig. 2).[18] In those studies, maximum depression of neutrophil adhesion occurred at 2.8 ATA (18 m equivalent depth breathing 100% O_2).

The evidence linking inhibition of neutrophil adherence and clinical effectiveness of hyperbaric oxygen in DCI is at best indirect, but what information there is supports the use of 18 m tables.

Summary

It is conceivable that, under some circumstances, shallower recompression depths, or even surface treatment, may achieve a similar degree of success. But, because 18 m recompression is so successful, the burden of proof remains on the side of individuals suggesting a change in therapy. I conclude that the weight of clinical experience and some new insights into pathophysiology suggest that 18 m should remain the preferred depth for treatment of divers until proven otherwise.

AUDIENCE PARTICIPATION

David Doolette, Adelaide

In your last slide, the neutrophils were from normal volunteers. Have there been any studies from neutrophils that have been irritated with air bubbles beforehand?

Richard Moon

Not that I know of.

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SURFACE OXYGEN IS AN ACCEPTABLE DEFINITIVE TREATMENT

Alf Brubakk

Key Words
Bubbles, decompression illness, oxygen, treatment.

Introduction

Richard Moon has presented why he believes that decompression illness (DCI) should always be treated by recompression to 18 m. We were ask to present two radically opposed views on what is required for the successful treatment of DCI. Here I will give a background on why I think there is a place for less than conventional and optimal therapy of decompression illness; optimal treatment still being the use of USN 6 at a well equipped and staffed treatment facility.[1]

Decompression illness can strike anytime and at any place, even with strict adherence to the decompression schedules. Many factors not associated with depth and bottom time can lead to this. For the divers in the audience it is worth citing something about the risk factors, written in 1876 by Snell,[2] who was in charge of people doing caisson work. "Fullness of habit; age; grey hair; exercise after decompression and alcohol abuse." I think the only risk factor that we do not have here in Layang Layang is exercise. The point about this is to make us remember that a large number of divers are at risk, often far away from any proper treatment facility.

There is probably general agreement that the symptoms of DCI are caused by the presence of free gas. The actual symptoms are of course dependent on where the bubbles are located. If the symptoms are minor, like skin itches or pain in a shoulder, this could be a sign that a severe problem might evolve or it may be a single symptom. If local bubbles in the shoulder is the only problem, then it probably is not very serious to have some small area of necrosis in the joint, if there are no other bubbles present. I want to point out here that, in my opinion, it is not likely that there will be bubbles only where there are symptoms. Bubbles can probably form in the venous system at any supersaturation[3] and several studies have shown that 85–90% of individuals with signs of musculo-skeletal decompression sickness also have other clinical signs, mostly from the central nervous system.[4,5]

However, the important question is, in my opinion:

What is the risk of serious sequelae after decompression illness following non-standard treatment of a single or a few incidents ?

Sequelae after DCI

We do not know much about the natural course of DCI. However Snell said that pains in the limbs did not last more than 5 or 6 weeks and were not followed by any sequelae.[2] We know now that this is probably not correct, because there seems to be a connection between repeated cases of DCI and dysbaric osteonecrosis, whether treated or not.[6,7] Interestingly, DCI paralysis usually also passed off in from one to a few weeks. This was, of course, only when the individuals did not die, which would be not a very good end point for modern diving. The horrendous mortality and morbidity of the early series before adequate decompression and the recompression treatment of decompression sickness was introduced,[8] is not really relevant to modern recreational diving.

Using questionnaires, we studied the habits of Norwegian divers.[9] This study included sports divers, professional air divers as well as saturation divers from the North Sea. However we will here only present the data from the air divers, a total of 1,105 divers or about 63% of the diving population at that time. Figure 1 shows the incidence of treated and unreported decompression problems in these populations. Unreported decompression problems were defined as symptoms, which, had they been reported at the time, would have led to recompression treatment.

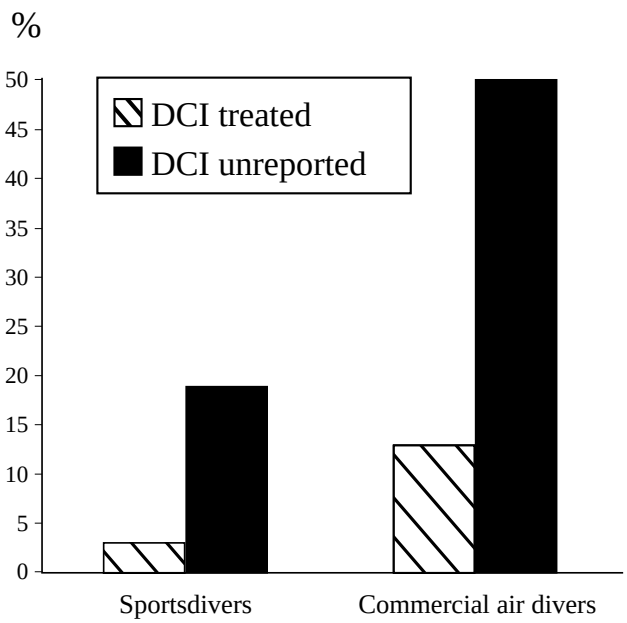

Figure 1. The incidence of treated and unreported DCI in sports- and air divers. Data from[9]

We furthermore used a standard set of questions that have been used for evaluating people who have had slight head trauma or who had been exposed to solvents. Using this questionnaire, approximately 15% of the population will have significant mental symptoms, such as short-term

memory loss, irritability, lack of concentration, or periods of depression.[10]

We will here only concentrate on the air divers, both commercial and sports divers. Our control group, which consisted of a large group of firemen and office workers, as well as the divers who had never had any decompression symptoms, all had approximately the same incidence of such mental problems as can be seen from Fig 2. We can see that the incidence of minor mental problems is similar in both diving groups and not significantly different from that seen in the control groups.

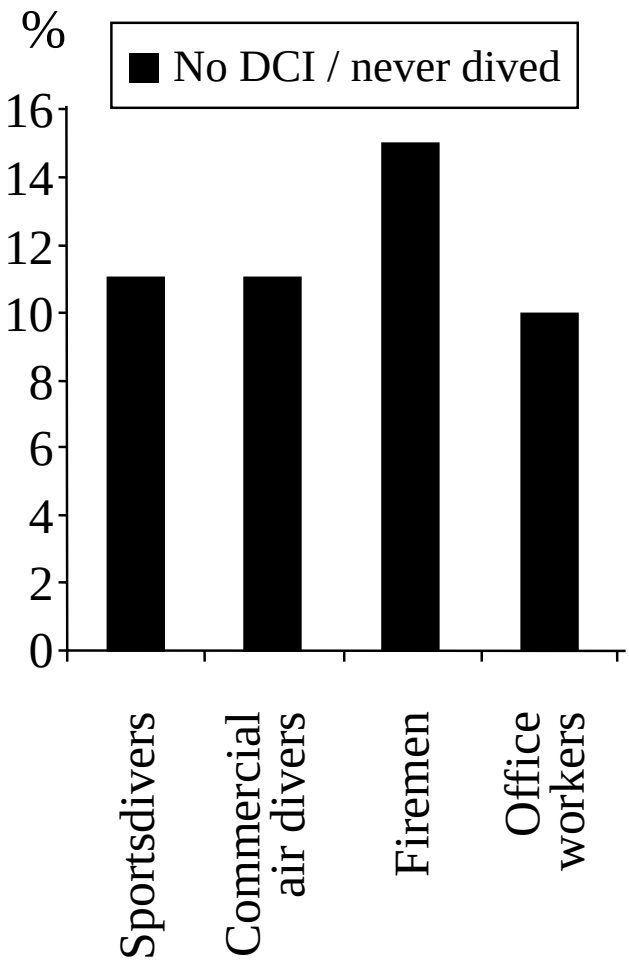

Figure 2. Incidence of CNS problems in divers who never had experienced decompression problems and in controls, Data from[9]

However, as can be seen from Figure 3, there is a relationship between CNS symptoms and unreported decompression problems. Statistical analysis showed that unreported DCI was a significant risk factor for future central nervous problems. This does not mean that these people were seriously handicapped in any way. They were all working and all claimed that they felt healthy.

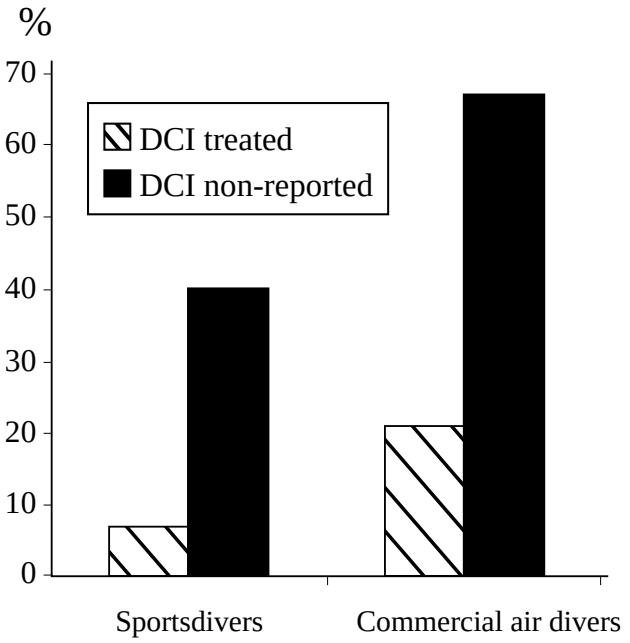

Figure 3. Percentage of divers with CNS problems who had experienced DCI, either treated or untreated. Data from[9]

The important message from this study is that it is important that all cases of decompression sickness are treated in some way, but perhaps also that even if some symptoms are ignored, the consequences are not major.

Different types of DCI may have different treatment urgency.

The idea that there might by different types of decompression illness (sickness), with regard to delay to treatment, is presented in the US Navy Diving Manual,[11] where there are three categories of diving decompression emergencies.

Category A: *Symptoms are severe, involve the inner ear, cardio-respiratory system and central nervous system; or are progressive or relapsing. Instituting treatment in these individuals should be considered an extreme emergency. An evaluation of the patient should not delay treatment or transport. These patients should preferably be treated immediately.*

Category B: *Urgent. The only severe symptom is pain. Symptoms are static, or have progressed slowly over the past few hours. Recompression is as soon as can be arranged, but there is time to conduct a full examination before beginning recompression. It is considered that you have time*.

Category C: *Symptoms are not severe and are not obvious without conducting a detailed examination. Any*

organ system can be affected, but the patient is in no distress. Symptoms are static or progressing slowly over a period of hours. There is time for a complete workover before treatment is started. It is inappropriate to institute recompression without having done this.

This seems to confirm what is clinically well known; that there are many different categories of patients with decompression sickness. The general rule, however, is probably that early treatment will be beneficial in most cases. It is possible that oxygen at surface may be adequate for at least some of the cases in category C.

Remote Locations

A remote location is a dive site that is at least four to six hours away from a chamber facility. Using this definition most dive sites can be considered remote.

In a place far away from a chamber, where it takes a long time to get help, the only medical advice you will get is through a telephone, if you are lucky. In such a situation we are talking about first aid, which would be rest, fluids and 100% oxygen if that is available. Treatment using pressure will be considered elsewhere.

Oxygen

Oxygen at 100 kPa (1 bar) is safe. There is absolutely no data, as far as I know, that shows that oxygen at surface is not safe. Oxygen at pressure is clearly more effective for treating DCI. So what are the advantages of considering 1 bar oxygen as a definite treatment?

Studies have shown that oxygen has a positive effect on symptoms[12] and in many cases the divers presenting at the chamber have no symptoms. As far as I know, there are no studies where divers have been treated with oxygen alone, but anecdotal evidence tells us that a large number of divers have breathed oxygen on the surface for mild symptoms, without ever going on to chamber treatment. It must also be pointed out that there also is anecdotal evidence about divers who have breathed oxygen for some time, but then go on to develop symptoms once oxygen has stopped.

There is no doubt that as oxygen gets more and more common on dive sites, then a large number of individuals will use this as self treatment, whether we like it or not. However, if we insist that all divers breathing oxygen will have to go on to chamber treatment, this will probably mean that a lot of divers will not report their problems, as was clearly documented in the Norwegian study.[9] It is quite ironic that one of the reasons for not reporting problems is that we, as doctors, want perfection, we want to give them the best care possible. Unfortunately that also causes inconveniences for the diver. If he is a professional diver, treatment perhaps means the end of his career. For a sports diver, his holiday will be ruined as may be that of his friends. So there are strong incentives to suppress minor symptoms, or even major symptoms. Divers will deny their symptoms and will go diving the next day. Sometimes that works out alright. Sometimes he develops a problem which cannot be ignored. In our study, about 50% of the unreported symptoms could have originated in the central nervous system.

Unfortunately there are no statistics on how many divers use surface oxygen as definite treatment today. But I believe that if we said that, under certain circumstances, surface oxygen could be regarded as definite treatment, many divers with minor symptoms would use it. If we define surface oxygen as a treatment which, in some cases, could be a definitive treatment, more oxygen would be carried on dive boats. Surface oxygen would be accepted by divers, especially if they think that by using oxygen they could save themselves a long trip to the chamber complex.

Clearly there are disadvantages. There is no doubt that pressure and oxygen is the standard treatment. Some divers will get sub-optimal treatment. There may be a higher incidence of sequelae. Perhaps there will be more divers who have some pathological changes in their body and, maybe, as a result of that, more long term effects, like osteonecrosis and minor cerebral changes. I will maintain, however, that these changes probably are minor. The Norwegian study showed that even without treatment, the consequences of ignoring symptoms may in many cases not be too serious.[9] And even if there are some case histories where osteonecrosis has been observed after a single decompression incident, this must be extremely rare.

Effects of oxygen at 1 bar

Oxygen has a number of effects that are beneficial in treating DCI. One major effect is that it replaces the inert gas in the blood, thereby increasing the gradient for inert gas elimination. Thus bubbles will shrink more rapidly than they would without oxygen.

We have tested this in an experiment where we measured gas bubbles in the pulmonary artery.[13] We dived the pigs to 500 kPa (40 m or 5 bar) for 40 minutes and decompressed them over 2 minutes. That produced a very large number of gas bubbles in most animals. In fact, the amount of bubbles produced proved to be rapidly lethal without treatment. We started treatment at the time of maximum bubble formation after the dive, which was 20 to 30 minutes after surfacing. We used many different treatment protocols; when using oxygen at 100 kPa (1 bar) we continued oxygen breathing until bubbles disappeared, gave a further 30 minutes on oxygen, then switched to air. Figure 4 shows the results.

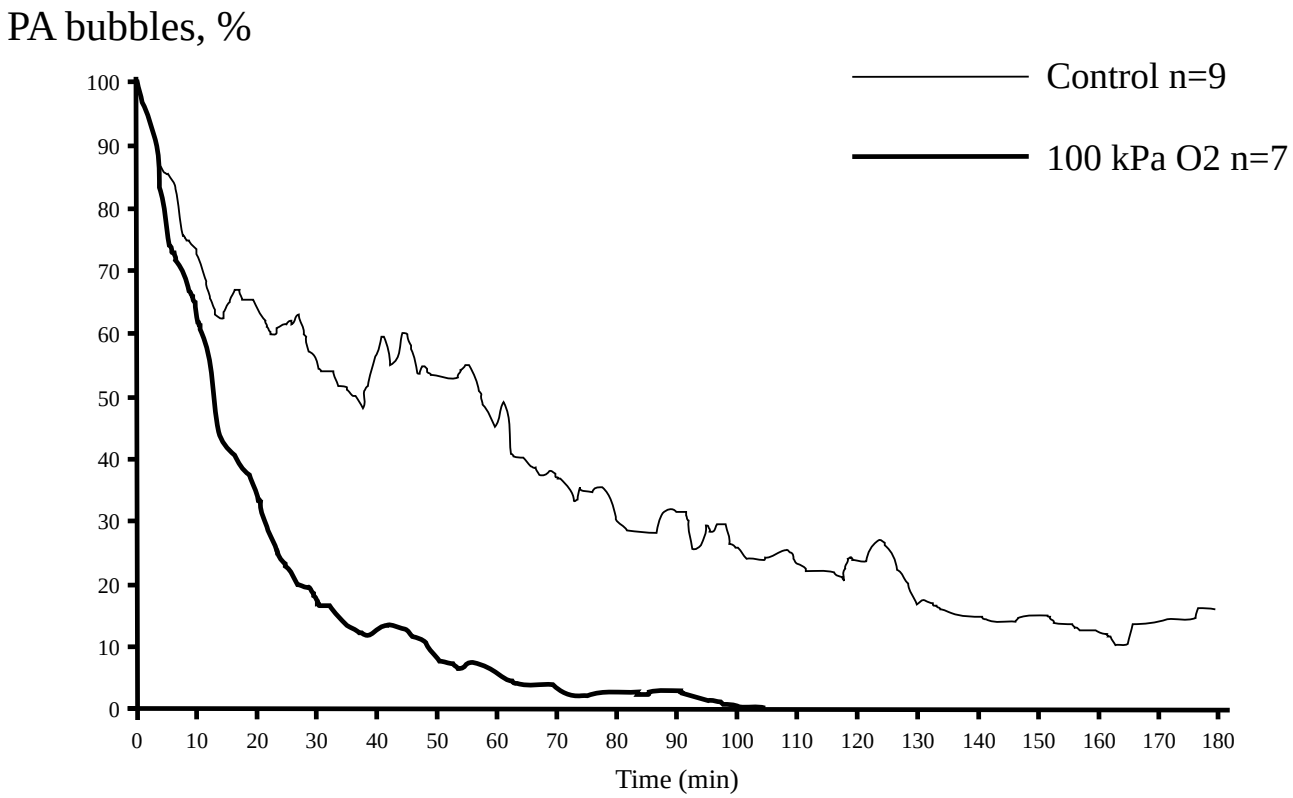

Figure 4. The effect of oxygen breathing on the elimination time of bubbles from the pulmonary artery. Data from[13]

Oxygen treatment at 100 kPa (1 bar), used immediately, is effective in removing bubbles. If no treatment is given, the extrapolation of the control curve will have the bubbles last for about eight hours, while oxygen made them disappear in an average of 74 minutes.[13]

When oxygen treatment was stopped, no further bubbles could be detected. This could indicate that the excess gas had been removed to a degree where no further bubbles could be formed, as can be seen from Fig 5.

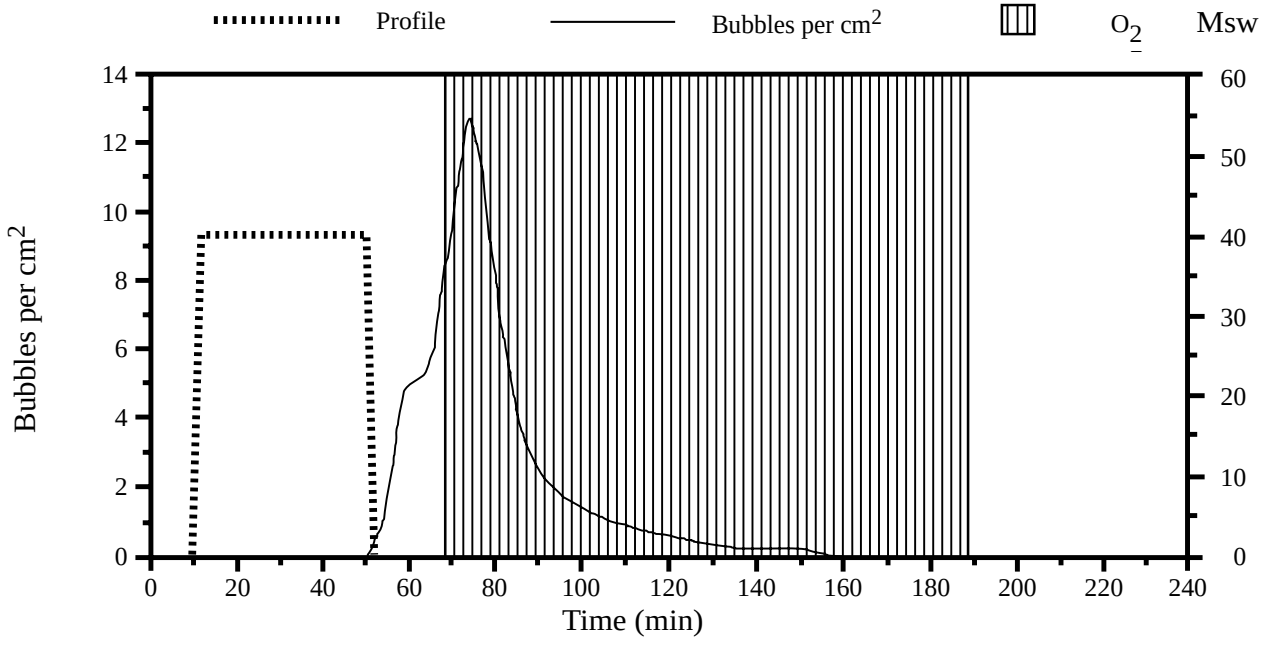

Figure 5. The effect of oxygen breathing on the elimination of gas bubbles from the pulmonary artery. The maximum bubble numbers seen is similar to a Grade 4+ on the Spencer scale. Data from[13]

These animals were kept alive for a week and we closely observed them clinically. None of them developed any sign of decompression illness. At autopsy, no changes could be detected in the brain, the spinal cord, the lungs or the pulmonary endothelium in any of the animals, indicating that at least in this experimental model, the treatment had been remarkably effective.

That means that this treatment, instituted early, and continued for only about 100 minutes, was enough to prevent animals, with a lethal amount of gas bubbles, from dying. Not only did it save them, but it saved them without any sequelae that we could detect with any modern method of histology. This is quite astonishing, but perhaps demonstrates that early treatment, even with 100 kPa (1 bar) oxygen, seems to be quite effective. That does not say that it is just as effective in humans. We do not know that. But at least it gives an indication that the use of only surface oxygen is not totally irresponsible and may be effective even as a definite treatment in some cases.

We assume that tissue oxygen increases as the oxygen tension in inspired air goes up. There is not very much data on measured oxygen tension in tissue, particularly at increased pressure.[14] But if we look at the tension of oxygen in the brain (Figure 6), we see that it is considerably lower than what would be expected from the increase in inspired oxygen, probably due to numerous regulatory mechanisms.

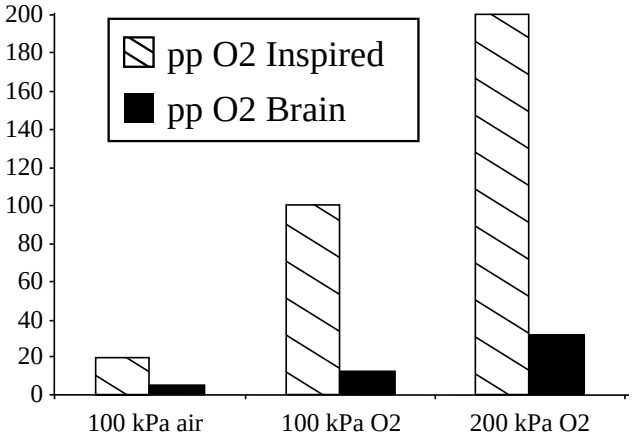

Figure 6. Oxygen tissue tension at various tensions of inspired oxygen. Data from.[14]

Another effect of oxygen, which is usually not considered, is the effect of increasing oxygen tension on shunt blood flow in the lung, blood that goes through the lung without have proper in contact with the alveoli, thus not being properly oxygenated. An increased shunt means that the oxygen tension in arterial blood will be lower than expected. We have demonstrated that the shunt is approximately 8% in a resting animal breathing air, increasing to something in the order of 15-20% when breathing 100% oxygen. Increasing oxygen tension further will further increase the shunt.[15] This means that the effect of increasing oxygen tension is smaller than would be expected from the changes in oxygen tension in the inspired air itself.

Results with oxygen at surface pressure

In Figure 7 you can see the results of treatment with oxygen from the DAN Europe database.[16] The study was published in 1996, and includes individuals who received oxygen before they got to a pressure chamber compared to those who did not get any treatment. Approximately 30% of the divers received some oxygen. On air, there was little change in symptoms, but in the oxygen treated group around 15% improved. But the interesting thing was that 20-25% "healed" during transport. The definition of healed was that the patients had no symptoms when they arrived at the chamber, it is therefore impossible to evaluate the result of the final recompression.

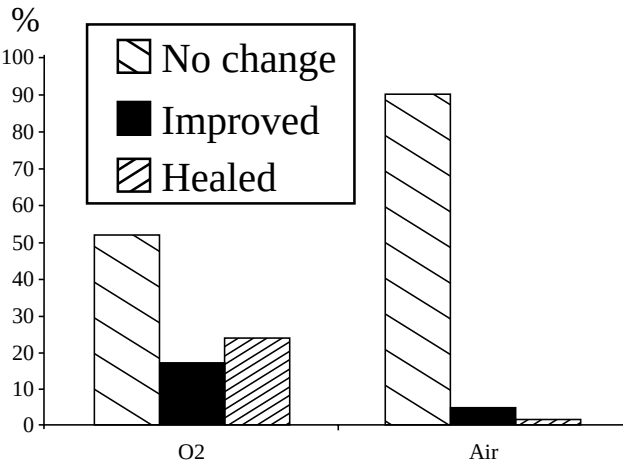

Figure 7. Clinical outcome of oxygen breathing prior to hyperbaric treatment. Data from DAN Europe 1994-95.[16]

What is an acceptable endpoint for treatment ?

An acceptable endpoint for treatment is not easy to define even when we talk about traditional treatment. Usually, treatment is continued until no more symptoms can be seen or there is no further improvement. However, there is virtually no data available on the long term effect of leaving minor symptoms.

What is the risk to the diver's future health if no residual symptoms can be detected following treatment with surface oxygen? If they say "I feel fine, I have no pain any more. I feel OK." after some hours of surface oxygen, I have a feeling that they probably will do alright. I do not know of any data to support the view that they will be at risk. I think that, in patients where no symptoms or signs

were detectable after an oxygen treatment, no further improvement can be expected by subjecting them to pressure. Obviously, if symptoms reoccur, further treatment is indicated.

When can surface oxygen be considered as definite treatment ?

The main indication will be divers who have minor symptoms that respond well to initial treatment with oxygen, where treatment is started immediately and where no recurrence of symptoms can be seen after treatment has stopped.

Other factors may also play a factor in making this decision. One reason would be if there is difficult or dangerous transport to the nearest chamber. There is no point risking the life of someone to get them to treatment. I do not think that is warranted unless you have someone who is dramatically sick or has serious symptoms. Even then they might improve considerably by having surface oxygen. I do not think transport time is significant, as studies indicate that if you do not treat immediately, then time to treatment will not seriously influence the outcome.

Equipment for surface oxygen

If oxygen as a definite treatment is to be considered, then the development of further delivery systems is necessary. The main problem is probably to have an adequate supply of oxygen. If a valve with free flow is used, the oxygen percentage in the inspired air will probably rarely go above 65% and a lot of oxygen is used. A demand valve will reduce the amount of oxygen used considerably. Even better would be closed circuit rebreathers, where only about 50 litres of oxygen will be consumed per hour.

Conclusions

Even in the absence of clinical data, I think there are enough other data to support the use of surface oxygen as a definitive treatment for DCI on a trial basis at remote locations. This can probably best be done by various training agencies and organisations like DAN. Initially this can be done by establishing a reporting routine for those that already practice this.

If we are going to encourage surface oxygen, we also have to consider training of the divers. In particular the people who run dive shops and are in charge of diving activities need to be able to recognise and evaluate symptoms better than they can do today. They have to be able to decide if further treatment is warranted.

Today, many divers are not treated at all. Will they be better off with some surface oxygen?

AUDIENCE PARTICIPATION

Guy Williams

To make things simple and to make a treatment regime easy to follow, would it not be a good idea to add yet another table. We have Tables 1, 2, 3, 4, 5, 6. Perhaps there should be a Table O_2 which says go on 100% oxygen for 4-5 hours, then have an air break, then resume oxygen, air break, with perhaps written underneath, “This table should preferably be used under medical supervision or medical advice”.

Alf Brubakk

That would be an obvious thing. But at present I do not feel that what we are discussing is entirely acceptable. But I agree, we should have a procedure that tells people what to look for and what is acceptable. Some of the questions to be settled are: What is an acceptable endpoint? When does one say “enough is enough”? In what situation does one say “this is good enough”?

Cathy Meehan

In Cairns we have a lot of tourists and the hyperbaric unit is in Townsville, which is 4 hours drive away. Sometimes we put affected tourists on 100% oxygen and use it as a diagnostic tool. If their symptoms do resolve, then we say it is likely to be decompression illness and they need to be recompressed. It would be nice to say they got better and so it is likely to be decompression illness, and that they do not need recompression. But if their symptoms have resolved, what do we say about flying?

Alf Brubakk

I think we should be even more conservative about flying, because, according to everything we think we know, this is a sub-optimal treatment. I think flying after an accident or surface oxygen should be restricted. One should wait longer than normal, perhaps double the time.

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RECOMPRESSION TREATMENT SHOULD ONLY BE ADMINISTERED IN A HOSPITAL-BASED FACILITY

Richard E Moon

Key Words
Decompression illness, hyperbaric facilities, hyperbaric oxygen, treatment

Introduction

The five components of appropriate treatment of a diving casualty with decompression illness (DCI) are:

1 Availability of a skilled practitioner to assess the patient and make the diagnosis;
2 ability to administer initial therapy such as maintaining an airway with adequate ventilation and fluid resuscitation;
3 a treatment chamber in which 100% oxygen can be administered at increased ambient pressure;
4 appropriate procedures (i.e. treatment tables);
5 ability to assess and monitor the patient during treatment.

If all five components are available at the site of the diving accident then, since delay in treatment may involve clinical deterioration, immediate treatment is preferred. The present discussion, however, is in the context of hospital-based treatment where all components are available compared with on-site treatment, in which one or more components are not available.

Assessment requires ideally a physician but at least a person who has had specific training in assessment, treatment and monitoring of diving casualties. In addition to the trained individual, equipment is necessary. A stethoscope, sphygmomanometer, percussion hammer, otoscope, urinary catheter, equipment for administering intravenous fluids and for performing a tube thoracostomy. Ideally one would want a portable X-ray unit.

Therapeutic procedures include treatment tables that have been proven effective in the treatment of decompression illness. The US Navy tables 5 and 6, and their equivalents, have a long track record of efficacy. While shorter treatment tables designed for use in monoplace hyperbaric chambers have efficacy in treating mild or moderate bends, the available data suggest they are less effective in treating severe bends.[1]

Monitoring includes verbal assessment and objective measurement of the progress of treatment. In addition, blood pressure, heart rate and respirations must also be measured particularly in the critically ill individual:

easy in the dry but almost impossible in a one-man chamber or in the water. A patient placed in a single patient chamber would ideally also have the mask seal checked periodically to ensure that the appropriate oxygen concentration is being administered; a semi-conscious or uncooperative patient is unlikely to maintain a proper seal. Urine output is a convenient clinical assessment tool for adequacy of fluid resuscitation, but difficult to assess inside a single man chamber or in-water.

Complications that can occur during treatment include loss of airway, hyperoxic convulsions, pneumothorax, and claustrophobia. A therapeutic plan should include the means to deal with these under adverse conditions, which may be difficult to accomplish during an on-site treatment.

Ways in which the principle of *primum non nocere* can be violated

One way to address this issue is to consider specific scenarios, such as patients with the following manifestations:

Scenario A.
Joint pain and paraesthesias.

Scenario B.
Malaise, monoparesis, unilateral hearing loss and vertigo.

Scenario C.
Paraplegia

Scenario D.
Seizures, unconsciousness.

Scenario E.
Joint pain, funny voice, crackly skin.

These five scenarios represent a range of severity and some diagnostic dilemmas, as follows:

Scenario A.
Does the diver have bends or musculoskeletal injury and anxiety induced hyperventilation?

Scenario B.
Does the diver have inner ear decompression sickness (DCS) or labyrinthine window rupture?

Scenario C.
Does the diver have spinal cord bends or extrinsic cord compression?

Scenario D.
Does the diver have arterial gas embolism or hypoxic encephalopathy due to near-drowning?

Scenario E.
Does the diver, who has symptoms of pneumomediastinum, have a pneumothorax?

In this group of scenarios there is also a range of risks and practical difficulties in placing a patient inside a portable monoplace recompression chamber or administering in-water recompression.

What is the risk of delaying recompression until the patient can be assessed and placed in a hospital-based chamber? The only real down side is the delay. Data obtained from 3,899 decompression accidents reported to the Divers Alert Network from 1989-96 (Figure 1) shows that, while it is clear that delay results in a lower probability of 100% relief, significant improvement may be achieved after several hours or even a day.[2] Severe neurological symptoms include convulsions or abnormalities of vision, gait, urinary/anal sphincter function, motor strength or consciousness.

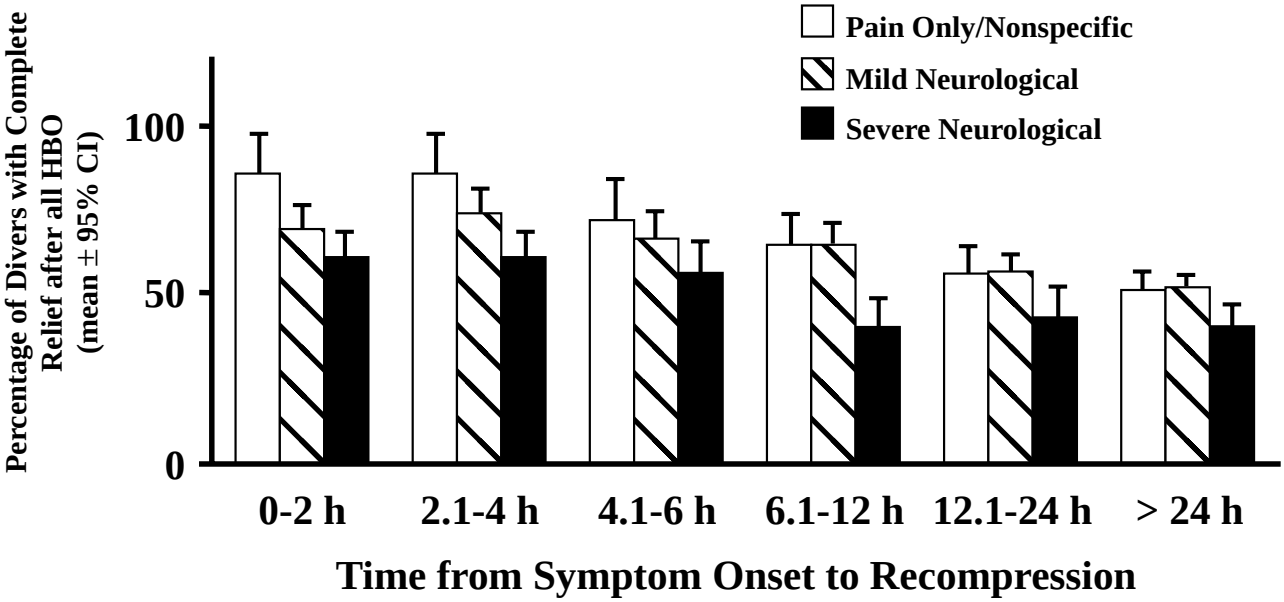

Figure 1. The effect of delay to HBO_2 on results of hyperbaric treatment. Severe neurological symptoms include convulsions or abnormalities of vision, gait, urinary/anal sphincter function, motor strength or consciousness. From Moon et al.[2] with permission.

Considering each scenario individually:

Scenario A.

This patient probably has decompression illness (DCI) and, even if the diagnosis is wrong, the risk of recompression treatment on site would be low. On the other hand, the risk of serious deterioration during transport to a hospital-based chamber is also very low.

Scenario B.

Decompression illness is highly likely here, although without direct inspection middle ear barotrauma cannot be excluded. Furthermore, recompression of a diver with inner barotrauma could be detrimental, as labyrinthine window tear could be worsened if there is difficulty with middle ear pressure equalisation. Even under ideal circumstances, differentiating between inner ear DCI and inner ear barotrauma can be difficult. While the risk/benefit of immediate recompression in this setting is at least debatable, the potential for exacerbating what could produce a permanent disability (hearing loss) must be considered. Anyone other than a trained diving doctor is unlikely to be qualified to make a reasoned decision.

Scenario C .

An acutely paraplegic patient is almost certain to be fluid-depleted and, in addition, to be hypotensive due to the loss of sympathetic tone in the lower extremities. Of the five scenarios this patient is probably the one who deserves the most rapid compression. However, it is arguable that aggressive fluid resuscitation to maintain blood pressure and administration of surface oxygen with delayed recompression might be as effective as immediate recompression without the ability to fluid resuscitate, assess and monitor vital signs.

Scenario D.

A patient with seizures and unconsciousness is likely to require sophisticated airway management, and is unlikely to do well in a portable recompression chamber or during in-water recompression. For this patient delayed treatment in a hospital is inevitable.

Scenario E

It is unlikely that many diving doctors would recommend that a patient experiencing mild bends symptoms, but with obvious clinical evidence of pulmonary barotrauma, should be treated in the water or in a single man chamber, particularly when the means to assess and treat pneumothorax are not available.

In this range of clinical scenarios it can be seen that on-site treatment of individuals with severe decompression illness (those most likely to benefit from early recompression) is accompanied by practical difficulties and real risks. On the other hand, whenever the risk of on-site treatment is low, so is the benefit.

Summary

In an ideal world all divers with decompression illness would receive immediate expert assessment and recompression treatment with or without the necessary adjunctive therapy to maintain blood pressure and ensure appropriate pulmonary gas exchange. However, in recreational diving the ability to administer such prompt and sophisticated therapy rarely exists. While in-water recompression procedures have been available for several years,[3] special equipment is required and there are definite risks associated with its use. Safer alternatives, such as portable recompression chambers, are available. However, the other components that are usually necessary to achieve the desired therapeutic outcome are missing. Specifically, trained individuals, suitable procedures, the ability to monitor the patient appropriately and to administer adjunctive therapy such as airway control and intravenous fluids cannot be applied in these monoplace chambers. Given that excellent results can often be obtained even after many hours' delay, the evidence thus far supports the contention that recompression should only be administered in a hospital-based facility.

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AUDIENCE PARTICIPATION

John Knight

In-water recompression has its place. The person with the crackly voice and the limb pain, will probably get completely better with surface oxygen, 100% oxygen, over the next 3-4 hours. The person with the paraplegia is the one I would put in the water, because he is the one who has the most to gain. If one can get him 3 hours of in-water oxygen and he has his power back, one will save him being a late case with a poor prognosis when he gets to the hospital. Even if he develops an oxygen convulsion he will not come to harm with a full face mask, and the treatment laid down for oxygen convulsions is to pull them up. There is no harm. The main advantage of the in-water oxygen table is it removes a lot of nitrogen as well as providing some pressure to compress bubbles.

The point about in-water oxygen treatment is that it is for places hours from evacuation. In some places it takes 12 hours to fly in and then a 12 hour flight out, plus the time bureaucrats spend messing about organising the flight. Put a paraplegic in the water on oxygen and if he gets movement back in his legs, even if he hasn't got his full power, he is a lot better off than a paraplegic who has not had any movement in his legs for 24 hours before he is flown away to a chamber. In-water oxygen was originally designed for remote areas, with the assumption that ordinary divers would be able to diagnose DCI which needed prompt treatment.

I fully appreciate people's worries about the oxygen toxicity, but a lot of people spent a lot of time trying to find cases where Carl Edmonds' oxygen treatment had caused problems. There has been only one reported case, from Townsville. The person who reported it said he was quite sure it was oxygen toxicity while Carl is quite sure it was salt water aspiration. For about 5 years, the hyperbaric world was looking for cases that had gone wrong and they never found any. Eventually the US Navy was convinced that it was a reasonable thing to do.

I think it would be stupid to do in-water recompression if one can get a patient to a chamber in an hour or four, but if I have to wait 8 hours before I can get a plane to take me to a chamber to get my paraplegia treated, I would be out there with the oxygen, even sitting in the bight of a rope which gets very painful, hoping that the bubbles would be shrunk enough, and enough nitrogen taken out of me, that I will be better when I come to the surface.

Richard Moon

I think that is an arguable point but let me suggest that fluid resuscitation, monitoring and maintenance of blood pressure are as important in treating severe spinal cord injury as immediate recompression. There are few cases of severe neurological bends in which there is no clinical response to recompression, even after a delay.

John Knight

Intelligent divers can make reasonable conclusions from injured divers. With training they can deal with a patient who has a swollen bladder who needs catheterising as the Broome pearl divers did 100 years ago. Part of a diver's kit was his catheter for when he would be paralysed. A lot of diving is done without doctors within cooee, or even much further away. I think that hospital treatment advocates want a perfect world, where medical attention is easy to get and divers report their illnesses early.

But on an outer island off the coast of Australia, there may be 7 or 8 hours to get to port, and a diver is getting worse before ones eyes. What should a diver, not a doctor, do. Divers know that the common cause of paralysis after a dive is decompression sickness. They have got oxygen on board, they have got the full face mask. Would it be a better chance for his future to cure him quickly? We know that if a commercial diver comes up with anything wrong, he is put in a chamber immediately and made better.

Richard Moon

I am not arguing that there should be no treatment before the hospital; I am only arguing the point that it should not necessarily be recompression treatment.

Unidentified speaker

Firstly for John Knight. You have just told us that in water recompression is very safe, that there has only ever been one adverse case reported, and that SPUMS has brought a full face mask and kit. Why is it not here?

Secondly for Richard Moon. Your scenarios have suggested two question marks. Can I put it to you there are two groups of patients, one who would benefit from in-water treatment, and another you, and I agree, definitely would not put back in the water.

John Knight

SPUMS purchased the in-water oxygen equipment in 1977. We took that kit with us to every meeting until Chris Acott became President in 1985. Because Chris Acott is a firm believer that a doctor must run all treatment and only in a chamber we gave up the in-water oxygen equipment.

I do not think SPUMS should be carrying in-water equipment now. At the time, 1977-1984, it seemed a good idea to be able to treat people if anyone developed DCI. We were happy they never did. It was an easy insurance policy with a middle sized bag of equipment and a big cylinder of oxygen.

Richard Moon

I have no experience whatsoever with in-water recompression, but the published data are all self-reported by local fisherman, with no recorded corroborative observations by medically trained individuals. It could be argued that anecdotal reporters are more likely to recount successful cases than unsuccessful ones. A few years ago I asked Carl Edmonds whether there were any cases of in-water recompression in pearl divers (in whom the largest series of in-water recompressions has been collected) that had been documented by a physician. He said he was not aware of any. It may well be that in-water recompression is a good idea, but there are few data on its effectiveness or safety.

Alf Brubakk

I would like to make several points. Even if it is self reported, the majority of cases of in-water recompression have actually been treated with air. It is not just that oxygen is probably more efficient, but it also seems that air is also quite efficient if used with early treatment. I fully agree that, if a hospital is close by, it makes absolutely no sense to

do in-water or on site recompression. You may not be aware that we have evacuated one of our participants from this island, half an hour ago, because of decompression sickness. We treated with fluids and oxygen, the patient has been taken away, and is probably already now in a chamber. Even if we had the in-water recompression kit here, I do not think that anybody would suggest that we should put the patient back in the water. It is clear that in situ treatment is something that one does in a remote location.

There are very good examples from other areas, like in climbing, where some teams now are going very high. Some take some method of over-pressure to deal with mountain sickness. There is no doubt that the ideal treatment is to take the patient down and to fly them out to proper treatment at lower levels in a hospital facility. But the problem is what to do if one is far away? Is it reasonable to believe that results will be as good from treating someone with fluids and oxygen and waiting for 8 or 9 hours, as recompressing the sufferer immediately? We know that even recompression with air, if there is no oxygen at all, is better.

There is no real argument that a hospital is the best place to be with a serious diving accident. On the other hand, if there is no hospital, and no doctors, does that mean one should only use oxygen and fluids on the surface, if you has a compression system available.

Mike Bennett

I think Richard Moon has successfully deflected the focus of the meeting onto in-water recompression, which is indefensible. That is making the case look very good. I would like to ask your comments to the proposition of a functional chamber, outside a hospital. This is an option in New South Wales. There are chambers that we know of where people who are bent could be treated quickly. But chambers are not run by people with medical skills.

Richard Moon

I would support the use of on-site chambers for recompression of selected cases of decompression illness. In the absence of haemodynamic instability and claustrophobia and, this is most important, if there is somebody on site who knows what they are doing, at least a subset of bends could be treated. The first scenario in my presentation I think would fit into that category. But, if the complexity of treatment is even slightly greater, with the necessity of monitoring blood pressure, administering fluids and measuring urine output, I think it is extremely difficult, and perhaps impractical, to treat outside a hospital. I could modify my statement to accept that a subset of bends could be treated with immediate recompression provided a two man chamber is available and a physician who is qualified in diagnosis is on site as well.

Robyn Walker

The Royal Australian Navy sends a portable two-man chamber with all diving teams. I have certainly supervised treatments remotely. The divers have treated in a small chamber. The difficulty is it does not have monitoring capabilities, nor ventilator capabilities. Basically our guidelines are that if there is anyone who is haemodynamically unstable, or an unprotected airway, or altered consciousness, they are not to be put into the chamber until it has been discussed with a diving doctor. For all other accidents, such as a spinal hit with no altered conscious, the diver is treated immediately. They have done very well indeed.

Oxygen fits are unpleasant but safe with the proper equipment. The RAN has divers who dive on oxygen, and a number of them fit from oxygen convulsions in the water every year. The divers call it the “chicken dance”. The divers have a buddy beside them, they have a line, and people survive oxygen convulsions in the water. There is no reason, if one is going to use in-water oxygen recompression, why divers should not survive a fit. One certainly needs to know what to do. Using in-water oxygen treatment at 5 m instead of 9 m, would certainly reduce the risk of oxygen convulsion.

Richard Moon

That is true, but it is likely that the efficacy of treatment would also be reduced at 5 m. Before recommending it, data should be collected on the efficacy of such treatment.

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ON-SITE RECOMPRESSION TREATMENT IS ACCEPTABLE FOR DCI

Alf Brubakk

Key Words

Accidents, decompression illness, treatment.

Introduction

"I think it has been clearly established, that treatment in a recompression chamber by people who are trained and competent probably constitutes the best scenario. On the other hand, if that treatment can't be carried out for six or seven hours because of the location of the dive or for any other reasons mentioned today, then transport may not be the best decision for that diver." Overlock 1999.[1]

There is general agreement that treatment of DCI using the USN 6 with oxygen at 18 m is the standard treatment.[2] However, in most parts of the world, the diver is far away from any proper treatment facility for DCI. Pressure chambers are only available on site in commercial operations in parts of the industrial world. Furthermore, many of these chambers are operated by individuals with only limited experience and certainly little medical know-how. Thus, proper treatment and diagnosis is only available to divers after lengthy and often difficult transport. Due to the fact that it is accepted that the time to treatment is important, transport is often performed under dangerous conditions. All the above would indicate that it is well worth exploring if there are other possibilities.

For many years there was a discussion about the advisability of training the average citizen in cardio-pulmonary resuscitation (CPR). The discussion was mostly centred around the problems and the risks to the patient, ignoring the fact that there were few alternative to prevent death of the patient. It is recognised today that even if the treatment performed by a layman is not optimal, it can be of benefit to the patient. This analogy is not perfect in so far as we are in many cases not dealing with a life threatening condition, but still one which may lead to serious morbidity.

Why on-site recompression treatment?

It is accepted that pressure and oxygen are the main ingredients of DCI treatment. Oxygen at the surface is now widely used as a primary treatment for DCI symptoms and data indicate that the use of oxygen will reduce symptoms before definite treatment can be instituted.[3] However, for definite treatment, pressure is also needed, in particular in severe cases. The main point about on-site recompression is to reduce the time between injury and treatment.

What is the result of traditional treatment ?

In a report from the treatment chamber in Barcelona, the majority of the patients arrived after 1-6 hours, but many with a considerable longer delay.[4] Most of the diving was done within one hour's flight of the chamber and many sites were much closer. Even so, the usual time to treatment was quite long. Their results showed that about 30% had mild sequelae and 4-5% had serious sequelae or handicap after the treatment. The results are similar to those seen in many centres, approximately 70% of those who get treated after a 6 hour delay get better or are healed.[5,6] The results can be seen in Figure 1.

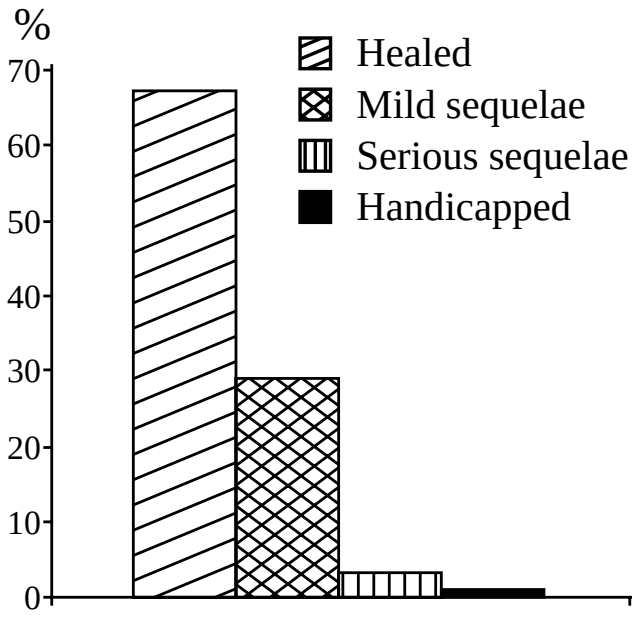

Figure 1. The outcome of treatment in a major treatment centre (Barcelona).[4]

In another study, from Hawaii, the delay to treatment was considerably longer.[7] Here over half the patients had a delay of 12 hours or more before they got to treatment. Regrettably their data are not presented in the same way as that from Barcelona, but rather as improvement in symptoms. Their results show that about 21% of the patients with AGE and 30-35% of the patients with spinal and cerebral DCS had only slight improvement by the treatment. There are always problems with comparing results from different centres, but the results seem to indicate that time may matter, in the sense that a significant number of patients had sequela after finishing treatments.

The conclusion of the study by Desola et al. was that time to treatment is not important, they could not find any correlation with time and the end result.[4] The same conclusions could be drawn from a study by Ross et al. in Aberdeen.[8] In their study of 269 cases of DCI, the median time to treatment was 5.5 hours and 14% had significant sequelae after treatment.

One explanation for these findings could be that after a rather short time period, probably in the order of 30–60 minutes (see later), time is not any longer of major importance for the final outcome, but rather, as Ross et al. point out, the severity of the symptoms.

It is interesting to note that in a much older study, looking at the treatment results after using USN air treatment tables (1A and 2A), the failure rates were 21% and 19% respectively.[9] This eventually led to the abandonment of the air treatment tables.

Why can we expect on-site treatment to give better results ?

This is based on the following hypothesis. Initially, I believe that the mechanical effects of the bubbles are the main problem. Following decompression, there is a time delay before bubbles start to grow, this delay is shorter the more severe the decompression insult. From air dives this delay is typically 20–40 minutes. If recompression is started at this point, then the bubbles will be reduced in size and removed. If however the bubbles are allowed to stay on, the early mechanical effects of the bubbles are no longer reversible, and the secondary effects of the bubbles is now what has to be treated. These could be ischaemia due to vascular obstruction or secondary inflammatory effects set off by the bubble surface or the injury. Once the secondary effects have really started with all their inflammatory processes, then apparently that is still treatable, but the effectiveness of treatment is less, so that the time to further treatment is not particularly critical. Based on this scenario of the pathophysiology of this disease, the time to treatment is the most important factor in determining the outcome of the decompression accident.

There are some clinical experiences to support this scenario. Surface decompression using oxygen is a standard method used in commercial diving all over the world. Using this method, divers are rapidly decompressed and then recompressed within 5 minutes in a deck chamber, usually to 220 kPa (2.2 bar). Studies have shown that this procedure has no higher incidence of DCI than other procedures,[10] while studies both in man[11] and in animals[12] have shown that accepted decompression procedures produce a significant amount of bubbles in the surface period before recompression

During the development of new decompression schedules for the Navies around the world, it is customary to test the procedures in human dives, some of which produce serious decompression sickness. These trials are designed so that the individuals with symptoms are treated immediately and it is the belief of the testing agencies, as expressed in their application for approval to ethics committees, that immediate treatment does not leave any sequelae.

There are also some clinical decompression studies that would seem to support rapid treatment. Ball found that delay in treatment did not influence outcome in mild cases of DCI, but that delays over one hour in the severe cases did.[13] Lam and Yao found. in tunnel workers. that delay increased the depth of relief.[14]

Does rapid recompression give better results?

In animals we performed a study where we recompressed animals to 200 kPa (2 bar) breathing air following a dive to 500 kPa (5 bar) for 40 minutes, decompressing at 200 kPa (2 bar)/minute.[15] The animals were recompressed at the time of maximum bubble formation, 20-40 minutes after surfacing. The animals were kept at pressure until all gas had disappeared then 30 minutes more, after which they were rapidly decompressed to the surface. The animals were observed for a week and then sacrificed. Only one out of seven animals developed symptoms of decompression sickness, at autopsy this animal had a small infarct in the spinal cord, no pathology was detected in the central nervous system, the lungs nor in the endothelium of the pulmonary artery in any of the other animals. The pressure exposure which these animals were given produces a large amount of gas, which in many cases was lethal. We were also very impressed with the effectiveness of treatment in these animals, some of the animals were dying with no respiration and hardly any heart activity at the time of recompression; they immediately improved at pressure and their experience had no long term effect.

This study is supported by the results from in-water recompressions where probably the recompression is in most cases rapidly performed. In a study from Hawaii, 525 divers

were treated, the result of the treatments can be seen in Figure 2.[16] All treatments in this study were performed on air, the difference to the traditional treatment shown in Figure 1 is apparent. A later prospective study on 86 cases, where 94% of the cases were treated on air only, showed a similar trend, but here only 58% were termed asymptomatic after treatment.[17] It must however be pointed out that this last study is severely biased, as nearly all of these cases are divers who sought additional treatment. This may reflect a change in attitude in the diving population, but it is reasonable to assume that a large proportion of those who did not have any symptoms after treatment returned to work.

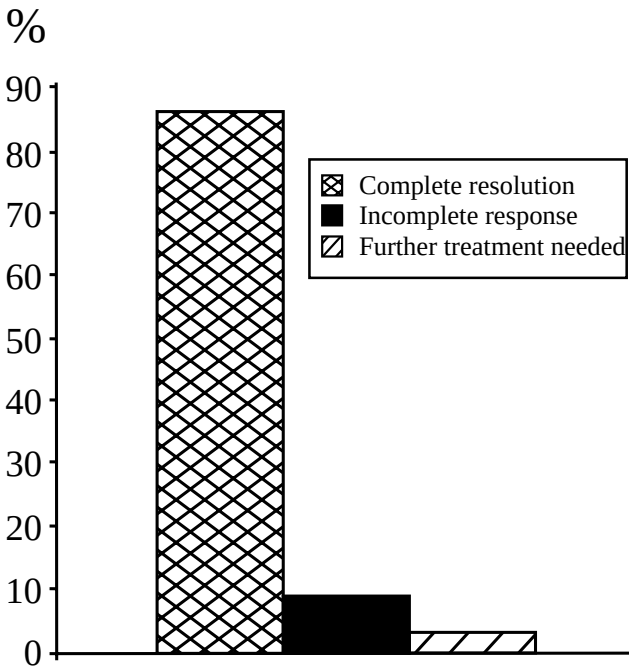

Figure 2. The outcome of in-water treatment.[16]

In the Australian experience, oxygen has mostly been used at 9 metres. According to the report by Edmonds,[18] about 500 cases of DCI was treated with underwater oxygen, only one individual required Medevac and further treatment.

In the Australian oxygen procedures only 9 m treatment depth is used. That this may be adequate for immediate treatment is supported by the study of Koteng et al. who compared the time to disappearance of gas bubbles from the pulmonary artery following recompression on various procedures to 200, 280 and 400 kPa breathing either air, oxygen or a nitrogen or helium/oxygen mix.[19] This can be seen in Figure 3. The addition of pressure increased the time to disappearance significantly, as compared to the use of oxygen on the surface, but there was no difference between the different treatment regimes. A subsequent study showed that the addition of pressure probably did not increase the elimination time for inert gas, this time is only dependent upon the composition of the breathing gas.[20]

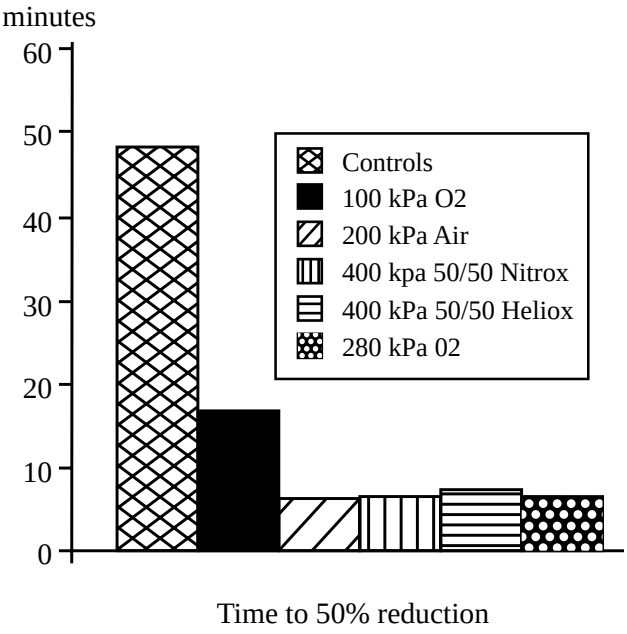

Figure 3. Time from recompression to elimination of 50% of the gas bubbles from the pulmonary artery.[19]

All the above indicate that rapid recompression treatment can by effective, at least as a first-aid measure, and it is reasonable to assume that this procedure can be performed safely and effectively.

On-site treatment options.

Oxygen at the surface has now been recommended for years as a useful first aid for diving accidents. As mentioned above, the data from DAN Europe show that oxygen is effective in relieving symptoms.[3] However, a recent study from DAN USA show that of 179 divers who received oxygen before recompression, 71% experienced complete relief after recompression compared to 64% of the 250 divers who received no oxygen before recompression treatment. This would indicate that the effect of oxygen as first aid on the final outcome was less than could be hoped.[21] It would support the idea that on-site treatment should include pressure.

In planning to use on site treatment, two things are important. First, what kind of equipment are available? Second, what is the danger of performing a treatment, for the patient, the treaters and all those involved.

In-water recompression

Medical experts have had a long, and very heated, debate about whether this treatment modality is acceptable or not. It is important to remember that this is medical first-aid, with the aim of saving the patient's life or reducing his or her risk for permanent damage. As was pointed out above,

all published accounts of this treatment modality indicates that this is a very efficient treatment. Both in the recreational and technical diving community there is a large group of people who simply do it and they do not make much fuss about it. In many cases, I suspect that the treatments are not even reported. That is particularly the case for the so-called technical divers who have the expertise and equipment for performing this procedure. Perhaps that is one of the reasons why the incidence of decompression problems in this community is so low, in spite of some of the extreme diving that is done.

It is now recommended that all in-water recompression is performed on oxygen. The main difference between the Hawaiian and the Australian procedure, is that the former uses a deep spike to depth of relief plus 30 feet (9 m), down to a maximum of 165 feet (50 m).[22] This recommendation is probably based on clinical experience, but has little experimental support, most treatment centres now use a maximum pressure of 280 kPa (2.8 bar corresponding to a depth of 60 feet) for all treatments.[2]

The treatment procedure which was advocated in Australia is at 9 m, usually with a surface supply of oxygen, using a full face mask. One needs a tender, an underwater attendant, a method to control depth, and the Australian Underwater Oxygen table.[23] For mild symptoms 30 minutes at 9 m, then a gradual reduction in pressure of 1 m every 12 minutes (or 1 foot every 4 minutes), if improvement has occurred. If there has been no improvement the patient stays at 9 m for a further 30 minutes before starting the ascent. The total treatment time for mild symptoms is 2 hours 6 minutes to 2 hours 36 minutes in more severe cases. Divers with severe symptoms spend an extra 30 minutes at 9 m and surface at the same rate as those with mild symptoms. Ideally, if the treatment is performed from the shore, one can have the patient moving slowly up the sloping bottom. The reality is probably in many cases not like that. It may be in open water, hanging on a line. It cannot be very easy to follow this table accurately unless one uses 1 m stages.

One potentially serious problem in using oxygen, as described above, is oxygen convulsions. A dive on oxygen to any toxic pressure involves a risk which is difficult to assess. Donald concluded that there is a risk for convulsions from oxygen toxicity in water deeper than 7.5 m (25 ft), that is an oxygen tension of 170 kPa (1.7 bar),[24] or less than that of the treatment tables 190 kPa (1.9 bar). Donald showed that sensitivity to oxygen toxicity of the individual varies considerably over time and that it varies quite a bit between individuals. An individual who has been treated on oxygen on one occasion with no problems, can easily get convulsions with a second treatment. In spite of this, this may be more a potential problem than a real one, as there has to my knowledge been no published reports of such an incident. Due to the seriousness of this complication no in-water recompression should be attempted without the tender being trained in how to handle this problem as it is described in the USN diving manual.[25]

In-water oxygen is not a treatment that should be lightly considered, but it is clearly an alternative, and seems to be very efficient as a first treatment, in many cases even as a definite treatment. One needs, however, to consider whether one has the equipment to and the proper training to do the job.

An important question is of course, will the results justify the risks? The risks are numerous, including convulsions; cold, even in warm water, because the person is sitting motionless in the water for several hours; also dangerous animals have to be considered.

Because of this, I believe one needs a training program if this treatment is going to be used more extensively. One needs personnel who are trained; the patient needs a face mask with oxygen compatibility; there must be some way of keeping absolute depth control. There must be thermal protection. One must have procedures and training to handle convulsions, not an easy matter. Both equipment and training programs are needed for in-water recompression to be a serious alternative.

Single person emergency chambers

An alternative is to use one man chambers. Up till now the alternatives have been rather big and also quite expensive. With the introduction of new materials it should be possible to develop a much simpler, lighter and cheaper type chamber that can be part of any diving operation. Such a chamber, if generally available, would be an alternative to in-water recompression. Figure 4 (page 165) shows one such chamber, which fits into a tube about the size of a golf bag, that was demonstrated at the 1999 Annual Scientfic Meeting. The bag is unrolled, the air supply is plugged in and the patient enters feet first. When all is ready the bag is folded over at the end and a U tube is slid over the folded end to seal it. As can be seen there is an oxygen mask for the patient.

Treatment on land has several advantages. The patient is not in the water, which means that the risk of oxygen convulsions is much lower and the consequences, should it happen much less severe. In Donald's studies it took, approximately, between 2.5 and 5 times longer to get convulsions in the dry than in the water.[26]

In such a chamber, air may be used as a treatment gas if the initial treatment is performed quickly as is described above. Figure 5 shows the result from one of the experimental animals from our study.[15] The amount of bubbles after the initial dive was at the level comparable to Grade 4 + on the Doppler scale, a very severe gas load, that

Figure 4. Portable chamber demonstrated at SPUMS 1999 ASM

in the majority of cases is lethal in pigs. Note that the bubbles disappear quite quickly upon compression, but some bubbles return when the animal is decompressed to the surface, this indicates that the pressure exposure probably was too short.

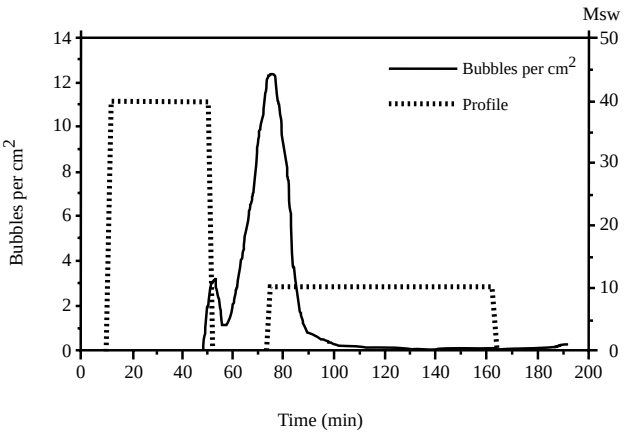

Figure 5. Effect of recompression to 200 kPa (2 bar) on pulmonary artery bubbles.[15]

What are the advantages and disadvantages of on site treatment?

In my opinion, one of the major advantages is that there seems to be better results from immediate treatment. We can avoid immediate transport and can postpone that transport until it is safer or more economical to do so. We have a fully controlled situation, because if we have the capability to treat initially. It is also possible that the on-site treatment, at least in some cases, could be definitive treatment. If the patient is free of all symptoms and signs after the initial treatment, transport by air over long distances is probably not even advisable.

One major advantage of initiating on-site treatment would be that the threshold for reporting symptoms and signs would be lower. The fact that divers often will deny symptoms is well known. We did a survey of Norwegian divers and found that 20% of the sports divers and 60% of the experienced professional divers had had clinical symptoms of decompression sickness without reporting it.[27] If on-site treatment gets recognised as a useful primary treatment, then it is possible that more divers will report problems.

There are also clear disadvantages to introducing such procedures. The most serious one is perhaps that the a significant number of divers will not receive adequate treatment or that it will be postponed. Another is that if the divers know there is a treatment possibility close by, then they may perhaps take more risks. In addition there are of course problems related to the procedures itself, in-water treatment has already been mentioned.

A possible procedure for treatment of DCI in remote areas

On-site recompression is only an option in remote areas. If, however, one defines a remote area as one that is more than six hours away from a proper treatment facility, then most areas in the world would qualify. In 1998, only 20% of the divers in the DAN study were recompressed within six hours.[28]

Oxygen on the surface (if available) is already accepted as a useful first-aid measure, Furthermore, oral fluids are also recommended. Intravenous fluids and drugs may by considered but will obviously require more skill and equipment than we can expect the average dive team to carry.

It is the argument of this paper that pressure should also be considered as an additional treatment option, either using air or oxygen. In order to introduce this possibility there obviously has to be improved training and the introduction of adequate equipment. In particular, we have to train our divers much better in recognising the signs and symptoms of DCI. The on-site option will, in my opinion, be much less effective and useful if many hours have passed since symptoms were detected.

I think, however, that the most important point is to get the medical and diving community to accept that the majority of the diving is done at locations where optimal treatment facilities are not available. Thus, we must be willing to accept solutions that could benefit the patient, solutions that may not be totally adequate from a medical

point of view, but that would improve the end results. Such an acceptance would encourage the production of suitable equipment and the necessary research into the many problems that still exist in this area. I think there is sufficient data to show that on-site treatment is worth further investigation.

A final thought is that there is a lot of commercial diving going on in the Third World that is totally unregulated and where the incidence of DCI is extremely high. These individuals usually have no access to proper treatment facilities and simpler methods of treatment may benefit them considerably.

AUDIENCE PARTICIPATION

John Knight

You asked if any one here had any experience with in-water oxygen treatment. I have not had any experience, but I have carried out, at the SPUMS meeting in 1977, a demonstration of it. We had a victim who was wearing a wetsuit, we had a line, which she sat in, to the diver, and that line was marked in metres. We had a stand by diver on the surface, and we had an attendant diver with her. We put her down, and then we brought her up at 1 m every 12 minutes, a 12 minute pull is very difficult, but a 1 m lift every 12 minutes is easy, which is what we did. We used nine 1 m stages each of 12 minutes. This is a very much slower ascent than any other treatment table (108 minutes for 9 m). In imperial units, one comes up a foot every 4 minutes, which means that the steps are less steep, which may, or may not, be better for avoiding bubbles reforming. Our volunteer complained that her bottom felt that it had been cut in half while sitting in the bight of the rope. If one is going to do in-water recompression one must give the patient a seat to sit on. They need extra weights on their legs, because we found the legs floated up. There have to be at least 2 attendants, a rope tender and an oxygen tender, who can be the supervisor. He has to make sure the oxygen does not run out. The patient can be assessed any time by sending the stand-by diver down, and the attendant comes to the surface and reports the patient's condition.

It was a most useful exercise for SPUMS, using the full face mask, oxygen, etc because we were at Truk Lagoon. The hospital there had a one person chamber, but there was a problem with the gas supplies. The only compressed oxygen normally on the island was what they used for the anaesthetics for women who could not deliver and had to have a Caesarean section. We were so far away from Guam, the nearest USN chamber, that we thought we really should take everything necessary with us, so we really could treat anybody who was unfortunate enough to get decompression illness.

Robyn Walker

Talking about in-water oxygen, we must remember that technical divers are using oxygen at 9 m to decrease their risk of DCI. These guys are out there using oxygen now. There was a technical diving conference in Sydney last weekend, where there was a debate on in-water decompression. Despite having listened to it, I do not believe any consensus came out of that. However, I am told that on the Web site, it was already saying that the consensus of that meeting was that in-water recompression is the way to go. Perhaps we will see more people using it.

Alf Brubakk

Technical divers use in-water oxygen regularly. They use surface oxygen for treatment of symptoms. I am convinced that one of the reasons they report so few symptoms is that they do that. They have the equipment and experience to do it. Whether we like it or not, in-water recompression is here. The question is whether we can do it better, or are there alternatives? But the technical diver is a special breed.

Mike Bennett

Amongst the things that you said, the thing that I did not hear was how I am going to improve the situation around our area. The problem is not transport time. When we look at our figures and delays to treatment, we cannot see a trend where the outcome is worse the longer the wait. That is because we have so few people with very short times to treatment. They are not late because of some problem with distance. They are late is because they diagnose themselves late. Typically, people coming by air ambulance transport, where flight times are an hour to an hour and a half, actually get to the unit 24 to 48 hours after their injury. That is where the problem lies. We are not going to get a chance to treat them on site.

Alf Brubakk

There are several studies which show that there is a long time to reporting. My argument for seriously discussing the possibilities of increasing on site treatment is that I believe it is easier to report symptoms if you know you can be treated on site and not have to have the long transport. I remember some years back, when I talked to someone in the airline industry, where they had a lot of problems with people not reporting errors. The way they solved this was by introducing non-punitive reporting. The reporting has no consequences for the reporter. This means that even if you do something very stupid, everybody just notes that it happened and you and others can learn from it.

In diving it is something very similar. People feel that the rigmarole of treatment and follow up that they start when they report symptoms interferes too much with their lives. I believe that many symptoms would get reported more quickly if they knew they could get treatment and that was the end of it.

I agree that late reporting is a serious problem. Even people who are very experienced, when they start getting

symptoms, they deny them. The best example of denial that I know of was when I was a medical student. We had an excellent Professor of Surgery. For one lecture, he came with a couple of x-rays and gave us a talk on how an x-ray of ulcer could be mistaken for cancer. It was quite obvious, even to a student, that it probably was cancer. They were his own x-rays. He simply denied what was reasonably clear. That was, for me, a clear indication that we have very powerful forces of imagination when things are happening that we do not want to happen.

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PANEL DISCUSSION ON THE TREATMENT OF DECOMPRESSION ILLNESS

Moderator Dr Chris Acott

Panellists
Drs Michael Bennett, Alf Brubakk, Richard Moon and Robyn Walker.
(with audience participation)

Key Words
Decompression illness, treatment.

Moderator (Chris Acott)
What symptoms would the panel treat?

Alf Brubakk

With minor symptoms which do not progress, I do not think there has been anybody who has shown that not treating with recompression leads to serious damage. As we have shown ourselves, non-treatment does, however, lead to mild CNS symptoms. I think there is a considerable under-reporting, minor symptoms are in many cases not treated today. However, if someone has neurological symptoms, these should be treated. I believe that if we insist that everybody should be treated with the standard procedure, a large number of patients will not come forward. I admit that this is perhaps a dangerous statement.

Richard Moon

I think that anyone with symptoms that could be attributable to decompression illness should receive recompression treatment. That would include classical, well defined instances of pain not attributable to other causes, and neurological symptoms. Occasionally it may be worthwhile to treat someone complaining of extreme fatigue.

Mike Bennett

I am pretty much in agreement with Richard Moon there. As many people in this audience are aware, and as we have heard several times over the past few days, the experience of what exactly is decompression illness and who presents can be vastly different in different settings. In most of our recreational diving settings, the patients are, in the vast majority, not extremely seriously bent, in a sense of having dramatic symptoms and signs. Most of them have some subtle signs, but mainly they are complaining of fairly non-specific symptomatology. When we see such people who have not been treated, and we often, perhaps a dozen times a year, see people several weeks after their last dive who have been feeling this way for that time, their lifestyle is seriously affected. They are not happy people. The question of whether, after several weeks, it is worth recompressing them, is not really my point. Actually most of the time we end up recompressing them as an act of desperation as much as anything else. But those people who have apparently fairly trivial signs in our opinion need to be compressed, otherwise they end up with ongoing minor illness, which actually takes up most of their attention, and they do not work well. They continually ring us up to complain about their performance at work and so on. While some sort of one atmosphere oxygen immediately after the dive might have been adequate treatment for their symptoms, we seldom see that situation. When people get to a facility with a recompression chamber and complain that they have had symptoms since diving, then I think they should all be taken seriously.

Robyn Walker

I agree with the others.

Richard Moon

I would like to comment on what Mike Bennett just said. It has been said that only a small proportion of patients who have been treated for decompression illness have long term sequelae, and that most of these are minor. In my experience, the anxiety that is induced by even minor symptoms is extremely important. Divers with ambiguous or minor symptoms may not need to be treated, and if they are treated, the degree of improvement after recompression may be similarly ambiguous. But the fact of their having received the ultimate in treatment, such as a Table 6, means that the patient can be reassured that the bubbles that may have been causing their symptoms, have now gone. This goes a long way toward relieving anxiety.

Chris Acott

It has always appeared slightly illogical to me that we have the same treatment table for a disease which presents in so many different ways, but also from so many different gas loads and diving profiles. However I think Table 6 has been the only table with any data to support using it.

Moderator (Chris Acott)
Does the panel think that in the future we will be able to go towards a strategy of treating a particular illness or gas load with a particular table, or do you think we will just stick with Table 6?

Alf Brubakk

It is a difficult question to answer, because I believe, like you do, that different treatments should probably be used for different patients, or different gas loads, or different symptomatologies. But it is a very difficult job to work out exactly how these differences should be modelled, and the exact procedures to be followed. It would require a lengthy research project to try and find out how to do this. It may be that the results may be marginal; that one would not find firm experimental evidence that actually one procedure is better than another. I think that is particularly likely when the time between the symptoms starting and the start of the treatment is long. In fact I think that the time to treatment may be more important than the procedure used. The damage goes back to a common pathway. The search will be very interesting, but I am not sure that it will result in a very different treatment protocol.

Richard Moon

I think in the very early treatment of decompression illness, it might be possible that the treatment table will be affected by the dive profile preceding it. Consider, for example, a diver on a oil rig who has spent some considerable time at a depth of say 100 m, and then due to a procedural problem, blows up to the surface. For that diver Table 6 may not prevent continuing evolution of inert gas, and for adequate treatment probably a deeper table would be required. But I would submit that after a few hours, at which point the inert gas partial pressures in the tissues and bubble may have reached some quasi-equilibrium, then the major effect of recompression is the pharmacological effect of hyperbaric oxygen, rather than compression of bubbles.

Mike Bennett

I absolutely agree with Richard Moon's and Alf Brubakk's remarks. We think we are giving the same treatment in giving the same table. But of course in many ways the dosage of oxygen we are giving is dependant on the body build of the person. Big people dose themselves up with larger quantities of oxygen by dint of their higher lung volumes. So it is not true that everybody is getting exactly the same. However, if we think of it in terms of partial pressures they are. I think the most important point is that we are dealing with late changes, and bubbles are bubbles and they produce the kind of changes that Richard Moon waxed so eloquently about the other day.

Robyn Walker

One of the interesting clinical cases that I have seen, and I still do not understand, is why someone who presents after embolising in a swimming pool at a depth of 2 m, and is in the chamber within 35 minutes, does not have any recovery of a paralysed limb. Yet, the people who present with lots of these vague but constitutional symptoms, even two weeks afterwards, get a fabulous improvement. I still do not think we have the answers about what we are treating, or the question of what it is that we are treating, to be able to work out what is the most appropriate table.

Moderator (Chris Acott)

To digress a little bit, earlier Alf Brubakk was speaking of teaching diving and physiology. On our boat this week there has been some discussion other than what we saw on the dive. We discussed whether it would be better to teach divers how to read a particular table or whether we should teach them diving physiology, and in particular decompression physiology, so that they could then go and look at a table and have a good understanding of how to read the table or of what their computer can do, and dive accordingly. Would you like to comment on that Terry?

Terry Cummins

One of the things that we have noticed on the boat that we were on, and I assume it was much the same on the other boats, was the general lack of comparing a dive table with the computer. This stimulated some discussion on our boat. From a training agency perspective, we really would like to see the divers checking the computer against the table more regularly than we do. We sampled our boat, and there were only two people who had a table with them on the trip. I think that this is an appropriate observation. We are very solidly into promoting the use of the dive planner with PADI. We also think people are relying too heavily on computers without understanding the physiology and decompression theory.

Robyn Walker

Terry, can I just say that there are a lot of people who do have experience with tables, and to plan a multi-level dive using standard tables can be very difficult. I think a lot of people have, in the back of their mind, that square wave profile, and they know and have an understanding of where they are in relation to a particular a square dive profile. I hope they do.

Unidentified speaker

I think I have seen published guidelines for diving with a dive computer. One sees people diving with a computer, who seem to disregard normal diving practice. They are following what the computer says, but going deeper at the end of a dive, or doing essentially what we used to call two or three dives. Without quite breaking the surface, they will start deep and work their way up and then go deep again. It is probably something for which guidelines should be more widely published, for what you do if you are going to do computer assisted diving. There are some typical, normal things, such as starting deeper and progressively going shallower, which one does if one learns tables, but perhaps forget once the computer is strapped on.

Moderator (Chris Acott)

Yes, a lot of divers that I have seen have done it. I have spent some time in Outpatients discussing their dives. It is quite important to ask why they did a shallow dive first, followed by a lot deeper dive and then a shallow dive later. The usual response is "My computer lets me do it". There is no understanding of what they are actually theoretically doing. Mike, would you like to comment?

Mike Bennett

The first comment that I would like to make is that it is a quirk of statistical fate that both these sets of tables were on the same boat. I did not see any tables on mine.

I approach our divers in the same way as Chris. One gets some extraordinary responses. The impression I get is that whatever people are taught about diving physiology and tables in courses is going to be forgotten soon after a computer is bought. "The computer told me I could do it so I should not be here" is the usual response.

Alf Brubakk

We are working in our laboratory on different models that we can give students and people who dive so that they can actually see some of the consequences of all the different types of tables and behaviour on bubble formation. I think a thing like that, if developed, would be very useful, as then one can demonstrate graphically some of the consequences of a particular type of behaviour. A lot of the teaching of divers is too theoretical. One needs to be able to visualise the lessons in a better way. We need some better teaching tools.

Unidentified speaker

One of my dive buddies and I were writing a dive plan. He has dive planning software for trimix diving. The program included bubble evolution and a graphical display. We were both quite surprised when we put in some poor diving practice, like doing a shallow and then a deep dive, to see how it affected the bubbles on the graphical display. It may not mean anything, but shallow dive followed by a deep one shows a lot more bubble formation than a deep dive followed by a shallow one. As Alf Brubakk suggested, seeing the graphics on the screen, even though we understood the physics, made us believe it a bit better.

Richard Moon

The suggestion to place ultrasound machines on dive boats is a good one. It would be an excellent way of bringing home to divers the message that bubbles do form even after routine, uneventful dives. Perhaps that might in some way influence their behaviour.

Alf Brubakk

It would probably scare them !

Drew Richardson

Just a few comments. In terms of published recommendations, there are several sources for recommendation in terms of diving with a computer which have been out for a number of years. DAN have them; PADI and other training groups have them. Every computer manufacturer puts them in the instruction manual. But reading and acting on them is a different matter.

Using a community or peer approach would be a way to address this topic in the future. If the diving community itself, and on the boat, in discreet ways could take each other aside and say "Look, I wouldn't have done what you did. Did you realise?" Maybe that is a way to keep diving safety in their minds. This is the top of the drawer here in this room. Some have expressed concern about what was observed this week. In public education it is difficult to get people to make the right choices. The question is whether it is because of ignorance or intention. One never hears anybody talking about what they did during the dive. We all speak about what we saw. The divers' desire to see or chase an animal seems to overpower the intelligence needed to decrease risks. I just throw that out in terms of perhaps more community interaction over the course of a diver's career.

Moderator (Chris Acott)

If we perhaps put in more preventative measures, we would not have to talk about treatment. Perhaps SPUMS will do that at another time.

About training divers in first aid and recognition of the problems associated with diving. At the Royal Adelaide Hospital we are one of the few courses in the world recognised by the Health and Safety Executive of the UK (HSE) for the training of Diver Medical Technicians (DMTs) for the commercial diving industry.

To answer some of Dr Brubakk's questions from my clinical experience. Can we train them to clinically recognise that they have a problem? I think we can, very much so. Can we train them to evaluate an outcome? I am not sure of that. Handling complications? I would say no. The use of drugs and intravenous fluids? Yes. As you know, DMTs are our eyes and ears on the diving platform. Perhaps we really should be looking at training the majority of diving instructors up to the DMT levels. Maybe that is a pie in the sky. Alf, would you like to comment on that?

Alf Brubakk

It is quite obvious that your suggestion would be an improvement. I do not know if that would be possible or practical. It would need a change of attitude and acceptance that medically unqualified people will have to do work that is normally regarded as requiring medical qualifications. Because there are not enough doctors to do

it. It is our responsibility to train them to a level where they can do this safely and feel confident enough to do the right thing. Confidence is important, because in many cases people dare not do the right thing because they simply have not been trained to feel that it is right.

Richard Moon

I think the primary responsibility should be in prevention. Our data suggest that a large percentage of individuals with decompression illness have had some problem with their diving procedures, such as ascent rate.

It would be fantastic if we could train our diving instructors to DMT level.

Robyn Walker

Unfortunately it is difficult to obtain continuing education for them. It is not good practice to have people do a course and then not have regular follow up or regular exposure or updating of that experience.

Moderator (Chris Acott)

In the commercial industry, the DMTs are required to have a refresher course every 3 years. In our courses we teach the first timers and use the same time to refresher the others. They spend a week upgrading their practical skills in our Hospital. Unfortunately, we have been unable to persuade any other hospitals in Australia to follow our example.

John Knight

Robyn has said most of what I wanted to say. We are dealing with a relatively infrequent occurrence and very few people in a short while, say a year, will see more than perhaps, if they are very unlucky, 3 or 4 cases. The reason that the MICA ambulance people are so good at their job, is they see those cases every day, and they can keep their skills up. We should be offering to teach these people the skills. We will just have to hope that their memory is about as good as the junior doctor's memory, and when something that they have never seen before but have been told about comes up, there is about a 60-70% chance that they will do the right thing.

Moderator (Chris Acott)

In closing, Alf and Richard have covered quite a lot of the things which will appear in the SPUMS Journal at a later date. Table 1 shows some conclusions we have agreed upon about the acceptibility of various treatments.

Alf Brubakk

USN Table 6 is the only one that has had reasonable clinical testing so it is the basis of all treatment procedures.

Moderator (Chris Acott)

If one has a patient on Table 6, who has not got better, or deteriorates during decompression, is going deeper the answer? Or should say he got better at 18 m, so let us keep him there and saturate him? Or should one continue decompressing and hope that extra treatments in the following days will do the job?

Alf Brubakk

I do not think there is enough data to support one over the other. In this case there is no standard treatment.

TABLE 1

TOPICS DISCUSSED BY THE PANEL AND AUDIENCE

Accepted treatments	Possibly efficacy	Not accepted
Recompression using USN TT6 is the only definitive treatment with enough data to support routine use.	IV administration of lignocaine in "cardiac" doses in severe neurological DCI (where appropriate equipment/monitoring exists).	Breathing air at 1 ATA.
There is consensus for the administration of fluids to restore hydration.	Recompression procedures other than USN TT6, e.g. deeper schedule, heliox.	In-water air recompression
There is consensus for keeping the patient flat in the supine or lateral position prior to recompression in early onset neurological cases.	Saturation recompression schedules (but require special facilities)	High-dose steroid administration
Data supports the use of surface oxygen (as close to 100% as possible) .	Non-steroidal anti-inflammatory drugs including aspirin	
	On-site recompression in a chamber (Accepted by some)	In-water oxygen recompression (Accepted by some)

When one has to tackle those who do not respond or who get worse, then it depends on the experience of the people at the treatment centre. Sometimes they will try going deeper, sometimes saturation.

Richard Moon

I agree. All of these possibilities are legitimate options. Under various circumstances, one might choose any one of them. For example, if you are on a remote island with only a small deck recompression chamber, surfacing may be the only viable option. On the other hand if you have all of the facilities available in Adelaide, you might want to institute saturation. It is difficult to insert too many details into guidelines, without taking into consideration the wide variety of circumstances under which they may be used. However, it is reasonable to elucidate the various options.

Moderator (Chris Acott)

It all comes back to clinical "I've been there, done that" as to what works.

Mike Bennett

I would suggest that in our statement, our policy, we do mention all those options, exactly the framework that Richard suggests. We all agree fluids are extremely important, and there is adequate data to support that, whether it be intravenous or oral.

I know Richard is in favour of steroids, but whenever steroids are used it reminds me of what one of my teachers in medical school used to say: "If you do not know how to treat it, use steroids".

Richard Moon

I would not like to leave the wrong impression. My feeling on steroids is that I would personally use them, knowing full well that they will, in some patients, induce hyperglycaemia. There are many divers with glucose intolerance, and there is strong evidence that in the setting of CNS injury, hyperglycaemia is bad. If the diver is under medical care within 8 hours, and it is possible to monitor glucose on a frequent basis, then the use of corticosteroids is an option for the diver with serious spinal cord bends. However, other than anecdotal cases, at present there are no data supporting the use of steroids for spinal bends.

Robyn Walker

The only thing in the teaching I received was that no one should be allowed to die before being given steroids. I do not use them routinely.

Mike Bennett

Non-steroidal anti-inflammatories are strictly question mark territory. It was a common practice for some patients when I arrived in Sydney. The patients who did not respond to recompression very well, who had residual symptoms after the first one or 2 recompressions, were often given non-steroidals and told they would feel better. And they often did, but whether that was a placebo effect, we are not sure. So we are doing a controlled trial which will be finished in about a year. We are wondering whether we can break the cycle of minor irritating symptoms which people focus so much on and become so anxious about. Perhaps we can stop the symptoms, perhaps not even modifying the basic disease process very much, but just convincing them that they are going to feel better.

Alf Brubakk

There is some quite interesting experimental evidence which indicates that it might help. That has been done a long time ago. But a question mark, yes.

Moderator (Chris Acott)

Unfortunately I was not here for the first session today, when on-site recompression was discussed. Can the panel enlighten me?

Mike Bennett

As a neutral, I do not think we came to any consensus. The question of on-site recompression became a little bit bound up in whether we are talking about in-water or a chamber. I think we were a little bit more disposed to consider on-site chamber treatment than in-water treatment, as a group, which is why it is in the middle column instead of on the end.

Robyn Walker

I think it depends on the level of equipment. There is a whole range of chambers that one could have on-site. It depends on the level of expertise. It depends on the clinical condition of the patient. In some circumstances, it may be a useful option.

Richard Moon

The question regarding on-site treatment versus later hospital based treatment really depends, exactly as Robyn says, on what kind of on-site treatment one is talking about. Some people are reluctant to recommend in-water recompression, but there should be no reluctance on anybody's part to recommend recompression if one has an on-site diving doctor, plenty of oxygen and at least a 4 foot (1.2 m) diameter deck recompression chamber of sufficient size to accommodate the diver and a skilled tender. Now, between the water and a traditional chamber we have a relatively new option, the one man chamber. Before recommending the use of that device, adequate procedures for dealing with both treatment and complications have to be written. How does one deal with a convulsion? How does one deal with somebody who is hypotensive? All of these issues need to be thought out very carefully before recommending the use of such a chamber.

Alf Brubakk

I fully support that, and I agree that there is a lot of work to be done. It needs proper documentation and procedures in order to make sure one knows what one is

doing. Of course, one of the advantages of these simple solutions is that the consequences if something goes wrong are much less dangerous than they are if someone is convulsing in the water. It is an option that can be useful as an alternative to the in-water treatment, which, as I understand it, is done quite a bit. I know, at least in the technical diving community, a lot of in-water treatment is done today. I feel that if it is possible to do something on land, it is a better option.

Unidentified speaker

One of the great advantages of on-site treatment is, for example what has happened here, that one does not have to worry about getting a pressurised aeroplane or making sure that the aeroplane flies below 1,000 feet. One can just shove the patient in a bag and keep them at one atmosphere, and take the plane to whatever height, and they are breathing oxygen. I like that idea.

Moderator (Chris Acott)

We dealt with saturation earlier in the week and deeper tables, when we were talking about US Table 6. What about using heliox?

Alf Brubakk

My personal opinion is that different gases belong in the question mark area. There are some people who swear by heliox treatment for decompression sickness caused by air bubbles. It is not very well documented that it works. There is no doubt that it works at times, as there are single cases where there have been dramatic improvements in very seriously ill patients. It has to be an option and it is in the US Navy Diving Manual as an option.

Moderator (Chris Acott)

In the 'Not Accepted' column we have; air, 1 at one bar; in-water air recompression; high dose steroids; and in-water oxygen (which is accepted by some).

Pauline Whyte

My first question is, if RN 62 is the only treatment table with enough data to truly support its use, is there any role for a shorter treatment table as a trial of pressure in equivocal cases where the diagnosis of DCI is uncertain? The second question is, with divers who require 2nd and 3rd treatments, is there any role for 80/60/30s, or should they again receive RN 62s?

Richard Moon

I do not think that a "test of pressure" is a very useful concept. Consider the rate of resolution of symptoms of decompression illness treated after a significant delay. Sometimes relief is immediate, but more typically the response may not occur until after two or more oxygen cycles. Therefore one cannot use the response to a short oxygen exposure, or test of pressure, as a diagnostic test for bends. I believe that after clinical evaluation, if decompression illness is believed to be a possibility, then a complete treatment should be administered, irrespective of the response within the first few minutes.

Pauline Whyte

I thought I read in Alert Diver a recommendation for a shorter treatment table in equivocal cases, maybe six months ago.

Richard Moon

There have been articles in the Alert Diver referring to use of USN Table 5.[1-2] In the USN, if the medical officer feels most strongly that the diagnosis is musculoskeletal trauma, rather than bends, but is not entirely sure, and no improvement in symptoms occurs after two oxygen cycles at 18 m, decompression may then be initiated using USN Table 5.

Whether shorter or shallower treatment tables are ever appropriate is a worthy question. Alf has proposed that shorter or shallower tables may be sufficient for some types of bends, but before accepting their routine use, I think more information is needed.

Regarding follow up treatments, the question is entirely open. Operational concerns of the hyperbaric facility usually override any specific recommendation regarding the appropriate table, particularly in view of the relative absence of data. The only information that I know of regarding the choice of follow up treatment table comes from the Alfred Hospital in Melbourne, and was presented at the 1989 UHMS meeting. Their conclusion, using retrospective data, was that after an 18 m follow up table the relapse rate was lower than after a 14 m table.

Unidentified speaker

As David Elliott has said, "a trial of pressure is Table 62".

Moderator (Chris Acott)

That is right. There is certainly a tendency in my unit, and I suspect at other places, for this concept to creep in. We try and squash it every time we hear somebody use the term. We do not do tests of pressure. However, having said that, there are times when there is an election made to treat with a Table RN 61, or US Table 5, for someone with mild pain only symptoms. Again, that is not my personal practice, but some of the other physicians do, and I do not have any data with which to beat them over the head. That is still written in the US Navy Manual.

Richard Moon

There are data supporting the efficacy of US Navy Table 5. Green and colleagues published a paper[3] showing that when used according to US Navy Guidelines, that is to say for pain only, skin or lymphatic bends in which the symptoms resolve within 10 minutes at 18 m, the outcomes after USN Tables 5 and 6 are statistically indistinguishable.

Mike Bennett

I am not convinced that there is such a thing as non-neurological DCI.

Moderator (Chris Acott)

Bob Green, your article was looking at various Tables. You pointed out that USN Table 5 was used inappropriately in quite a number of cases.[4]

Bob Green

Reviewing the RN data, I fully agree that USN 5, if used appropriately, has good results. But in a large number of cases it was used inappropriately. When it is used to treat neurological DCS it has very poor results. My personal feeling is that one should use USN Table 6, and forget about Table 5 because if it is available, it will be used inappropriately from time to time.

Alf Brubakk

That is probably correct. On the other hand, with on-site recompression in remote locations, there might be limited amounts of treatment gas and limited possibilities of running a full Table 6. Should we take into account Kindwall's, and some of the other data which exists, and try to design some protocols to give options for treatment when a full Table 6, which is perhaps the best that we can have, is not possible? Should the patient stay at 18 m as long as possible and then come up, after all the ascent was designed for the tender's safety, or would it be better to go to 9 m or even shallower, and stay longer, because that would save gas and be better than surface oxygen?

Mike Bennett

I would not accept treatment in an on-site recompression chamber if it had not got enough oxygen to complete a Table 6. I would be calling for the nearest plane, as obviously they do not know their job.

Richard Moon

I agree. I think the effort should be convincing people to buy enough oxygen rather than designing tables to get around the system. Just one comment about altering the USN Tables. Remember that the stop at 30 feet/9 m, was designed not for the diver, but for the tender. If the chamber is quickly decompressed to the surface after a prolonged stay at 18 m one may create another case.

Robyn Walker

The only time the RAN would consider using Table 5 is in mass casualties. If one is faced with 40 survivors, who have escaped from a submarine, all with decompression illness, giving a short Table 5 to treat as many people as fast as possible may be better than completing a formal Table 6 and making people wait a considerable time for a place in the chamber.

Michael Loxton

Please confirm that these recommendations are for sport diving only. We are not making any comments relating to commercial or military diving?

Moderator (Chris Acott)

We are only discussing the treatment of recreational divers.

I would like to, on behalf of the Society, thank Alf Brubaak and Richard Moon for some interesting discussions, and Robyn Walker and Michael Bennett for participating in the panel discussions in this session. Thank you very much.

References

1 Thalmann E. Whatever happened to Table 5? *Alert Diver* 1996; September/October: 30

2 Dovenbarger J. Old injuries can complicate the diagnosis when evaluating DCS symptoms. *Alert Diver* 1998; July/August: 26-27

3 Green JW, Tichenor J and Curley MD. Treatment of type 1 decompression Scikness using the US Navy treatment algorithm. *Undersea Biomed Res* 1989;16 (6): 465-470

4 Green RD and Leitch DR. Twenty years of treating decompression sickness. *Aviat Space Environ Med* 1987; 57: 362-366

Speakers who were not mentioned after earlier papers in this section are mentioned below.

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Drew Richardson is Senior Vice-President, Training, Education, Environment and Memberships of PADI Worldwide and President, Diving Science and Technology, Inc.

Dr Pauline Whyte was an Anesthetic and Hyperbaric Unit registrar at the Royal Adelaide Hospital at the time of this panel discussion.

Captain Bob Green, DDHM, RAN Reserve is currently (August 2000) Officer in Charge, Submarine and Underwater Medicine Unit, HMAS PENGUIN.

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GLEANINGS FROM MEDICAL JOURNALS

Cerebral arterial gas embolism in air force ground maintenance crew: a report of two cases

Lee CT.

Aviat Space Environ Med 1999; 70: 698-700

Abstract

Two cases of cerebral arterial gas embolism (CAGE) occurred after a decompression incident involving five maintenance crew during a cabin leakage system test of a Hercules C-130 aircraft. During the incident, the cabin pressure increased to 8 inches Hg (203.2 mmHg, 27 kPa) above atmospheric pressure causing intense pain in the ears of all the crew inside. The system was rapidly depressurised to ground level. After the incident, one of the crew reported chest discomfort and fatigue. The next morning he developed a sensation of numbness in the left hand, with persistence of the earlier symptoms. A second crew member, who only experienced earache and heaviness in the head after the incident, developed retrosternal chest discomfort, restlessness, fatigue and numbness in his left hand the next morning. Both were subsequently referred to a recompression facility 4 days after the incident. Examination by the Diving Medical Officer on duty recorded hemianaesthesia and Grade II middle ear barotrauma as the only abnormalities in both cases. Chest X-rays did not reveal any extra-alveolar gas. Diagnoses of decompression illness were made and both patients recompressed on a RN 62 table. The first case recovered fully after two treatments, and the second one after one treatment. Magnetic resonance imaging (MRI) of the brain and bubble contrast echocardiography performed on the first case 6 months after the incident were reported to be normal. The second case was lost to follow-up. Decompression illness (DCI) generally occurs in occupational groups such as compressed air workers, divers, and astronauts. This is believed to be the first report of DCI occurring among aircraft ground maintenance crew.

Key Words

Air embolism, case reports, cerebral arterial gas embolism, hyperbaric oxygen, treatment

Three cases of spinal decompression sickness treated by US Navy Treatment Table 7

Ito M, Domoto H, Tadano Y and Itoh A.

Aviat Space Environ Med 1999; 70: 141-145

Abstract

For patients of type 2 decompression sickness, recompression therapy using US Navy Treatment Table 6 (TT6) and its extensions is the most common means of treatment. However, some cases are resistant to the recompression therapy, and the outcome of TT6 is not always satisfactory. Although a new table, the US Navy Treatment Table 7 (TT7) was described in 1985 in the US Navy Diving Manual, to date few cases who were treated using TT7 have been reported. Here, we report three cases of spinal decompression sickness who received treatment according to TT7. Two were sports scuba divers and the other a commercial diver. TT7 was applied later than 4 days after onset in all three cases; two patients were remarkably improved during the recompression therapy, while the other improved to a certain extent after additional repetitive TT6. Mild impairment of lung function, probably due to pulmonary oxygen toxicity, was observed on lung function testing in one case. In all cases, after additional TT6 and/or rehabilitation, patients were able to return to active daily living.

Key Words

Case report, decompression illness, hyperbaric oxygen, tables, treatment sequelae.

Round window membrane defect in divers (English translation)

Bohm F and Lessle M.

Laryngorhinootologie 1999;78:169-175

Abstract

The rupture of the round window membrane is a special form of traumatic inner ear deafness. Because of the changing pressure levels, divers are at risk of developing such a membrane rupture, especially if tube function is disturbed. As the popularity of diving as a sport increases, ENT specialists have to deal with diving related problems increasingly frequently. Seven cases of divers are presented in whom a tympanotomy was performed following the diagnosis of a rupture of the round window membrane. The symptoms and intra-operative findings are discussed and the otological and diving literature is reviewed. Following a case report, the pathophysiology, clinical symptoms and differential diagnosis of round window ruptures are discussed controversially. Possible therapeutical consequences are described. None of our patients exhibited the classical triad of deafness, tinnitus, and vertigo as described in the diving literature. The leading symptom in our patients was the loss of hearing; only two patients had vertigo. Tinnitus was found in half of the patients. Intraoperatively a rupture of the round window membrane was presumed in five divers. If disturbance of inner ear function does occur concurrently with diving, a rupture of the round window membrane must be considered. An otological examination must be performed in any diver with a loss of hearing and/or signs of a barotrauma of the middle ear. After differential diagnosis to exclude other possibilities, a tympanotomy to cover the round window membrane should be performed if symptoms persist more than 24 hours.

Key Words

ENT, injury, treatment.