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To publish a journal.
To convene members of the Society annually at a scientific conference.

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1 Anderson T, RAN medical officers' training in underwater medicine. *SPUMS J* 1985; 15 (2): 19-22
2 Lippmann J, Bugg S. The diving emergency handbook. Melbourne: J.L.Publications, 1985

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EDITORIAL

The full impact of the changes which have been occurring over the past decade in the Australian diving scene is still not apparent to many. This issue contains a notification of great significance to the Society, its incorporation. The significant thing is that a change of this nature should have become necessary. It is but one more sign of the creeping intrusion of the threat of litigation, a trust destroying cancer in the guise of bringing justice to those who have suffered some wrong. Unfortunately it is the law rather than justice which wins out. The law is frequently said to be an ass, but it has a savage bite which enjoys a diet of precedents, an unfortunate habit in a world of uncertainties. This this can give rise to a "damned if you do and damned if you don't" situation is easy to illustrate in relation to deciding whether or not someone is medically fit to dive. What degree of safety under what manner of adverse circumstances is to be the decisive factor ?

In absolute terms there is not 100% fatality risk related even to conditions such as asthma, diabetes ,cardiac conditions or epilepsy in diving. However nobody would deny that these conditions would be likely to adversely increase the risk factors in a diver. Standards are dictated by a large number of considerations and rarely by any dispassionate evaluation of the evidence. Standards are a trade off. But in any court of law the standard would be likely to prevail over other factors and no protection would flow from a person declaring their willingness to accept full responsibility for not accepting medical advice if their medical examiner, or diving instructor, allowed them to undertake diving training and they subsequently suffered some misadventure for which their medical condition was adjudged a possible factor. A paper will appear in a later issue which suggests that there could be a case against passing as fit to dive anyone who has not been thoroughly investigated to exclude a cardiac septal defect. The basis for this view is that such a condition can increase the risk of gas bubbles, arising after "no-decompression" (more properly "no decompression stop") dives, reaching the brain. This is absurd as the test is not without risk, which could also lead to litigation.

Medical standards are not constant around the world, as evidenced by the fact that the British Sub-Aqua Club (BS-AC) Medical Advisory Committee has stated that a person with occasional allergic asthma can be permitted to scuba dive but should not diver if they have needed a bronchodilator in the previous 48 hours. Such an opinion would be treated as irresponsible, and possibly legally indefensible, in Australia and New Zealand at present, despite the research basis of the BS-AC opinion. However the BDS-AC does require all its members to obtain regular medical examinations, a situation in advance of that in Australia and New Zealand, if it should be demonstrated that medicals significantly improve diver safety. So much for absolutes.

The Queensland Government's legislation to improve the safety of recreational divers in the Great Barrier Reef area is one consequence of ignorance of the critical factors which are most important for diver survival. Understanding or what one is doing, a status which is most simply called "experience" but which does not necessarily equate with years since certification, appears to be the vital element in survival. This legislation was provoked, it is claimed, by persons who thought it would hurt their competitors and not themselves and now find an inflexible application of rules about to damage their commercial viability without any increase in the level of safety. Readers of Aesop will remember the story of the frogs who sent a petition to Zeus for a king and ended up with a stork which then proceeded to eat them. It seems appropriate to remember that the earl scuba divers were called frogmen.

Now is possibly the last opportunity the diving community will be give to command the high moral ground when faced with the threat of a government considering involving itself in regulating the sport diving industry. The only answer to a claim that safety standards are in need of improvement and that the government knows how this can be achieved by a few simple, non-intrusive regulations, is for the diving organisation to show they have considered safety of such importance that they have already set up and are supporting a scheme to collect information from divers and medical sources concerning all types and severities of diving problems, including those successfully managed. This scheme must have the same confidentiality protocols as govern the Bureau of Air Safety CAIR scheme and while making reports to the diving community must be completely independent. This scheme, it would be noted, communicates with overseas information sources to maximise the dissemination of information concerning dangers and the most appropriate way to reduce such dangers before they develop into major problems.

At the time no such organisation exists but there is good reason to believe that there is a growing awareness that it would serve a valuable function, not only in the politics of survival in face of inflexible bureaucratic rules but as a useful means of making information available. This would be an advance on being limited by having to restrict discussion to those details which some writer has chosen to discuss and comment on. Investigation based on original data is far more likely to be useful than that which is restricted to the examination of the conclusions of someone whose data base is inaccessible. Add to this the selling point that it costs the government nothing and is unlikely to create political storms and one has a product to sell.

Readers will find much of interest in the papers presented here. It is hoped that these will encourage an

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ORIGINAL PAPERS

THE BS-AC '88 TABLES

John Lippmann

Background

A number of factors influenced the BS-AC's decision to replace the RNPL/BS-AC Table. Some of these are:

1 The high level of misunderstanding of decompression procedures amongst users and potential users of the table, and

2 The inherent inflexibility of the table itself. The BS-AC recognised that with the advent of the dive computer, the RNPL/BS-AC Table has become more unattractive to the user. The club wished to have a set of tables that approach the versatility of a computer, and that can comfortably co-exist alongside the computer.

The table designer, Dr Tom Hennessy, has worked alongside Dr Val Hempleman, the designer of the original RNPL model, for many years. Hennessy initially decided to base the new tables on the same decompression model as the RNPL/BS-AC Table, since the model on which that table is based had been tried and tested over a number of years. However, since the RNPL/BS-AC Table has not really had the facility to be used, and hence tested, over series of three or four dives per day, Hennessy had to first ensure that the model could be safely extended to cover these multiple diving situations. He believes that very long dives can produce a similar gas load in the tissues to that produced by multiple repetitive dives and, after receiving some data which indicated that the model might be marginal when used for very long, deep dives, Hennessy decided to modify the model slightly.

The RNPL/BS-AC Table assumes that it is safe to ascend directly to the surface from saturation at 9 m, but this is no longer believed to be true. There has been some evidence that the depth a safe direct ascent from saturation can occur from is around 7 m rather than 9 m. This ascent criterion is included in the BS-AC '88 Tables.

Hennessy also believes that bubbles form after every decompression, and that these bubbles affect the gas uptake and release for each subsequent dive. For example, if a diver who has nitrogen bubbles in his blood or tissues descends on a repetitive dive, the nitrogen in the bubbles is exposed to the entire ambient pressure. So at 10 m, the partial pressure of nitrogen in the bubble is 2 ATA, which is higher than the 1.6 ATA partial pressure of nitrogen at 10 m on an initial dive. This means that a diver may saturate more rapidly during the repetitive dive than during an initial dive of the same depth and duration. The total amount of nitrogen will be a combination of this redissolved nitrogen and the nitrogen already dissolved, as well as the normal uptake of nitrogen delivered by the blood during the new dive. The gas in the bubbles does not redissolve as soon as it is recompressed. It takes a certain depth and time before the gas will redissolve completely, and, only then, will the tissue revert to its normal state where uptake and elimination can be described by the model used for the first dive. Hence, the rates of gas uptake and elimination will alter from dive to dive, and it becomes necessary to treat the second, and subsequent, dives quite differently to the first when trying to predict safe decompression.

Most decompression models assume that gas uptake and elimination occur at the same rate during any dive, and the models assume that this rate is the same on a repetitive dive as it is on a single dive. This may be acceptable if significant bubbling has not occurred within the blood and tissues but, if bubbles are present, they will slow down off-gassing and the rates may differ. The original RNPL model assumes that off-gassing is at 2/3 the rate of uptake, and these new tables also assume an assymmetry in the rate of gas uptake and elimination. Hennessy set out to design a set of tables which become progressively more conservative as the number of dives, depth and duration increases.

The US Navy Tables depict the amount of nitrogen in a diver by a single letter code, the Repetitive Group Designator, which is supposed to represent the nitrogen level in the 120 minute theoretical tissue compartment. The system assumes that, after a surface interval of ten minutes, this tissue compartment has the highest nitrogen load and, therefore, controls the decompression. The code is then used to determine the amount of residual nitrogen still remaining in this theoretical tissue (and, therefore, in our entire body) before a repetitive dive, and the original single dive model is used to predict the decompression for the repetitive dive. In reality it has been shown that on a typical "deepish" dive, seven or eight different absorption rates may play a part in controlling the decompression. The US Navy's approach also assumes that dives which give the same code can be treated identically, whether a short, deep dive or a long, shallow one. It assumes that, because the amount of nitrogen that is theoretically dissolved in this one tissue compartment is the same, the dives can be treated equivalently. Unfortunately, our bodies do not work quite so simply. What is not accounted for is that the distribution of the gas load between the various tissues may be quite different in each of the cases, so simply adding some residual nitrogen to the level in one theoretical tissue, is often not sufficient.

To avoid using a single dive model to predict repetitive dives, Hennessy has created a number of different tables to be used for different dives. In all, the BS-AC '88 Tables

consist of a set of seven separate tables, labelled Table A to Table G.

The first table, Table A, is used for the initial dive. After the dive the diver surfaces with a letter code (the Surfacing Code) which relates to the depth and time of the dive. Following a surface interval, the diver selects a new code (the Current Tissue Code) which relates to the nitrogen load in the tissues after the surface interval, and enters a new table (rather than the original table) which bears the same letter code. The minimum surface interval required to gain credit for off-gassing is 15 minutes, rather than the two hours previously used.

The new tables utilise depth increments of 3 m, rather than 2 m and, instead of giving bottom times, give the time from leaving the surface until arriving at 6 m during the ascent, or at 9 m on dives requiring a 9 m stop. The tables use initial No-Stop Times that are more conservative than those on the RNPL/BS-AC Table.

The BS-AC have not recommended a reduction in the 15 m/minute ascent rate, and have not included a safety stop after all "no-stop" dives, as is done on various other tables. Instead, the BS-AC '88 Tables require that the ascent to 6 m is at a maximum rate of 15 m/minute (which means that it may be slower than 15 m/minute), and the ascent from 6 m to the surface must take one minute (which means a rate of 6 m/minute).

Decompression stops are done at 9 m, 6 m *and at the surface*. It is stressed that a surface interval should in essence be treated as a decompression stop, and a diver's activities should be modified accordingly. No 3 m stops are given as they are too difficult to do successfully when there is wave action. Decompression times increase in increments of one minute, rather than five minutes as in the RNPL/BS-AC Table. The maximum decompression given is 22 minutes.

The BS-AC '88 Tables are presented in a compact, easy-to-read format *and do not require any calculations at all*, Tables A to G are supplied in a non-submersible but water resistant format, and Tables A, B and C are also printed, in an abbreviated form, on a submersible card, which should be carried by the diver and used in the event of a memory lapse or a change of dive plan. Presumably, Tables D to G are not included on the card due to the very restricted No-Stop Dive Times available to a diver with Current Tissue Codes of D to G.

The tables in their current form are presently untested but appear to be conservative when used for *NO-STOP DIVES.*

Comparing the BS-AC '88 tables to some other tables

When the BS-AC '88 Tables are compared to tables such as the US Navy Tables, the Buehlmann (1986) Tables, and the DCIEM Tables some trends appear to emerge. These are:

> The tables appear to be conservative for both single and multiple no-stop dives, with the initial NDLs comparable with those of the Buehlmann (1986) and DCIEM Tables. (Table 1)
>
> For single/initial dives requiring stops, the decompression given is often, but not always, more conservative than that given by the US Navy Tables, but is often less conservative than that suggested by the Buehlmann (1986) and DCIEM Tables.
>
> For repetitive dives requiring stops, the decompression given by the BS-AC '88 Tables is more conservative than that given by the US Navy Tables, and often comparable to that given by the Buehlmann (1986) and DCIEM Tables.

These trends are demonstrated in Table 2 and Figure 1.

Promoters of the BS-AC '88 Tables argue that even though the Total Decompression Time (TDT) given by these tables is sometimes shorter than that given by some other tables, the risk of decompression sickness is not only dependent on TDT. A longer decompression profile is not necessarily a safer one as other factors (procedural parameters) also affect the risk of bends. Some of these parameters are the ascent rate, the depth and duration of the initial stop, the ease of maintaining the depth of the required stops, the surface interval required before diving again (or flying) and the activities during the surface interval.

If one compares the ascent procedure suggested by the BS-AC '88 Tables to that given by the US Navy Tables, there are a number of differences which includes:- a slower ascent rate to 6 m, a longer stay at 6 m, a slower ascent rate from 6 m to the surface (although often a shorter ascent time) and a longer stay at the surface before diving again. Although these comparisons are valid for the US Navy Tables, they do not necessarily apply to other tables. When the BS-AC '88 Tables are compared to the Buehlmann (1986) and DCIEM Tables, especially for first/single dives, the BS-AC Tables often appear less conservative, not only with TDT but also with respect to some of the procedural parameters previously mentioned. Careful examination of Figure 1 will indicate this trend. Hennessy argues that the Buehlmann and DCIEM Tables are often overly conservative, but this is debatable. Although commercial divers may need to minimise decompression time for the sake of efficiency a recreational diver who decides to conduct a dive involving mandatory stops and who has planned the dive properly should have no reason not to use a conservative table to gain any extra security that it may provide.

The US Navy Tables, Buehlmann (1986) Tables and

TABLE 1
COMPARISON OF NO-STOP LIMITS
(Bottom Times)

Notes:
Times are in minutes unless otherwise specified
No-stop bottom time limits for the BS-AC '88 Tables are approximate
The ascent rate used by the US Navy Tables is 18 m (60 ft)/minute
The ascent rate used by the Buehlmann Tables is 10 m (33 ft)/minute
The ascent rate used by the DCIEM Tables is 15 m (50 ft)/minute

Depth feet	Depth m	BS-AC '88	RNPL/BS-AC	Buehlmann (1986)	DCIEM	US Navy
30	9	242	-	400	300	-
40	12	121	137	125	150	200
50	15	73	72	75	75	100
60	18	50	57	51	50	60
70	21	36	38	35	35	50
80	24	28	30	25	25	40
90	27	22	23	20	20	20
100	30	18	20	17	15	25
110	33	15	16	14	12	20
120	36	12	14	12	10	15
130	39	10	11	10	8	10
140	42	9	10	9	7	10

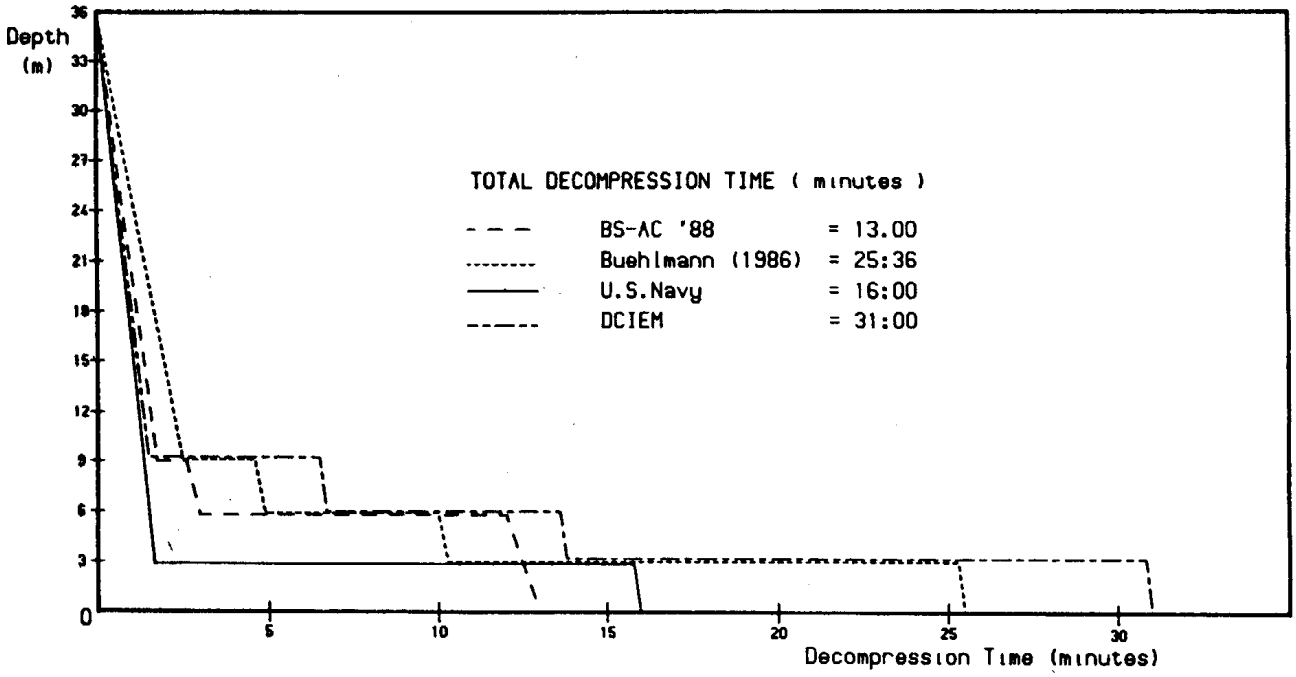

Figure 1 A comparison of the decompression profiles given by various tables for a dive to 36 m (120 ft) for a bottom time of 30 minutes.

TABLE 2

COMPARISON OF VARIOUS DIVE SCHEDULES
WHEN USING
BS-AC '88, US NAVY, BUEHLMANN AND DCIEM TABLES

Times are in minutes unless otherwise specified
No-stop bottom time limits for the BS-AC '88 Tables are approximate
The ascent rate used by the US Navy Tables is 18 m (60 ft)/minute, by the Buehlmann Tables is 10 m (33 ft)/minute and by the DCIEM Tables is 15 m (50 ft)/minute

EXAMPLE A

Dive 1 Max depth 33 m (110 ft) Actual Bottom time 25 minutes

	BS-AC '88	US Navy	Buehlmann	DCIEM
No-Stop (*Bottom* Time) Limit	15	20	14	12
Stops required	3 min at 6 m	3 min at 3 m	2 min at 6 m and 7 min at 3 m	10 min at 6 m and 10 minat 3 m

Surface Interval 2 hours

Dive 2 Max depth 21 m (70 ft) Actual Bottom time 18 minutes

	BS-AC '88	US Navy	Buehlmann	DCIEM
No-Stop (*Bottom* Time) Limit	9	24	23	17
Stops required	1 min at 6 m	None	None	5 min at 3 m

EXAMPLE B

Dive 1 Max depth 27 m (90 ft) Actual Bottom time 20 minutes

	BS-AC '88	US Navy	Buehlmann	DCIEM
No-Stop (*Bottom* Time) Limit	22.5	30	20	20
Stops required	None	None	None	None

Surface Interval 2 hours

Dive 2 Max depth 24 m (80 ft) Actual Bottom time 15 minutes

	BS-AC '88	US Navy	Buehlmann	DCIEM
No-Stop (*Bottom* Time) Limit	6.5	22	14	16
Stops required	3 min at 6 m	None	16 min at 3 m	None

Surface Interval 4 hours

Dive 3 Max depth 18 m (60 ft) Actual Bottom time 40 minutes

	BS-AC '88	US Navy	Buehlmann	DCIEM
No-Stop (*Bottom* Time) Limit	31	43	37	35
Stops required	1 min at 6 m	None	11 min at 3 m	5 min at 3 m

EXAMPLE C

Dive 1 Max depth 36 m (120 ft) Actual Bottom time 12 minutes

	BS-AC '88	US Navy	Buehlmann	DCIEM
No-Stop (*Bottom* Time) Limit	12	15	12	10
Stops required	None	None	None	5 min at 3 m

Surface Interval 1 hour 30 minutes

Dive 2 Max depth 30 m (100 ft) Actual Bottom time 14 minutes

	BS-AC '88	US Navy	Buehlmann	DCIEM
No-Stop (*Bottom* Time) Limit	No no-stop time available	11	8	10
Stops required	3 min at 6 m	3 min at 3 m	5 min at 3 m	10 min at 3 m

Surface Interval 8 hours

Dive 3 Max depth 27 m (90 ft) Actual Bottom time 20 minutes

	BS-AC '88	US Navy	Buehlmann	DCIEM
No-Stop (*Bottom* Time) Limit	13.5	23	20	14
Stops required	1 min at 6 m	None	None	10 min at 3 m

DCIEM Tables have been used for comparison with the BS-AC '88 Tables as they have all had a considerable amount of testing and/or usage. Although the basic model on which the BS-AC '88 Tables are based was tested and was used extensively, *the BS-AC '88 Tables are untested in their current form.*

The BS-AC considered mounting a series of trials using recreational divers, but it was decided that, since the bends incidence was expected to be low, unless a very large number of trials were conducted the results would not be statistically conclusive. The practical and financial constraints of a large test series proved prohibitive so, instead, a 4-month period of informal open-sea dives were conducted by a number of BS-AC members before the tables were released. No details of the profiles conducted and the number of dives have been released, but no cases of bends were reported during the period.

Although essentially untested, the BS-AC '88 Tables appear to be quite conservative for no-stop dives and should generally (but obviously not always) be reasonably safe for such dives. *However, divers who plan to use the BS-AC '88 Tables for dives requiring mandatory decompression stops are urged to do so very cautiously and conservatively as the tables are often less conservative in this area than some well-tested tables. Extensive testing needs to be done before the safety of these tables when used for dives involving mandatory stops is determined.*

A recent BS-AC report states that, in 1989 after the first full season of usage, there were 41 divers who developed bends after diving according to the BS-AC '88 Tables. Eleven of the divers had misused the tables, 22 had dived withn the tables and in the other eight cases there was insufficient information to determine whether the tables had been used correctly. The BS-AC estimate that possibly a million dives could have been conducted using the tables, which would yield an incident rate better than 1 in 10,000.[1] No information is currently available about how many of the bends cases occurred on dives involving mandatory stops and the number that occurred after no-stop dives.

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Recommended Further Reading

British Sub-Aqua Club Diver Training Material - Supplement to Sports Diver and Dive Leader Training Handbook (Lessons ST 6: ST 7: LT 6 using BS-AC '88 decompression tables with theory questions and answers). BS-AC, London, 1988

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AN ATYPICAL EPISODE OF DECOMPRESSION SICKNESS

Hamish Holland

Summary

A case is presented of a novice diver in whom symptoms consistent with decompression sickness developed after diving to a depth of 7 metres of sea water (msw). Resolution followed hyperbaric oxygen therapy.

Introduction

The diagnosis of decompression sickness (DCS) in divers is heavily dependent on the history with considerable weight given to the dive profile. Various "safe" decompression tables are published, and it is generally accepted that symptomatic DCS is extremely rare following exposure to pressures less than 2 ATA.[1]

A case is presented of a novice diver who developed symptoms after shallow training dives, in whom DCS is the only tenable diagnosis.

Case Report

The patient was a 16 year old girl, performing her first training dives in the open sea. The dive series commenced at 1300 hours, and consisted of two dives to a maximum of 7 metres for 15 minutes each, then a 30 minute break and four descents to 4 metres maximum over 40 minutes. The dive profile was confirmed by her instructor, and the dives were uneventful.

By 1800 hours, she reported aching knees and jaw, and a feeling as if her ears were not equalised. The pain continued overnight and was sufficient to disturb her sleep.

The next day, the jaw was easier but her knees had not improved. In addition, she had a headache, pins and needles in both legs, and occasional sharp pains in ankles, wrists, elbows and shoulders. She presented to her local hospital that day, and was transferred to the Royal Darwin Hospital (RDH) 2 days after her dives, arriving at 1330 hours.

On arrival at RDH, she still had aching knees, pain in her shoulders, and an occipital headache, but all other symptoms had resolved.

The patient stated she normally enjoyed good health apart from occasional attacks of tonsillitis. She had not noticed any fever, rash, weakness or lethargy although she had been resting in bed since the day of the dives. She had no abdominal pain, nausea or vomiting, and no urinary symptoms. She comes from an area where ciguatera occurs, and eats a large amount of fish, but no other family members reported any malaise. She has had no previous episodes of ciguatera.

On examination, she proved to be alert and fully orientated, with no nystagmus, no limb weakness, normal tone and no clonus. Reflexes, including plantars, were normal and no sensory loss was detectable. She was afebrile with no skin rash and no lymphadenopathy. Respiratory and cardiovascular examination was normal, as were her eardrums. Her joints displayed a full range of movement and did not show swelling, tenderness or localised warmth.

Chest x-ray was normal. Her haematocrit was 0.43 (normal 0.36-0.47). Over the previous 24 hours she had been allowed a normal oral intake as well as receiving one litre of cystalloid intravenously. She had been kept on 50% oxygen for this time.

It was decided to use hyperbaric oxygen treatment, with table RN 62, as DCS was possible. At the end of the second oxygen period, she still complained of knee pains and headhache but these vanished during the third oxygen period.

Symptoms did not recur and the patient was discharged four days after her dives, and reviewed a week later. Negative results for RA latex, Epstein-Barr virus, Ross River virus, and an autoantibody screen were obtained.

Discussion

The symptoms in this case are consistent with DCS, and did resolve with hyperbaric oxygen, even though the dive does not support this diagnosis. However, no evidence was obtained to support the alternatives of ciguatera, marine envenomation and unrelated polyarthritis. Current theories of DCS support the concept of "silent bubble" formation possibly occuring with any decompression, both intravascularly and in the tissues. The safe depth of 10 msw merely refers to the development of symptoms. It is to be expected that some people will either produce more bubbles, or suffer symptoms with fewer bubbles, than most of the population. This assumption predicts that occasional victims of DCS will be extremely susceptible and reinforces the statistical nature of the dive table. It is impossible to produce a dive table and say DCS will be eliminated by following its guidelines.

Bubble formation in tissues presumably causes symptoms by compression and ischaemia. Intravenous bubble formation (asymptomatic) has been reported after 18% of dives (depths ranging from 6-39 msw), but after 25% of dives deeper than 25 msw.[2] These bubbles are not sufficient to cause symptoms of gas embolisation, however they do cause complement activation in a proportion of the population.[3] The rise in right atrial pressure which follows immersion would encourage transfer of these venous bubbles to the arterial side of the circulation.[4] 37% of a sample of divers with DCS showed right to left shunting through a patent foramen ovale as against 5% of the normal population.[5]

It is apparent that anatomical and physiological factors predispose some divers to the development of DCS and these people should be very cautious about continuing their diving careers.

In spite of resistance from the patient, her family, and the local diving fraternity, she has been advised to accept the diagnosis of DCS and told that in her case, it is not possible to state a time after which further diving will be safe.

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REVIEW OF 1000 SPORTS DIVING MEDICALS

John Parker

Introduction

A diving medical is a medical examination which concludes in a professional opinion whether a person, by reason of their present medical state, is in danger of adversely affecting their health if they scuba dive.

Being a professional opinion it can be challenged. A doctor must be able to justify his decision in light of current medical knowledge and opinion.

No mortality or serious injury is acceptable in diving. Should a person be placed in any risk of this by their present medical state, they must fail.

Should a person be more liable to suffer a minor injury because of their medical state, then they must be forewarned of the dangers and instructed how to avoid such an injury.

The diving medical is necessary to protect:

1. The diver who may be totally unaware of the dangers of scuba diving and needs to be protected from putting his life at unnecessary risk;

2. The diving operators who need to know that the people that they are taking diving are medically fit and not a medical liability.

I have reviewed my most recent 1,000 initial sports diving medicals to identify the common problems encountered, to define the local diving population around the Whitsundays, to provide a baseline for future trends and to consider ways of improving diving medical services.

Unfortunately, my records cannot be considered representative of the whole of the Whitsundays, as my surgery is mainland based. Many divers, especially American and Japanese tourists, fly directly to the islands where they have a diving medical.

The Whitsundays is an area on the coast of central Queensland with 74 islands extending 32 km off shore. The adjoining section of the Great Barrier Reef is 64 km off shore. The islands and adjacent mainland host many tourist resorts and facilities. The local harbour, Shute Harbour, is the second busiest passenger port in Australia. The area has 11 diving centres run by eight independent diving companies. It is becoming a well-known and well-visited centre for scuba diving.

The Diving Population

The majority of divers were in their twenties. The average age was 25. 10% were over 30 and 6% were under 20. Only 3% were over 40.

Males predominated, but only in the proportion 60:40.

Surprisingly, only 22% were Australian, and British was the most common nationality (see Table One).

The low number of Japanese is not representative of the area but the other nationalities are probably representative. The vast majority of our divers are young tourists from overseas wishing to dive the reef as an adventure in the same way they will go white water rafting on the Tully River and four wheel driving in Cape York. It would be interesting to find out how many of them continue diving.

Discussion with other doctors in North Queensland strongly suggest that my figures are representative of the North Queensland area as a whole, from the Whitsundays to Port Douglas, where each week several hundred divers are taught.

TABLE 1

COUNTRY OF NATIONALITY OF DIVERS

United Kingdom	327
Australia	222
Canada	83
Germany	61
USA	57
Sweden	54
Switzerland	49
New Zealand	31
Holland	24
Ireland	22
Austria	14
France	13
Denmark	12
Finland	7
South Africa	5
Norway	5
Israel	5
Japan	4
Zimbabwe	2
Portugal	1
Spain	1
Malaysia	1

If one assumes that the vast majority of these divers will rarely dive again, then the estimated diving population of Australia, quoted by the diving instructor bodies based on dive course numbers and a higher retention rate, may be exaggerated.

The Medical

The diving medical consisted of a completion of a comprehensive questionnaire, a full physical examination and a spirometric measurement of the forced vital capacity (FVC) and the forced expiratory volume of one second (FEV_1). A chest X-ray was taken with any past medical history of lung disease or infection, or a family history of TB. An audiogram was performed if there was any history or clinical suggestion of middle or inner ear disease or hearing loss. Any candidate 45 years or older had an electrocardiograph.

The Failures

A total of 84 divers failed their initial medical, 27 of them provisionally and 57 permanently (see Table Two).

Despite all the dive schools screening their students, the commonest reason for failure is asthma. My present

policy on asthma is that anyone with an asthmatic history will fail unless they have been free of all attacks or the suggestion of an attack since the age of 12 and for at least ten years.

Two divers were found to be asthmatic who did not know they had it. Some tried to hide it but were identified clinically, whilst other were mild asthmatics and thought they should dive.

TABLE 2

REASONS FOR FAILURE

Asthma	39
No demonstrable ear equalisation	7*
Acute chest infection	5*
Severe scarring of ear drum	5
History of pneumothorax	4
Upper respiratory infection	4*
Middle ear effusion	3*
Impacted wax in ear	3*
Otitis media	2*
Severe Otitis externa	1*
Acute sinusitis	1*
Insulin dependent diabetic	1
Acutely infected wisdom tooth	1*
History of reconstructive maxillary surgery for congenital cleft lip and palate	1
Chronic active Hepatitis	1
Recent use of Bleomycin	1
History of chest surgery	1
Pleural adhesions from old chest infection	1
History of stapedectomy	1
Chronic bronchitis	1
Sarcoidosis	1

* provisional failures

The next most common reason for failing was the inability of the diver to demonstrate Eustachian Tube (ET) function by the Valsalva (blowing with the nose and mouth closed) or Toynbee (swallowing with the mouth and nose closed) manoeuvres despite full instruction and numerous attempts, yet with no obvious pathology. In the novice diver, this is a dilemma. It may be only technique and lack of practice, but to pass them would expose them to likely early aural barotrauma and possible future hearing loss, and failure to complete their diving course with considerable financial loss.

Only one of the seven who could not autoinflate returned able to demonstrate normal ET function. The other six either continued to have no function or moved on in their travels or to another medical examiner !

I recorded the incidence of some of the commoner conditions and problems significant to diving (see Table 3).

A diver's ears were only syringed if the external canal was occluded with wax or debris, making it impossible to view the ear drum adequately. For the diver to dive with such a blocked ear may cause external ear infections (especially in the tropics) and a possible danger of reverse squeeze (external ear barotrauma of descent) if water cannot enter the ear canal to the tympanic membrane.

TABLE 3

INCIDENCE OF COMMON PROBLEMS

History of hay fever	98
Previous chest infections	69
Migraine history	74
Glycosuria found (none found to be diabetic)	5
Heart murmur at examination	29
Needed to have ears syringed	72
Smokers	311

Discussion

Sports diving medical standards still vary immensely from doctor to doctor. Only after attending the diving medical courses at both HMAS PENGUIN and the Royal Adelaide Hospital's Hyperbaric Medical Unit, have I discovered that there are certain absolute contraindications and many relative contraindications. The relative contraindications are open to much interpretation.

There is a need for a Diving Medical Standards handbook where the medical standards for sport diving are actually defined in much the same way as aviation medical standards. This will allow medicals to be more consistent, more credible and more acceptable. Too long has it been possible to fail a medical then go down the road and pass.

I am now in the process of writing such a handbook for use in my practice. It is a teaching process in itself, making oneself justify each decision, researching the evidence and opinion in diving medicine.

A controversial subject in diving medicine is the prospective diver with a borderline history of asthma or a history of childhood asthma who has not had an asthma attack for many years. In the city they may be referred to a respiratory laboratory for full assessment. In country areas this is not practical. I have found it necessary therefore to set up histamine and hypertonic saline provocation tests in my

surgery. Another very useful tool is the impedance tympanometer for assessing the tympanic membrane, the ossicular chain and middle ear function.

Conclusion

There has been little information published on the results of sports diving medicals. By reviewing my last 1,000 sports diving medicals I have attempted to highlight interesting points, identify problems and shortfalls in medicals, with possible solutions and, hopefully, create further discussion on the needs of diving doctors and the diving industry. Only by showing how diving medicals help the diving industry will they become totally accepted and supported.

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PAPERS FROM THE SPUMS 1989 ANNUAL SCIENTIFIC MEETING

PROBLEMS WITH LESS THAN 2 ATA EXPOSURES

Jimmy How

Introduction

Diving and working in compressed air tunneling are similar in many respects. After noticing the 10 cases of decompression sickness (DCS) arising from compressed air work at less than 1 bar gauge pressure, it brings to mind that sports diving, even at shallow depths can carry risks of DCS. Aside from DCS, the commonest diving accidents and deaths that are seen in Singapore result from divers experiencing problems at shallow depths. I will discuss:

(a) DCS at shallow depths
(b) Medical problems in diving
(c) Diving in unfamiliar situations

Brief Historical Background

People have been diving for food, pearls, sponges for thousands of years. Divers have been known to be in existence during the time of the ancient Greeks and the Trojan War.

Breath-hold diving was the earliest form of diving that evolved. Breath-hold divers are still in abundance everywhere where shallow, calm and warm waters provide the recreational diver a chance to immerse himself amongst the abundant marine flora and fauna found in the tropical and subtropical regions of the world.

It is noteworthy that breath-hold diving for commercial gain still exists among the natives in the Pacific Islands and among certain traditional occupations in Japan and Korea.

Sports diving with self contained equipment only became popular after 1943 when Jacques-Yves Cousteau and Emile Gagnan developed the modern demand intake valve. Today, there are thousands of recreational divers who venture out into the sea daily. With the explosion of the sport in the 70s and the 80s, diving physicians are concerned about the safety of the medical selection and diving training provided by various diving operators. Inexperience among the new entrants to the sport and the overconfidence of the experienced diver have resulted in unnecessary fatalities.

Surface supply equipment is another method of diving commonly practiced. Many of the cases of decompression sickness treated in Singapore in the late 1970s and the early 1980s were fisherman divers suffering from DCS who had used surface supply equipment. Abalone divers in Australia use surface supply equipment. Based on our experience with the Singapore Mass Rapid Transit (MRT) Project, it may even be possible to suffer from DCS at shallow depths of less than 10 metres.

I will discuss the problems that may be encountered by the sports diver at less than 1 bar gauge (or 10 metres sea water) exposures and highlight certain diving related problems that can arise either through ignorance or overconfidence. But first I will discuss the 10 cases of DCS arising in compressed air workers during the MRT project.

Decompression sickness after less than 2 ATA exposures

Decompression sickness occurring at pressures of less than 1 bar gauge or (14.7 psig) is very unusual. A literature search revealed that probably only Behnke[1] has ever reported instances of cases of DCS at less than 1 bar gauge exposures for compressed air workers. In his report, he noted 9 cases of DCS occurring in less than 1 bar exposures of compressed air workers at the Bay Area Rapid Transit (BART) Project in San Francisco, California.

In conversation with other hyperbaric physicians, I have heard of other such cases here and there but I have been unable to trace any publications reporting these. Eric Kindwall mentioned to me 4 cases of DCS occurring at less than 1 bar gauge.

In the Singapore MRT Project, there were 10 confirmed cases of DCS at less than 1 bar gauge exposures. They all responded to recompression therapy with complete resolution of symptoms.

The tunnel projects were completed in record time and because of the haste in completing the projects, long working hours, sometimes exceeding the limits of the Blackpool Tables, were employed. This may have resulted in some of the DCS cases.

Analysis of Singapore cases of DCS

Eight cases of DCS, following exposures to below 1 bar gauge pressures, occurred at Contract 109 while the other two cases were seen in Contract 301. Seven of the cases occurred after exposure times of 12 or more hours (maximum 12 hours 22 minutes), including the time for decompression. The other 3 cases occurred after exposures of between 10 hours 45 minutes and 11 hours 45 minutes.

The lowest working pressure where DCS cases were reported, was 0.8 bar gauge while the highest was 0.95 bar gauge. One case was seen in a surveying assistant, and another was seen in a foreman. The rest of the cases occurred amongst compressed air workers (Table 1).

ENVIRONMENTAL FACTORS

During the exposure periods shown in Figure 1, tunnel temperatures were between 28 and 34 degrees centigrade with an average humidity range between 60% to 100%.

CLINICAL PRESENTATION

Pain was the commonest presentation noted. They were described as deep joint pains. Tenderness was present in one worker. Two workers complained of warmth. No swelling or rashes were seen. One worker had numbness around his joint. Table 2 shows the distribution of symptoms.

TREATMENT

All cases were treated with recompression therapy. Table 61 (oxygen table) was used in 50% of the cases. CIRIA 1 (air table) was used in 40% of the cases. CIRIA 1 and Table 62 were used in Case 9 when there was an initial lack of response to the CIRIA 1 table at depth. Complete relief of pain were recorded in all 10 cases (Table 3).

DISCUSSION

The cases manifested characteristic symptoms of DCS and responded to treatment. The 10 cases represented a DCS incidence of 0.005% of man decompressions for all pressures and 0.008% of man decompressions below 1 bar.

None of the men in this study was obese. The maximum percentage body fat was 20% and the minimum 6%. Average percentage body fat was 14.5%. Although obesity is recognised as more susceptible to DCS than thin people, there were only 27 obese persons (>24% body fat) in the Singapore MRT Project out of 1,737. This accounted for the bias of thin persons developing DCS. In addition, as the duration of exposure was limited for the obese persons, the likelihood of any of them getting DCS was reduced.

In our study, the oldest worker was a 39 year old foreman. With the exception of a survey assistant, the rest were compressed air workers. The youngest compressed air worker affected was 18 years old. The average age of those affected was 29.2 years. As the number of cases was small, we are unable to prove any correlation of DCS with age, obesity and type of work performed. However the cases occurred after very long exposures and the men were involved in heavy work. The long hours probably allowed for complete tissue saturation with nitrogen, even the very slow tissues, those tissues which take a very long time to become saturated with nitrogen.

Two cases worthy of mention were not treated by the Navy. These two compressed air workers completed their work in Singapore and were flying back to Bangkok, when they reportedly felt joint pains. These joint pains subsided when the plane finally landed in Bangkok.

Interviews with various Korean workers also revealed that 3 of them had developed joint pains after such exposures but they did not report this to their supervisors. Subsequent exposure to compressed air relieved their symptoms.

Possible mechanisms of DCS in less than 1 bar

It is uncertain what caused DCS in these 10 men. The tendency for bubbles to form is governed by the principles of fluid mechanics. It has been shown that a large force is required to form bubbles in vitro, unless bubble nuclei are present. These forces may be due to tribonucleation, cavitation or even from spontaneous in vivo nuclear fission. We feel the following mechanisms are likely to be implicated.

The compressed air workers were doing heavy work involving lifting and the use of vibrating tools. This can cause the formation of micronuclei by the process of tribonucleation. Tribonucleation is induced in vivo when two

TABLE 1

DECOMPRESSION SICKNESS (DCS) AT LESS THAN 1 BAR PRESSURES IN RELATION TO RACE, CATEGORY OF WORKER, BODY FAT, AGE, WORKING PRESSURE AND TIME OF ONSET OF SYMPTOMS.

S/No	Case	Race	Contract	Type of Worker	% Body Fat	Age	Working Pressures (bar)	Date of Incident	Exposure Time	Time of Onset After Decompression	DCS Type
1	Mr S P	Thai	109	CAW	11%	32	1.80	1.2.85	12 hr	A few hours	1
2	Mr S K	Thai	109	Surveying Assistant	6%	28	1.80	1.2.85	10 hr	A few hours	1
3	Mr D C	Thai	109	Forman	20%	39	1.80	8.2.85	12 hr	A few hours	1
4	Mr S T	Thai	109	CAW	9.4%	18	1.95	23.12.85	12 hr	A few hours	1
5	Mr N	Thai	109	CAW	15%	24	1.95	25.12.85	11 hr 45 m	3 hours	1
6	Mr S B	Thai	109	CAW	11.3%	31	1.95	29.12.85	12 hr	1 1/2 hours	1
7	Mr U S	Thai	109	CAW	12%	25	1.95	29.12.85	12 hr	3 hours	1
8	Mr V A	Indian	109	CAW	15%	29	1.75	16.2.86	10 hr 55 m	4 1/2 hours	1
9	Mr KYK	Korean	301	CAW	19%	31	1.90	21.1.87	12 hr 22 m	6 hours	1
10	Mr CJC	Korean	301	CAW	14.2%	35	1.94	16.3.87	12 hr 22 m	28 hours	1

CAW stands for compressed air worker.

FIGURE 1

HUMIDITY AND TEMPERATURE CHART FOR CONTRACTS 109 AND 301

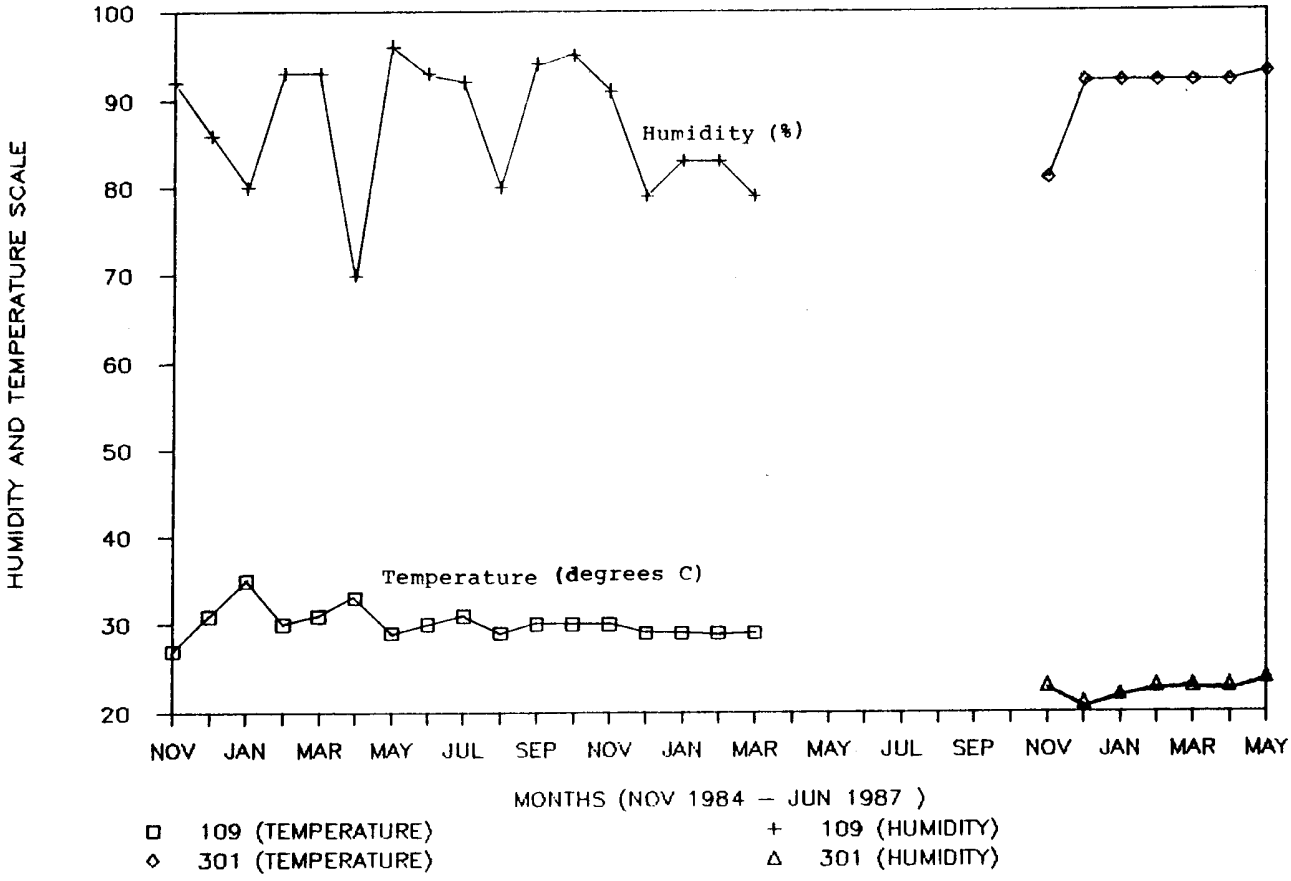

TABLE 3

SUMMARY OF SYMPTOMATOLOGY AND TREATMENT USED

Case	Affected Site								Treatment Table Used	Outcome of Treatment
	Shoulders		Elbow		Hips		Knees			
	Uni	Bi	Uni	Bi	Uni	Bi	Uni	Bi		
1								*	61	Complete Cure
2						*		*	61	Complete Cure
3	*					*		*	61	Complete Cure
4							*		CIRIA 1	Complete Cure
5		*							61	Complete Cure
6							*		61	Complete Cure
7							*		CIRIA 1	Complete Cure
8				*		*		*	CIRIA 1	Complete Cure
9			*				*		CIRIA 1 and 62	Complete Cure
10				*					CIRIA 1	Complete Cure

Note: Uni = Unilateral Bi = Bilateral

TABLE 2

CLINICAL PRESENTATION

Symptomatology	Number
Pain	
Deep	10
Constant	2
Limitation of movement	3
Joint tenderness	1
Joint numbness	1
Warmth around joints	2

closely opposed surfaces separated by fluid are forced apart. The negative forces or low pressures generated as a result of the separation of the two surfaces result in the formation of bubble nuclei.

Another mechanism where microbubbles may be formed in the compressed air worker is related to the increased haemodynamics in compressed air workers performing heavy work often in temperatures of 40o C. At the molecular level, fast moving fluid particles, by cavitation, generate sufficient negative forces behind the particles to cause the formation of microbubbles. Other mechanisms which may cause microbubble formation include a suggestion by Walder and Evans[2] that spontaneous in-vivo nuclear fission may be the aetiology of gas micronuclei.

Bubble nuclei have been shown to be necessary for bubbles to occur in an elegant series of experiments by Evans and Walder on transparent shrimps.[3] Three groups of 50 shrimps were decompressed from seal level to 0.079 ATA. One group was pressurised to 389 ATA before being decompressed. A second group was not pressure treated. In the pressure treated group 4 shrimps were seen to contain bubbles while 48 did so in the non-pressurised group. The third group was pressurised and then electrically stimulated. 16 of these shrimps developed bubbles on decompression. It has been argued that the bubbles originated from gas nuclei, and that the first batch of shrimps had their in vivo bubble nuclei squashed out of existence before being decompressed. This reduced the capacity of supersaturated tissues to form bubbles.

The compressed air worker enters and exits from compressed air daily after spending long hours in the compressed air environment. This form of repetitive exposures greatly increases their chances of DCS. Experience from repetitive dives in fisherman divers have shown that there is an increase in incidence of DCS in the second and subsequent dives. In the first exposure, small asymptomatic bubbles may have been formed. In the subsequent exposures, these asymptomatic small bubbles formed sites of further bubble growth, accounting for the increased incidence of DCS in subsequent dives.

Bubble micronuclei do not cause symptoms by themselves. The compressed air worker must have enough gas loading in his tissues to enable the bubbles to grow. In addition, the rate of decompression must be great enough to overwhelm the compressed air worker's circulatory capacity to transport the excess nitrogen from the tissues to the lungs.

Although the compressed air workers were decompressed according to the CIRIA recommendations[4], they had spent more than 8 hours in the compressed air tunnels. It is possible that there are very slow tissues that become totally saturated only after long exposure times. During decompression, nitrogen is released, but because of the slow half-times, the rate of nitrogen elimination could have resulted in bubble formation localised in these slow tissues. Tissue bubbles, when formed, can cause physical distortion of the tissue planes and stretch nerve endings. This is manifested as the symptoms of pain and numbness.

Slow decompression to eliminate the problem of DCS in compressed air workers has been advocated since the the 19th century. Haldane[5] believed that bubbles were not formed if the drop in ambient pressure did not exceed a ratio of 2:1. The variable time course of nitrogen uptake and subsequent elimination could be simulated by a family of discrete hypothetical half-time tissues. Haldane took tissues with half-times up to 75 minutes to develop his decompression tables which were adopted by the British Admiralty. Subsequent development and improvements to Haldane's tables were later adopted for various compressed air tunneling projects in England. In 1958, Hempleman devised the current compressed air tunnel tables (Blackpool Tables) which were incorporated into the CIRIA report.[4]

The disadvantage of using the CIRIA procedures for decompression of our compressed air workers from less than 1 bar exposure was the fact that very long half-time tissues were not considered. Our workers worked for between 8 to 12 hours at pressures approaching 1 bar gauge with 12 hours on the surface. Benhke[1] had proposed that there are tissues with up to 120 min half-times which will require up to 14 hours to desaturate 99%. Calculations for the decompression times for divers with exceedingly long exposures must take this into consideration. The interval between exposures must also be greater than the 14 hours for these long half-time tissues. The compressed air workers therefore may have accumulated nitrogen due to the long exposures and repetitive nature of their work. This is a possible reason why some of our compressed air workers developed DCS as they had worked longer than 10 hours within the tunnels and had spent less than 12 hours at the surface.

The US Navy's experience with long and deep exposures have also revealed deficiencies in assuming Haldane's

2:1 ratio for decompression. They found unacceptably high rates of DCS when assuming Haldane's theory of using a 2:1 decompression rate. A better proposal was made by Workman.[6] He suggested that blood perfusion of the tissues (excluding tissue diffusion) is the chief factor affecting the rate of gas transport. In his calculations, Workman considered more tissues as well as slower tissues, some with half-times of 1,000 minutes. The critical ratio varied at each depth for a particular tissue. He devised a linear scale of "M" values, showing the maximal allowable supersaturation for each hypothetical tissue at each depth for the whole range of decompression for nitrogen and helium diving. These formed the basis for the derivation of the US Navy Tables.

The decompression tables of the US Navy and the tables promulgated in the CIRIA report may have, in general, prevented symptomatic bubbles from occurring in the compressed air workers. Brian Hills[7] proposed a thermodynamic model of DCS from his work with pearl divers working out of Broome, western Australia. He introduced the tern tissue un saturation based on the lower total partial pressures of gases in the tissues and venour blood when compared with the alveolar air and arterial blood. He suggested that although bubbles develop on decompression it requires a 6o mm Hg drop in pressure before the tissues become saturated with gas. He suggested that gas bubbles are formed during decompression with the US Navy tables and the decompression rate merely controlled the size of the bubbles. The primary event and the critical insult which produced the symptoms of DCS do not coincide. The primary event is the activation of one or more of a reservoir of nuclei normally present in tissue into growth and hence the inception of a stable gaseous phase. The inception of this gas phase occurs randomly.

It is possible that limb pains are caused by the local pressure of a bubble distorting a nerve ending beyond its pain provoking threshold. The onset of limb DCS is dependent upon the volume of gas separated from solution. The inception of bubbles in the limbs of compressed air workers can be profuse and rapid due to the presence of micronuclei created by tribonucleation and cavitation in the joint. As a result the tissue can only withstand minimal supersaturation before gas in excess of thermodynamic equilibrium forms bubbles.

The compressed air workers worked at high temperature and humidity for long durations. An additional contributing factor to be considered is dehydration. Dehydration reduces the circulating blood volume. During decompression, the increased gas load at the tissue levels may not be eliminated fast enough due to the reduced blood volume. This may cause a build up of bubbles in the tissues resulting in symptoms of DCS.

Alcohol is known to cause tachycardia and vasodilatation, which may cause haemodynamic changes in the body. Although alcohol ingestion was denied by all the workers, there is no assurance that alcohol was not imbibed before they entered the tunnel.

Age and obesity have been noted in factors that predispose workers in the development of DCS. Older workers tend to have less efficient cardiovascular systems which possibly result in a reduced capability to clear the excess nitrogen. In Singapore the manual labourers were less than 35 years old. The obese workers, who had greater than 24% body fat, were given limited duration exposures to compressed air work. The 10 cases of DCS were neither old nor obese and are therefore unlikely to be affected by these factors.

One of the patients gave a history of working from 8 a.m. till about 8 p.m. in the tunnel. Then from 4 a.m. to 7 a.m. he moonlighted as a newspaper distributor to supplement his income. This may have precipitated the development of DCS as prolonged exertion results in an increase in the number of bubble nuclei, and the sites where further bubble growth could occur.

Our experience was that 90% of the cases of DCS at less than 2ATA occurred when the pressure was greater than 0.8 bar gauge. Two cases, one at 0.94 bar gauge (Case 9) and one at 0.9 bar gauge (Case 10) exceeded the 12 hour limit of the CIRIA recommendations. A disadvantage of using the CIRIA regulations in that for exposures less than 1 bar, regardless of the exposure time, no decompression stops are required and the compressed air worker can be decompressed direct to the surface. The assumption that DCS does not occur if the exposure pressure is less than 1 bar must be questioned in the light of our experience.

The 10 cases of DCS occurred in young and healthy individuals of a mixed ethnic group. There were no obvious individual predilection to DCS. Various factors related to the nature of the work like heavy manual labour and repeated entries into the compressed air environment may have resulted in the formation of microbubbles through the process of tribonucleation and cavitation. The long exposure times and the rate of decompression caused the microbubbles to grow and produce symptoms in these 10 compressed air workers. Exactly why it happened in these 10 compressed air workers and not the other compressed air workers, we cannot be certain. Perhaps there is an individual predilection or an individual daily variation of susceptibility.

This conclusion seems to be in line with the conclusion drawn by Benhke[1] of the 9 cases of DCS that were seen following exposures between 11.5 and 16 p.s.i. gauge in the BART project in the USA.

DCS and diving to shallow depths

The mechanism in which DCS can occur at shallow depths is similar to that seen with the compressed air workers. The likelihood increases when repetitive dives are

done or when divers use surface supply equipment to prolong their stay underwater to allow them to dive for hours.

Even with breath-hold diving, DCS can occur if the duration and depth is long enough. This condition, called Taravana, was an observation reported by various authors amongst the Pearl divers of the Tuamotu Archipelago. Repetitive breath-hold dives up to 40-50 times a day are performed. The islanders hyperventilate for periods ranging from 2 to 10 minutes. During descent, the diver holds a lead weight between his feet and the rope in one hand. With the free hand the diver grasped his nose to assist in equalisation of his ears and sinuses. The divers dive to depths of 120 feet for 1 to 2 minutes. Cases of severe vertigo, nausea, paralysis, unconsciousness, mental derangement and deaths were reported among these divers.[13]

Medical Problems in Diving

The sea is strange and mysterious environment. Throughout the ages, legends about large sea monsters, about falling off the edge of the earth, the names Roaring Forties and Furious Fifties, all portray Man's fear and uncertainty about our oceans. The psychological aspects of sports diving must not be ignored by diving physicians as fear, ignorance and insecurity can contribute to or complicate a diving accident. Experienced divers can think through problems and go through the drills that have been taught to them, such as ditching the weight belt, controlled ascents, buddy breathing, but an inexperienced, anxious or fearful diver will most certainly find difficulty in doing the same tasks.

Proper medical selection therefore should take into account of the psychological maturity and confidence of the diving candidate. A weaker swimmer is less likely to be able to remain afloat for as long as a strong swimmer. A candidate psychologically fearful that his equipment will fail on him can run the risk of panic and rapid ascent, with drastic consequences. Similarly, one who is fearful of the dark or the deep, or has an intense fear of sharks, can endanger his own life, and even his buddy's, should he panic and do something silly.

Certain medical conditions should exclude a person from diving. These include asthma, epilepsy or a previous seizure episode, ischaemic heart disease, and cardiac arrhythmias which may lead to a sudden ventricular fibrillation (e.g. Wolf Parkinson White Syndrome).

Diving in unfamiliar situations

Inexperience plays a large part in many of the cases of diving accidents occurring in shallow waters. It may occur with newly qualified divers or with divers who have laid off for some time. Certain areas in diving are more risky, and divers require preparation and training prior to attempting the dive. Worthy of special mention is diving at night, in sink holes, caves, fresh water, springs and wrecks. Divers, experienced only in one area of diving, must be considered novices when doing another category of diving, as techniques and safety procedures are different.

Not every diver will dive regularly throughout the year. Frequently, the newly qualified divers take a diving course only to lay off diving for a considerable time. In temperate countries, the onset of the winter season usually heralds the end of the diving for the year. Approximately 4 to 6 months later, when the weather finally gets warm enough for diving, the diving season begins again.

Hazards of diving to shallow depths

In considering the hazards of diving at shallow depths, the following categories may be used.

(a) Environmental hazards
(b) Hazards due to equipment
(c) Hazards caused by individual factors

ENVIRONMENTAL HAZARDS

The problems of diving in unfamiliar situations are manifold. Even for the experienced diver, preparation and planning must be detailed. Planning should include a complete appraisal of the dive area, the equipment used and a self appraisal of one's capabilities.

The environmental conditions to be noted at the dive site include:

(a) Depth of the water
(b) Sea state, tidal conditions and strength of the current
(c) Water visibility
(d) Temperature of the water
(e) Time of the dive
(f) Types of marine life which will be encountered
(g) Obstacles expected to be encountered.

The dangers of breath-hold diving at shallow depths include hyperventilation hypoxia and hypocapnia. Hyperventilation hypoxia results when the diver hyperventilates before diving producing hypocapnia. During the dive there is progressive hypoxia, but because of the low levels of CO_2, respiration is not stimulated before unconsciousness overcomes the diver. The cause of death, if not rescued, is aspiration and drowning.

Careful consideration of the environmental conditions is important. Diving in poor sea conditions or strong currents is dangerous and should always be avoided. Breaking surf, currents greater than 1 knot, stormy weather, bad sea states and impending darkness all pose potential problems for divers and the crew of their dive boats. Aside from seasickness in the persons on board the dive boat, the diver attempting to return to the boat on the surface may lose sight

of his dive boat in heavy seas. An experienced diver is usually able to sustain a swim at about 1 knot for only a few minutes. Diving in currents in excess of 1 knot is foolhardy and can result in divers being swept away.

Diving at night and in poor visibility requires planning and coordination between the diver and his buddy as well as with the dive boat. Divers should practice safety drills and equip themselves with the appropriate buddy lines, torches, and if possible flares, in order that the dive boat can locate divers who have gone adrift.

Underwater entrapment is a common cause of diving accidents and this can occur in the experienced diver. Seaweed (especially kelp), coral formations, rock outcrops, caves, wrecks, ropes and fishing nets all pose potential hazards for the diver.

Cave diving, diving in sink-holes, springs and quarries requires good planning and adequate equipment. Lifelines, torches, reserve tanks and other safety equipment must be taken and used to ensure safety.

Adequate maintenance of body heat is necessary and the early symptoms of hypothermia, like shivering, should be heeded and preventive actions taken. If hypothermia is prolonged, it will lead to a progressive deterioration of mental function, and unconsciousness. The patient eventually develops ventricular fibrillation and dies. Adequate thermal protection, like wetsuits or dry suits, to prevent heat loss should be used in cold water.

Although shark attacks constitute less than 1% of all diving fatalities, prudence in selection of a dive site is required, especially if shark attacks or sightings have been reported lately.

EQUIPMENT FACTORS

A diver should become familiar with the equipment to be used. This is especially so for rented equipment. Particular attention should be focussed on the mechanism for the release of the weight belt and the inflation of the buoyancy compensator. Pre-dive checks should include recognition of the location of the buckle for the weight belt and to ensure that the weight belt buckle does not rotate out of reach while in the water.

An important piece of equipment is the buoyancy compensator (BC). This must be stored properly when not in use to prevent premature deterioration of the rubber and plastic components. Pre-dive checks should include a test for leaks in the BC to ensure that it functions properly. Checking the BC prior to the dive should also involve the inflating mechanism. Most BCs now are inflated from the diver's air tanks using a power inflator. This set up is elegant but divers should be aware that at the end of the dive, when the air has run out, the BC cannot be inflated. This can lead to a minor crisis especially if the diver is overloaded with a haul of treasure or abalone. Quick thinking and action is required to avert a tragedy. Other BCs are equipped with a CO_2 cartridge which inflates when a ripcord is pulled to activate a triggering mechanism. Corrosion, due to poor maintenance, can jam the triggering mechanism, preventing its activation. Alternatively, the CO_2 cartridge may have already been used or has leaked. Checking the cartridge is therefore important.

Steel air tanks should be properly maintained. The air used for charging steel cylinders must be free from moisture. Corrosion can occur on the inside of the tanks and deplete the oxygen within the tank. There is a danger of hypoxia if a steel tank that has been stored full for a long time is used. In any case a corroded steel or aluminium tank is dangerous and may crack or explode when charged. Owners of scuba tanks should be aware of this danger and ensure regular hydrostatic testing by an approved authority.

Two other important but often ignored pieces of equipment are the depth gauge and the contents gauge. Depth gauges should be calibrated regularly as, in our experience, they are often inaccurate by up to 15%. This can result in a miscalculation of the diving profile and increases the risk of decompression sickness. Contents gauges should also be maintained properly to ensure correct readings. Divers should regularly consult their contents gauges to check on the amount of air left.

Diving in polluted and heavily silted waters can result in failure of the second stage regulator. Fatalities have been reported when diving in quarries which were heavily silted.

For the sake of completeness, I would like to mention a few dangers faced by military divers at shallow depths. These problems usually arise with the use of specialised rebreathing apparatus. The problems include oxygen toxicity, carbon dioxide retention, hypercapnia and caustic soda burns.

Oxygen toxicity in the military diver occurs usually when the diver exceeds the prescribed depths. The divers have to perform strenuous tasks underwater and this increases the rate of development of oxygen toxicity. There is considerable individual variation in the tolerance to oxygen toxicity. In Singapore all military divers have to undergo an oxygen tolerance test to exclude those divers in whom there is a high susceptibility to oxygen toxicity.

Carbon dioxide retention occurs when the CO2 absorbent material (usually soda lime) has either been packed too loosely or when the potency of the chemical has diminished. Caustic burns occur when salt water comes into contact with the soda lime in the re-breathing set. This may occur with poor technique, with water leaking from the

mouthpiece, or when leaks occur in the system. Salt water combines with the soda lime to give off caustic soda (sodium hydroxide). This can be aspirated, causing burns to the lips and throat, as well as a nasty surprise for the diver. However, there is a danger of the diver panicking and rushing to the surface.

PERSONAL FACTORS

Individual factors can predispose to hazards and accidents in diving. It is usual for the beginning of the season to see accidents happen to divers who have not been diving during the off season. Divers must be aware of their limitations and capabilities. Meticulous planning is important in ensuring safe and enjoyable dives. Detailed planning of the dive should include the calculation of the amount of air required for the whole duration of the dive including allowances for delays and decompression stops. In shallow diving, planning before a dive is still required. Decompression sickness may occur, with very long exposures and especially with surface supply equipment.

Pulmonary barotrauma, with drastic consequences, is still likely when divers run out of air and they fail to notice it until it is too late. Frequently, divers in panic situations (e.g. running out of air or aspiration of small quantities of water) hold their breath and rush to the surface. This may result in fatal air embolism as the expansion effects of the bubbles is greatest between 1 bar and the surface.

Alcohol ingestion has been shown to be one of the main contributing causes to drowning. Drinking and diving can result in carelessness and poor judgement among divers. This can contribute to fatalities. In addition alcohol ingestion has been related to an increase in the risk of decompression sickness.

Untrained divers should not attempt to dive unless undergoing a proper dive course. In Singapore, there is no legislation preventing untrained persons from buying diving equipment. Proper medical clearance and diving training is essential in ensuring safe diving. Safety drills, buddy breathing, rescue and cardio-pulmonary resuscitation are important skills to be learnt by the budding diver.

Poor technique and improper training can lead to problems of equalisation of the ears. Conditions like sinus and aural barotrauma may complicate the dive. Frequent comments such as "Boy! My eardrums are bursting. Those divers must have eardrums made of steel", demonstrates the ignorance of the general public about the proper techniques in diving.

Vertigo due to unequal vestibular stimulation or tympanic membrane perforation can result in disorientation and panic in inexperienced divers. Of even greater concern is the danger of pulmonary barotrauma and air embolism in untrained divers who are asthmatics or who breath-hold during ascent.

Drugs can have side effects which may reduce the reflexes and motor coordination of divers. Taking antihistamines may reduce the nasal congestion of divers with colds, but at the same time, it may cause drowsiness in some divers. This is potentially risky and can cause accidents due to misjudgment. Addictive drug abusers should be condemned and disallowed from diving as they cause dependence, impaired consciousness and disorientation.

Heavy smokers run risks of chronic bronchitis and obstructive lung diseases after continuing with their habit for many years. These lung conditions can lead to air trapping and potentially hazardous pulmonary barotrauma. Smokers who intend to continue diving should stop smoking. It is also a good idea for all divers to have a proper medical every year which include a full sized chest X-ray and spirometry.

Wherever possible, the inexperienced diver should be accompanied by an experienced buddy when an unfamiliar form of diving is carried out. The buddy system allows the experienced diver to keep the inexperienced diver out of danger. Many reports of diving fatalities cite inexperience as one of the main contributing factors to fatalities. For instance:

A newly qualified diver was killed recently when strong currents swept him away from the dive boat. He was found later, with a fractured skull and bruises on his head, face and body.

Another incident occurred where a woman diver got into difficulties at depth and although she was diving with her boyfriend, only the boyfriend managed to ascend to the surface. Her body was never found.

Conclusion

Prudent planning and a level headed approach to diving preparation is essential for a successful and enjoyable diving expedition. There are dangers even in shallow water diving but these can be safely overcome.

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Editorial, continued from page 77.

awareness of both the advances in our knowledge of diving related problems and of how much still remains uncertain. This is shown by the numerous versions of diving tables and the occurrence of cases described as atypical decompression sickness. After a short period of total ignorance there grew a belief that all problems related to diving had been identified and were fully understood. There is now a growing awareness of the complexity of the changes which can follow an alteration of the ambient pressure ant that these are influenced by seemingly innumerable factors. Complacency is the one deadly sin we cannot afford to condone.

Although Adlai Stevenson once made the somewhat debatable comment that the function of an editor was to separate the wheat from the chaff and print the latter, it is believed that no such assessment should be made of the many and varied papers that have appeared in the SPUMS Journal over the years. It is hoped that readers have been informed, interested and sometimes provoked into considering that some hitherto unquestioned belief should be re-examined. If that is so the Editor has been successful. From the next issue Dr John Knight takes over as Editor. His years of experience in the production of the Journal as Assistant Editor and as Deputy Editor make him an excellent choice and I am sure that the Journal will continue to prosper.

SPUMS ANNUAL SCIENTIFIC MEETING 1990

SCIENTIFIC PAPERS

Saturday, 2nd June, 1990

Ruth Inall
SPUMS and the Science Centre Foundation

Warwick McDonald
The DITAA analysis of the recreational diving industry

Martin Sher
The future of recreational diving

Sunday, 3rd June, 1990

Chris Acott
Diving incident monitoring

Courtenay Kenny
Dysbaric illness treated at HMNZS PHILOMEL

Peter Chapman-Smith
Case studies

Greg Adkisson
Diving accidents in the United Kingdom

Monday, 4th June, 1990

Greg Adkisson
The 1988 BSAC decompression tables

Drew Richardson
PADI decompression teaching in 1990 ()

Raymond Rogers
The development of the DSAT (PADI) decompression tables

Raymond Rogers
Testing of the DSAT (PADI) decompression tables

Tuesday, 5th June, 1990

Greg Adkisson
SPECT studies and cerebral decompression sickness

Des Gorman
Pathophysiology of cerebral arterial gas embolism

Chris Acott
Psychiatric disorders in diving

Warwick McDonald
Women in diving

Wednesday, 6th June, 1990

Greg Adkisson
Submarine rescue

Lori Barr
Case reports of tank carrier's lateral condylitis

Lori Barr
A biomechanical model of tank carrier's lateral condylitis

John Robinson
Vertigo in diving

ANNUAL GENERAL MEETING.

Thursday, 7th June, 1990

DIPLOMA OF DIVING AND HYPERBARIC MEDICINE

From time to time there are enquiries about higher training in hyperbaric and diving medicine. This society, in its aim to promote the education of doctors in the field of hyperbaric and diving medicine awards a diploma to a candidate who completes the following requirements:

1 Is a financial member in good standing.

2 Has completed both basic and advanced, examined, courses in diving and hyperbaric medicine either at the Royal Australian Navy School of Underwater Medicine or a similar approved institution.

3 Has completed six months full time in an approved hyperbaric unit.

4 Has submitted for approval of the Board of Censors a thesis, treatise or paper on a topic, previously approved by the Board, suitable for publication.

The following alphabetical list is of those who have been awarded the Diploma since 1972.

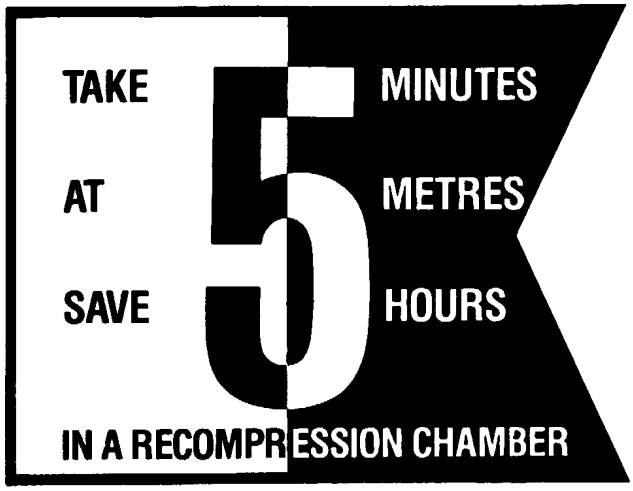
Dr C Acott
Dr J Anderson
Dr T Anderson
Dr N Barnes
Dr G Barry
Dr G Bayliss
Dr R Capps
Dr P Chapman-Smith
Dr J Chuah
Dr N Cooper
Dr D Davies
Dr M Davis
Dr G Dawson
Dr C Dillon
Dr C Edmonds
Dr M Fraundorfer
Dr A Gebbie
Dr J Gilligan
Dr D Gorman
Dr R Gray
Dr D Griffiths
Dr J How
Dr C Kenny
Dr D Kerr
Dr J Knight
Dr P Laverick
Dr G Lovell
Dr C Lowry
Dr M Loxton
Dr C MacFarlane
Dr S Maheson
Dr H Mahdi
Dr P McCartney
Dr B McKenzie
Dr I Millar
Dr R Moffitt
Dr J Monigatti
Dr W Murtha
Dr J Orton
Dr M Osborne
Dr H Oxer
Dr P Robinson
Dr R Schedlich
Dr A Slark
Dr D Smart
Dr R Stevens
Dr C Strack
Dr R Thomas
Dr B Turner
Dr D Tuxen
Dr I Unsworth
Dr R Webb
Dr J Williamson
Dr R Wong
Dr T Wong

SOUTH PACIFIC UNDERWATER MEDICINE SOCIETY INCORPORATED

SPUMS was incorporated in Victoria on 10th April 1990. This action, which was approved by the members in 1988, was taken to reduce the legal liability of the individual members if anyone brought a successful court action against the society. Without incorporation all members would have been collectively liable for any costs awarded against SPUMS. With incorporation the members' liabilities are limited to the society's assets.

Dr John Knight has been appointed the Public Officer of the society. This office must be held by a person resident in Victoria and is best described as the liason officer with the Victorian Corporate Affairs Office. **It is not a form of Public Relations officer.** All enquiries about SPUMS activities should be addressed to the Secretary. The Secretary and the President are the two official spokesmen for the Society. The Committee has the right to appoint other members as spoksmen on specific subject if it so desires.

The last rule approved by the Victorian Corporate Affairs Office stated, in essence, that all current members of SPUMS will be the first members of SPUMS Inc.



The Government of Western Australia has produced a decal, in dark blue and white, for divers to stick on their air cylinders, and anywhere else where it will be easily seen. It is hoped that the decals spread Eastwards so that the message gets to all Australian divers.

The decals are available from the
Department of Sport and Recreation,
Perry Lakes Stadium,
FLOREAT, Western Australia 6014.

ARTICLES OF INTEREST REPRINTED FROM OTHER JOURNALS

THE BC AS YOUR PERSONAL OCTOPUS

A Seldom-Taught Technique To Save Lives

You suddenly find yourself at 130 feet with no buddy in sight and no air to breathe!

You are certain you can surface in a controlled emergency ascent with ease from shallower depths, but you are afraid you will not make it from this depth.

What would you give for a reliable, super-easy-breathing regulator attached to a source of enough air to get you to the surface?

You do not have to wish. You are wearing one! It is your buoyancy compensator. You can breathe from it. It is simple and it works! Many divers have surfaced easily from the depths, some breathing *fresh air* all the way up.

Most divers have learned buddy breathing, and many are now equipped with extra second stage "octopus" regulators. Unfortunately, neither of these techniques will furnish air to you if your buddy is unavailable, unequipped or also low on air.

Unquestionably the best method of obtaining air is to have a backup supply, dual tanks with dual outlet valves, a pony bottle with an extra regulator attached, or "Spare Air", a bail-out bottle that has a regulator built into the unit.

But, what do you do now when you have none of these and no air left from any source?

No air from any source?

There are air sources you may not have considered. There will be residual air left in your lungs, in your tank, and in your buoyancy compensator.

You think your BC is empty? Not very likely. The deeper you are, the more likely you have put extra air into your BC to adjust your buoyancy. Even if you thought you squeezed out all the air at the surface, you still have an inflator hose full. Furthermore, air will frequently remain in the upper part of your BC. If you added air with your power inflator, it will be pure air. If you blew it in through your oral inflator, it will still have 16% oxygen. (Remember: we use exhaled air for artificial respiration.)

Your tank, even if "empty", will always have residual air that will become available as you ascend.

You can re-breathe many breaths

Re-breathing that same air will reduce the oxygen and increase the carbon dioxide content, accordingly making you hungry for fresh air. But re-breathing up to 13 times without becoming overly hungry for fresh air will be relatively easy. I have re-breathed over 40 times this way. Others have also. Dr Robert Leahy and Dr Peter Lynch conducted a study on BC breathing at Temple University, testing 22 basic scuba students who had used a regulator underwater only twice before. They were monitored while they re-breathed from their BCs for one minute in a pool 10 feet deep. They had no problems.

Your break-point will be surprisingly long in coming. You see, by inflating and deflating your lungs, you are stimulating stretch receptors in your chest and partially relieving your air hunger. Also, the available oxygen is being used and not exhaled into the open water.

You can add more air as you rise, by holding open the power inflator. The residual air from your tank will expand and flow into the BC, furnishing pure air to freshen the BC air you are re-breathing. If you wish, you can wait until you are near the surface to open the valve to replenish the air when you need it most. So as not to forget that air will be available later, hold that valve open all the way up.

You wont be tempted to hold your breath

The obvious advantage of BC re-breathing is having air to breathe. But just as important, you will avoid a lung rupture or embolism since you wont be tempted to hold your breath as you ascend. The build-up of carbon dioxide will cause you to steadily increase your breathing rate. You will be breathing in or out constantly and not closing off your airway.

A "poor man's octopus"

With the inflator hose from your tank, you can use your BC as an extra second stage regulator, a "poor man's octopus". You can give your regulator to an out-of-air buddy, while you breathe from your BC, both inhaling fresh air! Open the valve from your tank intermittently, breathe the fresh air from your BC and exhale it through your nose. Both of you can continue this way breathing fresh air for as long as your tank has air. To be confident of your ability when an emergency occurs, practice this first in calm shallow water.

I know of one actual rescue from 100 feet deep in which this technique has been used. After giving up his

regulator to buddy breathe, the diver could not retrieve it from his panicky muscular buddy. He calmly valved fresh air from his tank into his BC and breathed that on the way up.

Another diver saved two others who came to him out of air. He gave the first one his octopus, the second his primary regulator, and he used his BC. All three then breathed fresh air to the surface.

Three, or even two divers breathing through the same first stage could over-breathe that regulator, especially if they are deep, if the tank is low on air, or if they are all air-hungry. Some regulators will not supply enough air for easy breathing under these circumstances.

Avoid this possibility by waiting to feed air into your BC until your buddy stops inhaling. Then intermittently, between your buddy's breaths, feed the air in, keeping a comfortable volume in the BC for breathing and for proper buoyancy. Since it does not involve the first stage, you can inhale from the BC at any time without over-breathing. You cannot over-breathe your BC and it wont freeze up. As long as it has a breath full of air, it will feed it to you no matter how fast you breathe. It is a real easy-breather.

It will work if your second stage malfunctions

Having that inflator hose from your tank, you can also use your BC to obtain fresh air if your second stage malfunctions. The first stage at the tank valve will still feed air into your BC. Remember to exhale through your nose to insure a continuing supply of fresh air.

Purge the water from your BC mouthpiece

To be certain you will have no problem breathing from your BC in an emergency, you should practice clearing the ounce-or-so of water from the inflator hose mouthpiece. The type of BC mouthpiece that has openings in the end is designed for easy purging. Why not take advantage of that design feature?

Some of the newer BCs have hidden openings. If you cannot see any holes in the end, aim the end down and pour a little water in the mouthpiece. If the water runs out, it will clear easily.

You can purge *all* BC mouthpieces by using the following method: bend the mouthpiece up, seal your mouth, look down, and, as before, blow air in as you push the valve. The water will flow from the mouthpiece into the hose.

Take your first breath cautiously

Your first inhalation after clearing your BC should be a cautious one to be certain that no residual water will cause a coughing spell. Inhale slowly and carefully. If you do cough or strangle on a few droplets, do not remove the mouthpiece. Cough into the BC. Then that air will be available for you to re-breathe after your coughing spell is over.

A little water in the hose should not bother you. Unless you inhale in gulps, you can easily breathe without getting any water. Do not let go of the valve or remove the mouthpiece from your mouth as you ascend. Releasing your hold on the valve of a mouthpiece that has no holes will not allow water to enter, but it will cut off your air from the BC. Removing your mouthpiece will require clearing it again.

Little air to breathe back?

If you first discover you have no air when you have exhaled your last breath through your regulator and then get no air back, do not panic. Blow what air you have into your BC and re-breathe that. If the air you blew into it is only enough to go to the top of your BC, you may not get it back right away. Again, do not panic. Start up immediately and keep trying to inhale and exhale as you should through a regulator on a tank with no air. Remember that you can probably do without air for at least 20 seconds. As you rise, more and more air volume will become available. Even if you get only a low volume, you will be breathing. Keep breathing that small volume in and out. As you breathe in and out that volume will increase. You know that the deeper you are, the more molecules of oxygen are available in the air in your lungs, in your airways, in your BC, and in your tank.

Too much air!

Paradoxically, although you were out of air on the bottom, as you near the surface you might find that you may be too buoyant and are ascending too fast. Exhaling through your nose will reduce your buoyancy. Air will escape under the skirt of any mask, but a purge-valve mask will make nose exhaling easier. Your speed of ascent can also be reduced by flaring out horizontally.

It is possible that your BC will fill with air as you rise. Air inhaled from a turgid BC could be at a pressure high enough to over-expand your lungs. Exhale enough through your nose to keep the BC from getting completely full and you will avoid this. It takes more than five full breaths to completely fill almost all BCs, so if you never dive below 130 feet, you are not likely to end up with a full BC at the surface. If you added tank air to compensate for bottom overweighting, your BC could get too full as you ascend. If so, however, you will have lots more fresh air to breathe from your BC and can avoid overfilling it by exhaling more frequently.

Develop skill at handling your BC

Keep the mouthpiece valve closed tightly while intermittently putting air into your BC. The placement and operation of these valves differ depending on BC design. An inflator valve that has a feather-touch will be easier to control. Get confident that in an emergency you can manage both valves simultaneously. Practice in calm shallow water.

Flush out CO_2 after each cartridge inflation

If you have a BC that has a carbon dioxide inflator, do not breathe from your BC if it is full of CO_2. You will be warned very quickly. Pure carbon dioxide is stingingly pungent. Although CO_2 is initially odourless and tasteless, the gas reacts with the moisture in your mouth and nose, becoming carbonic acid. Although breathing high concentrations can be deadly, you are not likely to inhale much. A little is no problem (we consume it in carbonated drinks).

Keep your BC clean

Moisture in your BC when stored in a warm, dark place may encourage the growth of bacteria and fungi. Breathing from such a BC might cause an infection. When you are out of air in a life-threatening emergency, you certainly should not be concerned about an infection that may flare up days later. For practice, however, you will want a clean BC.

Gary Keller, president of Sonoform, a manufacturer of BCs, states that they use ether-based non water-permeable polyurethane bladders. This, plus their treatment with anti-bactericides and anti-fungicides, makes them highly resistant to contamination. Sonoform also makes buoyancy compensators for the US Navy and oxygen re-breathers for the US Coast Guard. They have reported no problems.

Temple University professors Robert Leahy and Peter Lynch conducted a study of 25 subjects who each re-breathed air from a BC for one minute. An analysis of their exhaled air revealed no dangers, nor was there any compromise to their physiology.

Rinsing your BC frequently with a disinfectant will keep it safe. The Coast Guard uses Listerine. Or, you can use benzalkonium chloride, which can be purchased from a drug store under the brand name Zephrian chloride. An ounce of concentrate will make a gallon of solution. It will kill bacteria and fungi and it will not hurt the BC. Rinse with tap water after treating.

(Note: Although sodium metabisulfite is 100% effective in killing bacteria, do not use it. This compound has caused a fatal allergic reaction in a small percentage of people.)

An ideal solution

In 1983, at IQ14 in Chicago, Dennis Graver talked on "New Perspectives for Out-of-Air Emergencies". He gave these as considerations for an ideal out-of-air procedure:

1 That it can be used independently of other divers.
2 That breathing can be continuous.
3 That no extra gear is required.
4 That it is simple to accomplish.
5 That expense be minimal.
6 That it will assist buoyancy.

Graver stated that of all of the presently available methods of coping with an out-of-air emergency, BC breathing was closest to meeting this ideal.

BC breathing should be taught

BC breathing for self-rescue should be taught to new divers and practiced by experienced divers. Now that the newer BCs are coming through as standard with easy-to-clear inflator hose mouthpieces, inflator hoses that furnish fresh air from the tank and have no CO_2 cartridges, all of which makes it easier and safer to obtain air from our BCs, should not we add these life-saving BC breathing techniques to our repertoire of skills and be more confident while enjoying this fabulous underwater world?

Undercurrent Editor's Note: Of the agencies we spoke with, none officially teaches BC rebreathing in a basic course. NAUI discusses it in their textbook and teaches it in the advanced course. However, training director Jim Brown said that some instructors probably do teach it selectively in basic classes. "We believe that a diver has to be comfortable in the water and in diving", he told *Undercurrent*, "before they will have the presence of mind to use the technique. That is why we offer it in advanced courses."

John Gaffney of NASDS says that "We teach the safe second and have had no fatalities. We think that it is the best method".

SSI's Bob Clark said, "We do not teach the technique. We have established a pattern of "what to do's" and feel that if several options are given it tends to confuse people and reduces the time available for an action to be taken".

The YMCA decided several years ago not to teach it and has not reconsidered it.

PADI, IDEA, and MDEA failed to return our calls.

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BUOYANCY COMPENSATORS A PERSONAL OPINION

Bob Halstead

I am constantly being amused by people who, by trying to prevent a particular occurrence from happening, actually cause it to do so. I remember watching an approaching truck whilst driving along an unsealed road. The occupants of the front bench seat, of which there appeared to be half a dozen, concerned that a loose stone might be thrown up from my wheels and shatter their windscreen, reached forward, placed their hands on the inside of the screen and applied pressure. The result was, of course, that the windscreen popped from its mount and crashed onto the road.

Nowhere in diving does this irony occur more often than under the guise of SAFETY. Some joker recently invented a device that looked like a ping pong ball which when released underwater ascended at exactly 18 m per minute, thus enabling divers to make SAFE ascents. But divers following this marker would miss vital safety stops. Many authorities consider 18 m a minute too fast at the shallower depths anyway and retrieval of the balls on the surface would not seem to be without hazard either, or were they just left to pollute the ocean? Perhaps there is a world surplus of ping pong balls.

Remember the snorkel with the ping pong ball valve, designed to prevent snorkellers from choking on inhaled water but having the minor disadvantage of also inhibiting breathing? Fitted to a full face mask the device was reported to have suffocated several children. This rather dulls the amusement.

The J valve, fortunately now seldom seen, was the device invented to give a diver warning that the air supply was getting low. The results of attempting to pull the lever were often quite dramatic if the user had forgotten to place it in the right position at the beginning of a dive, or it had been knocked down during the dive, or if some home servicing had replaced the lever the wrong way up. Sometimes filling stations failed to place the lever down when filling the tank, resulting in short fills. The J valve was responsible for more than a few emergency ascents.

I also have grave doubts about the buddy system, buddies are completely unpredictable yet, for safety, we are told never to dive without one , give me a shark any time.

Now we have the BUOYANCY COMPENSATOR, called the BC by those that are unsure how to pronounce "buoyancy". These marvellous devices are supposed to make diving SAFE. Let us analyze that:-

1. It enables you to adjust your buoyancy underwater to achieve neutral buoyancy.

I agree that neutral buoyancy is extremely desirable for safe diving, but the only buoyancy adjustment that is needed during a dive is to compensate for compression of your wetsuit as you go deeper. In other words, if you are not wearing a wet suit and are already neutrally buoyant you do not need one. Probably choked up a few of the world's crummier instructors there did I? These incompetent fools teach our new divers to put heaps of lead on their belts (that gets them down) then blast air into their BC to achieve neutral buoyancy (to get them off the bottom) and call that buoyancy control.

Now, if you are wearing a wet suit, how much buoyancy compensation do you need? 15 kg, 20 kg, 25 kg? May I suggest FIVE kilograms as being more than adequate for most full 5 mm wetsuits. So what is all that buoyancy for?

2. It gives support on the surface.

There are various ways of interpreting this. First, let's try "life jacket". BCs started off as "adjustable buoyancy life jackets" and looked like the classic aircraft inflatable life jacket, many were exactly that, stolen from aircraft. Although no one has officially announced the demise of the BC as a life jacket, in fact that is what has happened. It no longer looks nor acts like a life jacket ... and I say, GOOD. If surfers and swimmers do not need life jackets, neither do divers.

However, the second interpretation, that of a device that assists a diver to rest or swim on the surface also needs some thought. A fully inflated BC on the surface restricts movement and can act as a sail or sea anchor. If any part of the inflated compensator shows above water it is contributing not to lift, but to drift. The air pocket has to be underwater to provide lift but then it also produces drag. So how much buoyancy do you really need on the surface? What are you doing on the surface anyway? If you are on the surface away from your boat or the shore, and have no air you obviously have made a MISTAKE. So let's be charitable, you made a mistake, just go on your back and kick your fins. Having trouble? Shame on you, OK, better drop your weight belt. Still not OK? Not fit, eh! Are you sure you should be diving? As a last resort put some air into your BC, five kilograms of lift is PLENTY.

So why are BCs made with 15, 20, and I even saw one advertised with 30, kilograms of buoyancy ??? The diver as a human lift bag perhaps? This is madness.

What else is a BC for?

To hold the tank? A standard backpack does that much better in most cases.

To organize your accessories? My advice is, if they need organizing, leave them in your dive bag.

To make money? Hmmm.

So lets look at this marvellous safety device and see how it makes diving safer:

It compensates for loss in buoyancy due to wetsuit compression, thus maintaining neutral buoyancy.

It can be used to give support to divers who should be playing golf.

Now let's look at some of the marvellous consequences due to the development of BCs.

1. So much drag is created that divers can no longer swim through the water above a nudibranch's pace without exhausting themselves. If you think I am exaggerating I suggest you get hold of a standard backpack and just try swimming a few lengths in a swimming pool using that instead. Feel the difference. As water is difficult stuff to move through, streamlining is essential. Did you ever figure out why Cousteau's Calypso divers look so good in the water? (Answer: they do not wear BCs and their tanks are streamlined with a fibreglass cover).

2. Divers cannot control themselves in a current, again because of drag.

3. Divers make fast, out of control, ascents, including missed safety stops, either because of expanding air deliberately put into the BC, but not vented on ascent, OR because of malfunction of the inflator causing a continuous or instantaneous inflation of the BC (or because the CO2 inflator snagged on a coral).

4. Divers on the surface are unable to swim back to the boat because of over-inflation of the vest causing restriction of movement and drag, and allowing any wind or current to take effect.

5. Divers get into distress through relying on a BC that ruptures, dumping the air and thus producing immediate negative buoyancy. I have seen many cases of bladder and hose failure.

6. Divers do not understand the gentle art of buoyancy control by changing breathing patterns. They are forever pumping the inflator and pulling the dump valve, which results in bloated monster divers smashing up the reef one moment and thrashing the surface the next. It also results in a rapid depletion of the air supply. How many times have you seen divers unable to descent, and causing a disturbance because they were unable to vent the air from their BC? (My oath!! Is that a killer whale eating a great white shark? Answer: No, it is just Jim trying to descend).

7. Divers are forced into an unnatural head up, feet down position, while trying to swim horizontally, by air in the compensator pooling around the neck. This is tiring and uncomfortable. The buoyancy needs to be near the weight belt.

8. Divers are much poorer - BCs cost a fortune!

So should I be so amused? Does the buoyancy compensator actually make diving MORE dangerous? Well, my answer is ... SOMETIMES. I find that sad because being able to compensate for compression of your wet suit IS useful, though personally I do not dive if the water temperature is less than 27°C so I do not wear one. And, if I am charitable, a little buoyancy on the surface IS also useful, particularly if you are unfit, cannot navigate and do not wish to keep buying new weight belts. I am aware that, statistically, the surface is a very dangerous place to be for a scuba diver, which is why it is important that divers be taught to AVOID the surface and return to the boat/shore underwater. It is also why it is essential that all divers have a "buddy" IN THE BOAT, looking out for them while they dive, who is able to move the boat to pick them up if necessary. I know divers that would not dive without a buddy, but they are quite happy to swim off leaving an empty boat. They are not thinking.

The problem here is the DESIGN of the BC rather than the concept. Some buoyancy compensators are much better than others, and this is what you should look for:

No internal bladder. The bladder causes the BC to bulge, giving no hope of streamlining, and these BCs trap water between the bladder and the outer skin which you drag around with you the whole dive.

A minimum of straps, flaps, pockets, velcro fastenings, D rings and valves, and definitely no CO_2 inflators. They all cause drag and clutter, and the CO_2 inflator is designed NEVER to go off at the right time.

A minimum of buoyancy. You do not need more than FIVE KILOGRAMS, and if this does suddenly dump, or inflate, it should not be disastrous. It is possible that divers wearing very thick wet suits, or dry suits, may need a little more buoyancy. I admit to knowing nothing about diving requiring these types of suits ... except that I never intend to try it. I once tried cold water diving (23°C) and hated it.

A snug fit to the body. If it is loose the tank will float around while you are diving, and yet again, you will have more drag. Try the size smaller than the one you think you need.

Good quality gear to minimize the chance of failure.

Try to find a BC that has been designed by a diver. Contrary to popular belief, most dive gear is designed by people who rarely, if ever, dive, which is why most of it works so poorly in the water. We have had divers bring new

BCs that were impossible to use, and were most depressed until we loaned them the spares we keep on the boat.

Needless to say, the ideal BC has not yet been made, but it has been designed - Mike Emmerman, Director of Research of Lifeguard Systems Inc. and I sat down and designed it. It was not a difficult exercise and it turned out that one size could fit all (after appropriate adjusting and trimming of straps), and it would probably retail at less than $150.00 and could be available in 59 colours. This would be an advantage to divers if not to manufacturers, but if there is a manufacturer reading this who is interested, please contact me immediately. Just imagine what you could do if you had one. Why, all you would have to do would be to throw away that stupid octopus, buy a Spare Air and a Safety Sausage, and you would start to look like a real diver, not a dive store dummy!

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WHAT TO DO IF YOU'RE ADRIFT

Dennis Graver

Despite plans and precautions, it is possible that you may one day find yourself adrift in the water. Divers have found themselves carried away by unexpected currents, left behind by a boat whose crew fails to count heads, or even victimised by a sunken boat.

To survive for any length of time, you need a positive mental attitude. And there are steps you can take to greatly increase your chances of survival.

The boat remains, but you're adrift

The first step is prevention. Plan ahead. Discuss sea survival and the rescue plan with a crew before diving. One can also file a "float plan" with a friend who will have enough information to be able to send assistance in the event you do not make contact by a certain time.

You should carry one or more long-range signalling devices in your BC pocket. You may not need much if diving off the Seven Mile Beach at Grand Cayman, but if you are in the Coral Sea you can not be too prepared.

an effective whistle;
a stainless steel mirror;
a light plastic "Safety Sausage"* which, when inflated from the regulator or lungs, stands ten feet above the water line;
small, waterproof flares and launcher (available at marine stores);
smoke bombs;
a waterproof strobe light.

The second step is prevention. Do all you can to prevent being carried away by a current. Be sure there is a long trail line behind the boat when a current is present. Dive up-current. When you surface upstream from the boat, swim so as to align yourself with the vessel long before you approach it.

If you are unable to reach the boat and you have air left in your tank, take a bearing that will place you ahead of the boat when you resurface. Then, return to the bottom where the current is weakest, and move against the current there. If you are on the surface and diving in kelp, or near rocks and coral, grab hold to maintain your position and conserve energy.

Most important, signal the boat at the first indication of difficulty making headway against the current.

The third step is to have someone quickly pick you up if you are swept away. Leave someone in the boat! Buddy teams can and should alternate dives in areas where currents are likely to cause problems. Many non-divers will gladly go along and be content to fish from the boat while you dive. Tell the person remaining in the boat your dive plan, including the time you plan to surface.

The final step is to cope with being adrift. It is best to simply make yourself buoyant, conserve energy, try to gain attention and wait for assistance. Swimming quickly leads to exhaustion. Your equipment should be retained if possible because it may be useful for survival. Your empty scuba tank and weights may be discarded if they become burdensome.

If you need to attract attention, try reflecting the sun from your hand-held face plate. Or splash water high into the air. If you have a camera, hold it high and wave it. Or flash the strobe. Remember, however, expendable signalling devices should be used only when there is a strong likelihood they will be seen.

As the length of your ordeal increases, the greater the loss of body heat. Minimise heat loss by keeping your head out of the water and using the Heat Escape Lessening Position (HELP). This involves keeping your arms close to

your sides and your legs pulled up to your chest to insulate the high heat loss areas of your underarms and abdomen. You should save your weight belt if you discard your weights because the belt may be used to help you maintain your knees in the HELP position.

If your boat sinks

Imagine being miles from land and aboard a dive boat that is sinking! As a diver, what actions would you take? There are quite a few things you should know and can do.

If time permits, don your gear before leaving the vessel. If not, at least ensure your gear bag will float. By packing your wet suit and a partially inflated BC in your bag (forget your weight belt) you can be confident your bag will float. A friend of mine stores all of his dive gear in a plastic trash can, and he happened to be aboard a charter boat when it swamped and sank. He donned his wet suit and fins, put all of his possessions, including his clothing, into the trash can, and swam it to shore with no loss whatsoever!

Wearing diving equipment is not the best option, however. You are better off out of the water than in it, even if fully equipped. A life raft or pieces of wreckage assembled into a raft can mean the difference when long periods of time are involved. Wear your diving equipment initially, but only as a backup to a better alternative.

The best sinking boat story I have heard involves several divers in Florida who returned underwater at the end of a dive to find their boat on the bottom! They managed to raise the sunken vessel, bail it out, and obtain assistance! That's putting diving gear to good use.....

If you can not salvage your boat, move upwind from the wreckage to avoid fuel on the water. Collect everything possible to form a raft, and remain near the wreckage to increase your chances of being seen.

Water is critical to your survival. You can go many days without food, but very few without water.

If you have time while the boat is sinking, rinse the inside of your BC, then partially fill it with fresh water. This simple act can greatly extend your survival time at sea. Drinking salt water is not recommended because it upsets the chemical balance of the body and increases the need for fresh water. The use of motion sickness medications is recommended because vomiting reduces precious body fluids.

If no water is available, food should not be consumed, since its digestion requires fluids and hastens dehydration. Eat only if you have at least two quarts of water per day. If fish is eaten, consume on a small amount and wait 12 hours. If no reaction occurs, the remainder of the fish may be eaten. Two-thirds of all seaweed may be eaten, but only in small quantities and in the same manner as that recommended for fish.

The keys to survival

The keys to surface survival are preparation, patience and the ability to think. You know how to prepare as far as equipment goes, so make it a priority. You can also prepare yourself mentally with pre-visualisation of potential situations. Imagine as vividly as you can being adrift with no boat or land in sight. How would you feel? What would you do as time went by? Live the experience and cope with it in your mid, then if it ever should happen to you, it will seem as though you have been there before and already know what to do.

Just like every problem you can encounter in diving, the keys to dealing with being adrift are knowledge and preparation. You now have the knowledge. But are you prepared?

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* *Safety Sausages are available in Australia and New Zealand from safety conscious dive shops. The are also available from themanufacturer, Bob Begg, PO Box 5216, Dunedin, New Zealand and from the Australian distributor, Diving Security, 34 College Street, Hawthorn, Victoria 3122, Australia.*

HYPERBARIC MEDICINE A FUTURE PERSPECTIVE

Des Gorman

Introduction

Hyperbaric medicine comprises the treatment of diseases and injuries with a raised ambient pressure, and usually involves the administration of an elevated partial pressure of oxygen, hyperbaric oxygen (HBO). It can be

used to either directly affect a disease process (e.g. reduction of bubble volume in decompression sickness) or to enhance host function (e.g. restoration of macrophage activity in ischaemic tissues). Although such treatment is definitive for several diseases, the future scope of hyperbaric medicine is uncertain, and will depend to a large extent on how the discipline interfaces with, and integrates into, conventional medicine. It is this process that should dominate current discussions of hyperbaric medicine.

Any future perspective, unless written by a clairvoyant, is essentially a description of the present with a series of predictions to enhance the author's theme.[1] Such descriptions and predictions are by necessity global, and yet the issues that determine the success of hyperbaric medicine in a specific area are often local. For example, in Australia and New Zealand, there are respectively an estimated 400,000 and 150,000 active recreational divers. This represents 2.5% and 5% of the populations of these countries, and it is clear that hyperbaric units can be justified there on the basis of diving accidents alone. However, the same is not true for much of Europe and North America.

Regional debates about the appropriate role of hyperbaric medicine in modern clinical practice have little to do with its actual risks and benefits, but much to do with its cost-effectiveness and local medical politics. Hyperbaric medicine threatens existing public health funding, and hyperbaric physicians should expect considerable resistance from both health bureaucrats and medical practitioners in other areas of medicine who are competing for the same funds.

Lay persons learning of the spectacular successes attributed to the hyperbaric treatment of patients other than divers are often surprised to learn that hyperbaric medicine is not lauded by the rest of the medical community. Indeed, it has only limited acceptance and in many areas of the world it is not even tolerated. This is particularly true in the western medical community.

A recent review of hyperbaric medicine[2] by an Australian medical student, who had never seen a recompression chamber, and a Stanford University Professor of Medicine and Physiology, was notable for the following. Firstly, it purported to be a risk-benefit analysis of hyperbaric medicine, and yet had none of the essential components of such an analysis. Secondly, it argued, on the basis of a very limited literature review, that available data only supported a role for hyperbaric therapy in the treatment of decompression sickness. Thirdly, the authors suggested that unless more data became available, that the baby, the legitimate uses of hyperbaric medicine, was in danger of being thrown out with the bath water. Even allowing for the negligible scientific merit of this paper, the final point it raises is unquestionably valid. Unless the hyperbaric community confronts the reasons for the limited acceptance of hyperbaric medicine, the baby may well end up with the bath water.

Proposed reasons for the limited acceptance of hyperbaric medicine

The limited acceptance of hyperbaric medicine may be due, at least in part, to:

(a) the conservatism of the medical community;
(b) the pharmaceutical bias of western medicine;
(c) the involvement of charlatans and adventurers in hyperbaric medicine, and bizarre indications for treatment;
(d) the hyperbaric treatment of patients with multiple sclerosis;
(e) the domination of hyperbaric medicine during much of its history by the military;
(f) an undue emphasis on the medicine of diving;
(g) vocal, disillusioned hyperbaric medicine pioneers;
(h) the expense of hyperbaric facilities and hyperbaric treatment; and, most importantly,
(i) a lack of controlled dose-response data.

The conservatism of the medical community

The conservatism of the medical community translates into a reluctance to accept new directions. Simply, hyperbaric medicine is the new kid on the block, and a different set of rules apply to such newcomers in comparison to established areas of medicine. Hyperbaric physicians often find themselves arguing the inequity of this situation. For example, hyperbaric medicine is often criticised by its opponents for a lack of controlled clinical data. It is easy to counter this with techniques such as comparing the data that support the hyperbaric treatment of clostridial gas gangrene with that which support the conventional treatment of brain-injured patients. The management of brain injuries is a suitable example as none of the accepted therapies, such as hyperventilation or diuretics, have been shown to make any difference to eventual patient outcome.

Additional ammunition for the pro-hyperbaric argument can be derived from a survey of medical journals such as the one summarised in Table 1. The journals reviewed were some of the popular current issues in the Royal Adelaide Hospital Library on 1st July 1988. it is clear from such surveys that very few clinical reports in the general medical literature are randomised, controlled, prospective studies, and that the criticisms of hyperbaric medicine in this regard are hypocritical. Regardless, such hypocrisy is unlikely to change, and the hyperbaric community must learn to live with this inequity, to initiate appropriate controlled clinical trials supported by in-vitro and in-vivo experimentation, and to offer hyperbaric therapy only to those patients with

TABLE 1

SURVEY OF CLINICAL PAPERS IN AUSTRALASIAN AND INTERNATIONAL JOURNALS

	Australasian Journals N = 4	International Journals N = 10
Case Reports (< 6)	35%	15%
Clinical Survey	50%	55%
Own-control	15%	10%
Retrospective Controls	0	10%
Prospective Controls	0	10%

Australasian Journals:
MJA, NZ Med J, ANZ J Surg, ANZ J Med.

International Journals:
Neurology, Stroke, Chest, Arch Intern Med, JAMA, Arch Surg, Ann Surg, NEJM, BMJ, Lancet.

diseases for which there is reasonable supporting logic or data.

The pharmaceutical bias of western medicine

This reluctance to accept new directions in medicine is compounded by the pharmaceutical bias of the medical community in the western world. This bias is actively maintained by the pharmaceutical industry, at considerable financial cost. Simply, when western medical practitioners fail to achieve the desired result with a particular drug, they automatically resort to a second, or third, or fourth drug. Unfortunately, this pharmaceutical bias is accompanied by an intrinsic distrust of physical medicine such as acupuncture and hyperbaric medicine. This distrust is not reduced by recent references in the hyperbaric literature to oxygen as a drug. However, this bias against physical therapy is not present in Eastern Europe and the Orient. Indeed, the acceptance of physical medicine in Asia probably underlies the remarkable range of diseases treated by hyperbaric physicians in China. The pharmaceutical bias of the western medical community will not be easily overcome. Nevertheless, it requires that hyperbaric medicine be practised in a regimented, scientific, and conservative fashion.

The involvement of charlatans and adventurers in hyperbaric medicine, and bizarre indications for treatment

The history of hyperbaric medicine, in particular the frequent involvement of charlatans and adventurers, and the persistence of bizarre indications for therapy has brought great discredit to the field, and further limited its acceptance. This probably reached its absurd extreme with the construction of a hyperbaric hotel, and with the advocacy of HBO as an effective treatment of stress, ageing, hair loss, reduced libido, and decreased sexual prowess. Similarly, the very public use of hyperbaric oxygen by a famous eccentric such as Michael Jackson to reduce his rate of ageing does nothing to increase its credibility. Again, the solution is to restrict practice to those diseases where there is supporting logic or data.

The hyperbaric treatment of patients with multiple sclerosis

The standing of hyperbaric medicine is further eroded by the treatment of multiple sclerosis sufferers. While this is an emotive and controversial issue, and the role of HBO in the treatment of this disease is yet to be established, the general medical community is fully aware that the majority of published studies have shown no significant role for HBO. These studies may be flawed, but the effect on both the image and perceived standards of hyperbaric practice is nevertheless negative. If hyperbaric medicine is to fulfil its potential, it must become a legitimate and accepted form of medical practice. It follows that in the context of available data, that the treatment of multiple sclerosis sufferers with HBO should only be undertaken as part of prospective randomised studies that investigate the widest possible range of oxygen doses.

Military and diving medicine domination and bias

Other historical limits to the integration of hyperbaric medicine into the mainstream include: that to a large extent it has been dominated by the military; and that the majority of hyperbaric physicians are primarily and originally interested in the medicine of diving. This is reflected in the standard of the presentations at hyperbaric conferences where in general the diving medicine and physiology papers are of a significantly higher scientific standard than those from the general hyperbaric area.

Vocal disillusioned hyperbaric medicine pioneers

The history and development of hyperbaric medicine has also generated some of the discipline's sternest and most damaging critics. Many of these were among the pioneers of hyperbaric medicine and saw it as a panacea, or at the least as a solution to the more difficult areas in emergency medicine such as brain and spinal cord injuries. When their efforts to establish a significant role for hyperbaric therapy in these areas failed, they became disillusioned, critical, and cynical, and lost sight of the important indications for hyperbaric medicine that had become established, such as

gas gangrene and arterial gas embolism. In many areas, the acceptance of hyperbaric medicine has been significantly delayed as a result of the actions and comments of these pioneers.

The expense of hyperbaric facilities and hyperbaric treatment

To many health bureaucrats, advances in medical technology simply represent increased expense. Such bureaucrats apply this logic to hyperbaric medicine, and given that most health budgets are allocated annually, and that much of the cost-savings incurred by hyperbaric treatment occurs in subsequent years, these bureaucrats remain unimpressed by the argument that hyperbaric therapy is often cost-effective in the long term. This is particularly true in the United States of America where the cost to the health insurance industry of an individual patient treatment varies from between US$250 and US$400. Such costs, whether reasonable or not, must put both hyperbaric medicine and its supporting literature under the health bureaucrat and insurance company microscope, and encourage the type of cynical review of hyperbaric medicine recently published in the prestigious journal, "Chest".[2] Nevertheless, the argument that hyperbaric medicine can be cost-effective is reasonable, and one that should continue to be developed. For example, the impact of the data from the Wilfred Hall Medical Centre showing a significant increase in resolution rate for patients with osteoradionecrosis of the mandible when they are treated with a combined regimen of surgery and HBO in comparison to either treatment alone, is dramatically increased by the observation that this improved outcome is achieved at a significantly reduced cost.[3]

A lack of controlled, dose-response data

The human metabolic reliance on oxygen is well understood. Similarly, the ability to manipulate the magnitude and direction of biological processes by altering the ambient oxygen tension is established. Because of the observation that many diseases have a final common pathway of ischaemia, it is clear that there are many potential applications for HBO.

Despite this potential, at this time, the available data establish an unequivocal role for HBO only in the treatment of decompression sickness, arterial gas embolism, clostridial gas gangrene, carbon monoxide (CO) intoxication, and osteoradionecrosis of the mandible. Among these indications considerable research is still required because data are often limited to a single oxygen dose. Furthermore, good criteria for patient selection frequently do not exist. It must be conceded though, that the lethality of some of these diseases, and the consequent difficulty in gaining ethical approval, may limit the scope of planned research. In these instances, there is an obvious role for a formal risk-benefit analysis.[4] For example, a preliminary, very conservative, analysis, done in the Hyperbaric Medical Unit at the Royal Adelaide Hospital, of clostridial gas gangrene survival data has shown that if the probability of gas gangrene is greater than 5% that the administration of HBO will save the lives of a greater number of patients than it will kill. It must also be conceded that in most of the conditions listed above, that HBO is the definitive treatment, is life-saving and prevents major morbidity. As effective alternative treatments do not exist, experience suggests that on the basis of these diseases alone, every population base of one million people should have access to a properly equipped hyperbaric unit, and that this unit must be interfaced with an intensive care service.

Hyperbaric oxygen therapy has also been demonstrated to have an important role in the promotion of wound-healing in specific contexts. For example, a prospective controlled study has shown a significant benefit for hyperbaric therapy in healing of diabetic ulcers.[5] Data exist, with historical controls, to show a role in the treatment of chronic refractory osteomyelitis. Perhaps, the wound-healing application of greatest potential merit is that of promoting the healing of thermal burn injuries. The data from animal models of thermal burns establish that HBO not only accelerates the healing of burn wounds, but also reduces the consequent mortality and retards the progression of partial-thickness burns to full-thickness. The data from burnt humans is consistent with these findings, but, like the assessments of other treatment modalities for human burns, are limited by inadequate control data and inaccurate methods of assessing burn wound depth.

Encouraging results have also been obtained from animal and/or human work with HBO in the treatment of compartment syndromes, sudden hearing loss, peptic ulceration, various poisonings and intoxications, soft tissue gangrenous infections other than clostridial myonecrosis, and non-healing bone fractures. Nevertheless, it is clear that while the potential applications of hyperbaric therapy are many, the established indications are few. There is an obvious and urgent need and opportunity for further research.

It is also apparent that the need for adequately controlled studies is not being met. For example, an analysis of the papers presented at the 1988 scientific meeting of the Undersea and Hyperbaric Medicine Society, held in New Orleans, shows that there were no prospective controlled studies reported (Table 2).[6] In the area of diving medicine and physiology there were 52 clinical papers. Three of these were case reports, 14 were clinical surveys, 32 used subjects as their own controls, and one used a retrospective control group. In the area of hyperbaric medicine and physiology, there were 17 clinical papers. Four of these were case reports, 8 were clinical surveys, only one used subjects as their own controls, and 4 used retrospective controls. Data

TABLE 2

SURVEY OF CLINICAL PAPERS PRESENTED TO THE 1988 UNDERSEA AND HYPERBARIC MEDICINE SOCIETY ANNUAL SCIENTIFIC MEETING IN NEW ORLEANS

	Diving Medicine N = 52	Hyperbaric Medicine N = 17
Case Reports	3	4
Clinical Survey	14	8
Own-control	34	1
Retrospective Controls	1	4
Prospective Controls	0	0

of this nature are unlikely to change the current standing of hyperbaric medicine. Perhaps the most disappointing aspect of these papers is, however, that the researchers continue to overlook the importance of establishing the dose-response relationship for oxygen and the condition being studied.

The first aim of hyperbaric research should be to establish oxygen dose-responses for those conditions that are accepted indications for hyperbaric therapy so that their treatment can be optimised. It is not justifiable to adopt the attitude that once a response to oxygen is established that more oxygen will have more of the same effect. An increase of the oxygen tension can actually reverse the direction of a biological reaction.[7] Also, attention must be paid to the frequency of hyperbaric administration.

The second research aim should be to rigorously test the use of hyperbaric therapy in those diseases where data are supportive but do not, as yet, establish a definitive role. These tests should compare financial cost and potential hazards with effect. For many conditions where heterogeneity is considerable, this will necessitate multicentre trials. It is most important that these trials not be based on a single oxygen dose, even a perceived megadose. In the past, such an approach has contributed to the rejection of hyperbaric therapy as a treatment for the disease in question. Varied experimental designs (e.g. disease definitions and oxygen-doses) have also resulted in different studies arriving at opposing conclusions about the efficacy of hyperbaric medicine.

Summary and Conclusion

There are obvious, recognisable reasons why hyperbaric medicine is not widely accepted by medical practitioners in other disciplines. A conservative and scientific approach to hyperbaric clinical practice is needed if that acceptance is to be forthcoming. The future of hyperbaric medicine depends upon a rigorous effort to establish adequate dose-response data, and on prospective, well-controlled studies being carried out.

Regardless of the eventual roles that may be established for hyperbaric therapy, appropriate facilities should be introduced for the treatment of patients with those diseases which are already unequivocal indications for hyperbaric therapy. The uncertainties that exist about the role of HBO in the treatment of brain-injuries and multiple sclerosis should not prevent or retard the treatment of patients with diseases such as clostridial myonecrosis and CO intoxication.

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We will be presenting other papers from the EUBS Aberdeen meeting in this and later issues of the SPUMS Journal. The first is Dr Lambertsen's paper on human oxygen tolerance which appears on page 109.

PHYSIOLOGIC FACTORS IN HUMAN ORGAN OXYGEN TOLERANCE EXTENSION

Christian J Lambertsen

Introduction

The respiration of oxygen over a range of partial pressures higher than in the natural environment has expanding usefulness in health and disease. It facilitates diving of many forms, improves safety in decompression after diving, provides for denitrogenation of the astronaut to prevent aerospace decompression sickness, and is the key to therapy of diving and iatrogenic gas lesion diseases. In the continuum of general and hyperbaric medicine, it is essential for sustaining viability of damaged or diseased tissues not adequately oxygenated at natural oxygen pressures.

Over the entire range of its key roles in clinical and operational procedures, the pressure and duration of tolerable exposure to oxygen is limited by adverse effects on multiple chemical targets, cells, tissues, and organ functions. The rates of development and the qualitative expressions of these adverse effects are different at different respired oxygen pressures.

Successful extension of oxygen tolerance, as by slowing the rate of development of adverse effects, will further expand the medical and operational usefulness and safety of oxygen in normal, hypobaric and hyperbaric environments.

A pre-requisite baseline for overall extension of oxygen tolerance is the quantitative investigation of early stages of toxic oxygen effects upon specific chemical and composite functions of multiple organ systems, including rates of development and rates of recovery. The practicality of limited oxygen tolerance extension by systematic interruption of oxygen exposure has been demonstrated and the procedure widely used. Broad present goals are to establish oxygen tolerance for specific tissues, and to optimise extension of tolerance over the full range of useful oxygen pressures. The result could open the path to large advances in diving, decompression and therapeutic procedures.

Tolerance to physiological effects of oxygen

The several important "physiological" effects of oxygen are, by definition, not toxic effects. They occur promptly, and most but not all increase with the degree of increase in respiratory oxygen pressure. They produce no important subjective symptoms; their effects are magnified when superimposed on a pre-existing hypoxic state, and they are fully reversible on discontinuing hyperoxic exposure.

Established examples of such physiological effects, most interactive and self-limited, include:[1,2]

The "biological burning" of normal O_2 metabolism;
Inactivation of haemoglobin roles in O_2 and CO_2 transport;
Physical transport of oxygen;
Generalised tissue hypercapnia and acidosis;
Arterial hypocapnia and alkalosis;
Respiratory stimulation;
Respiratory chemoreflex "suppression";
Bradycardia;
Alterations of vascular tone;
Haematopoietic suppression;
Displacement of inert gases.

These and other harmless physiological effects are present throughout continuous toxic exposures to hyperoxia, cycle with intermittent hyperoxygenation, and are not part of post-exposure residual effects of oxygen poisoning. Physiological factors do, however, have prominent influence upon specific aspects of oxygen tolerance and extension of oxygen tolerance.

The meaning of tolerance to oxygen

Tolerance to oxygen primarily means tolerance to the toxic actions of oxygen, since the physiological effects (except for suppression of red cell formation) exert no prolonged consequence.

The *Terminology* of oxygen poisoning and its cascading results continues to be very loose, and requires tightening.[2] Examples of terms are "Oxygen Toxicity", "Oxygen Poisoning", "Oxygen Tolerance", "Sensitivity", "Latent Period", "Adaptation", "Prevention", "Reversal". The oxygen molecule is not toxic but the term "oxygen poisoning" is established. Oxygen induces species which are "toxic" and which "poison". Very different degrees of poisoning are "tolerable" in different tissues when increased oxygen pressures are used for important operational or medical situations. Discrete biochemical targets may be more or less "sensitive" or "susceptible" to oxygen poisoning. For tissues or organs it is necessary to separate concepts of differences in "sensitivity" from differences in actual "dose" of oxygen exposure and the detectability of expressed effects.[1]

In undersea, aerospace and hyperbaric medical activity a positive emphasis upon extending oxygen tolerance is desirable, as opposed to the restrictive fear of oxygen poisoning. This is not a small distinction, and the underlying chemical and physiological factors determining tolerance are clearly multiple. They include: What is the "Oxygen Dose"? What are the biologic targets? How fast does poisoning develop? How fast does recovery occur? Does the poisoning matter?

The targets and expressions of adverse effects

Sequential Effects in acute oxygen poisoning at high inspired oxygen pressures can be considered as beginning immediately, and proceeding concurrently, in multiple systems at different rates. For example:

Oxygen free radical formation is accelerated;
Scavenger efficiency for free radicals is overloaded;
Attacks occur on susceptible chemical sites (more than one at once);
Effects occur on critical enzymatic reactions (more than one at once).

Basic Cellular Expressions of biochemical disruptions take time to become evident, and depend upon the characteristics of cell normal function. For example:

Cell energy metabolism;
Cell membranal integrity;
Membrane electric potential development;
Neurotransmission of impulses;
Secretion;
Contraction;
Synthesis.

Observable *Symptoms or Detectable Functional Decrements* eventually emerge, to include:

Sensory;
Respiratory;
Cardio-circulatory;
Neuromuscular;
Consciousness.

The actual basic effect and observable result will depend upon specific doses and durations of target single exposures, the number of exposures, and the patterns of intervals between.

The instantaneous "dose" of oxygen at a target site

The dose (partial pressure) of oxygen at any instant is far from uniform within an organ or even within a cell, even at a constant ambient or breathing apparatus oxygen pressure.[1]

Physiological Factors Affecting Oxygen Dose at the site of toxic biochemical effect in normal individuals include:

Relations of arterial oxygen content and oxygen pressure (Figures 1 and 2);
Tissue blood flow in different organs (Fig. 3);
Maximal oxygen partial pressure induced (inspired, alveolar, arterial) (Fig. 4);
Oxygen flow (arterial O_2 content times blood flow);

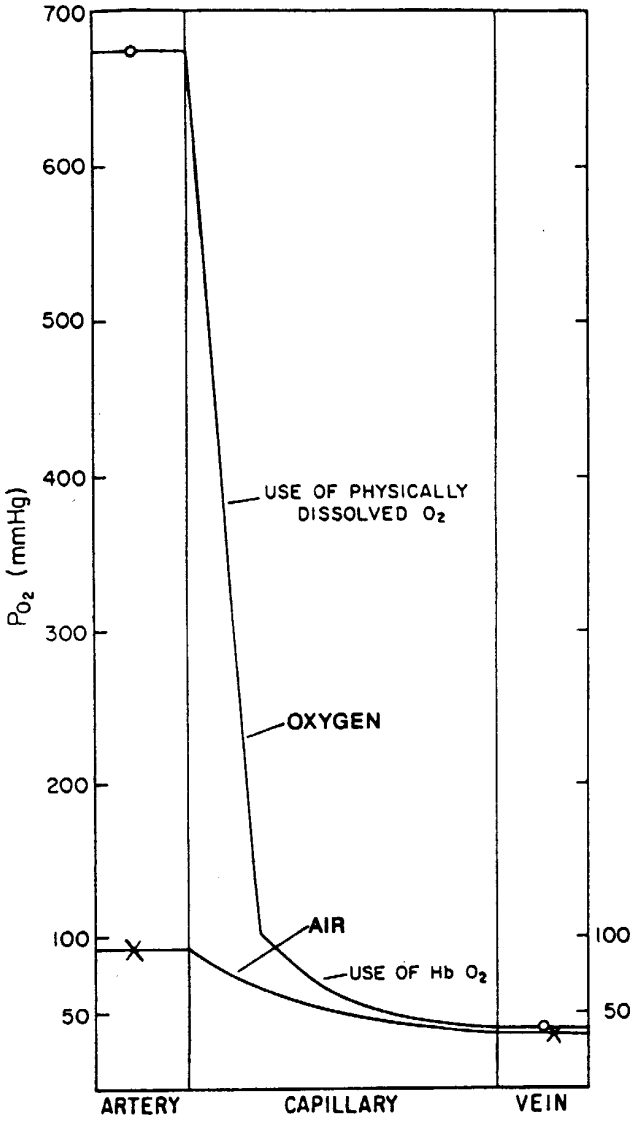

FIGURE 2

DERIVED PATTERNS OF PO_2 FALL DURING BLOOD PASSAGE THROUGH THE BRAIN CAPILLARY BED

Rates of decrease in blood PO_2 are computed for normal air breathing and 100% oxygen breathing at sea level. As arterial blood enters the capillary, physically dissolved oxygen diffuses down the PO_2 gradient to metabolic sites. Release of oxygen from haemoglobin limits the rate and degree of PO_2 fall both during air breathing and oxygen breathing. However, in oxygen breathing the metabolic use solely of physically dissolved oxygen in the first phase of transit rapidly lowers blood PO_2. Cells supplied from the "arterial" end of the capillary bed will be exposed to higher PO_2 than cells supplied from near the venous end.[28]

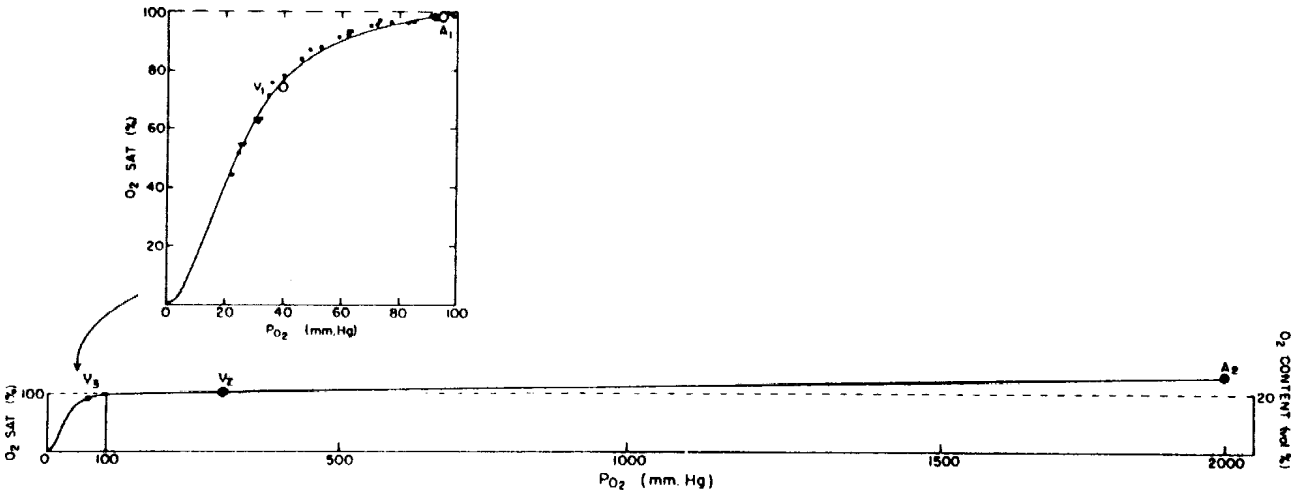

FIGURE 1. OXYGEN UPTAKE AND LIBERATION CURVES FOR BLOOD AT NORMAL AND HIGH INSPIRED OXYGEN PRESSURES. (DATA FROM NORMAL MEN AT REST)

In the upper diagram, A_1-V_1 indicates, for air breathing at sea level, the expected transition of PO_2 and percent Hb saturation from measured normal arterial values to an estimated normal value in mixed venous blood. The data points represent observations on arterial blood during administration of air and gas mixtures low in O_2 content. The lower diagram shows the additional O_2 uptake by arterial blood as inspired PO_2 is raised to 3.5 ATA. Somewhat above 100 mm Hg PO_2, haemoglobin becomes completely saturated; hence the slope of the O_2 uptake curve represents the physical solubility of oxygen per mm Hg PO_2. A_2-V_2 indicates the degree of change in PO_2 across the brain that would be predicted on the basis of the same degree of (A-V)O_2 extraction found during air breathing. This high level of venous PO_2 does not occur and A_2-V_3 shows the pattern of O_2 liberation that is found by actual experiment. The greater magnitude of PO_2 fall is due to the considerable decrease in brain blood flow with O_2 administration. From Lambertsen.[1]

Differences in cellular/tissue oxygen consumption;
Diffusion distance (capillary to chemical target site).

Such factors can generally be measured or calculated only as organ averages, within which wide differences in dose must be considered to exist.

The Highest Sustained Tissue Oxygen Dose At Any Inspired Oxygen Pressure is established by anatomical considerations which essentially eliminate factors of blood flow and tissue metabolism. Nearly uniform, highest oxygen exposure exists for:

Epithelium of the upper respiratory tract, larynx, trachea, and bronchi, bronchioles;
Gas exchanging surfaces of the lung (alveolar epithelium, capillary endothelial cells);
Endothelium of pulmonary veins, left heart, and sytemic arterials;
Renal glomerular capillaries;
Carotid body;
Arterial end of each capillary, of each organ, and each tissue.

Except for the sensations elicited by oxygen effect on superficial respiratory tract innervation, the consequences of possibly equivalent toxic action on the other anatomically "high dose" sites go unfelt and unmeasured, with no empirical indication at this time that they are important relative to the more evidently affected pulmonary tissues.

The Range of Specific Localised Oxygen Pressures is large among cells of any single perfused tissue, including those of lung, brain structures, liver and heart (Fig. 3).[1] Measurements of oxygen pressure in venous blood represent weighted averages of final oxygen pressures in many tissue capillaries. Exposures of cells served by this blood must range from near-arterial levels of oxygen pressure to pressures less than that of the venous effluent (Fig. 5).

Unique features of circulatory anatomy affect the patterns of oxygen exposure in several vital tissues. For example:

In the lung the dual circulation (pulmonary and bronchial) presents mixed venous blood and arterial blood input to the same organ.

The dual circulation of the liver presents portal venous blood and hepatic artery blood.

In the kidney the cells of glomerular filtration are exposed to the equivalent of arterial blood throughout their length. The metabolism of

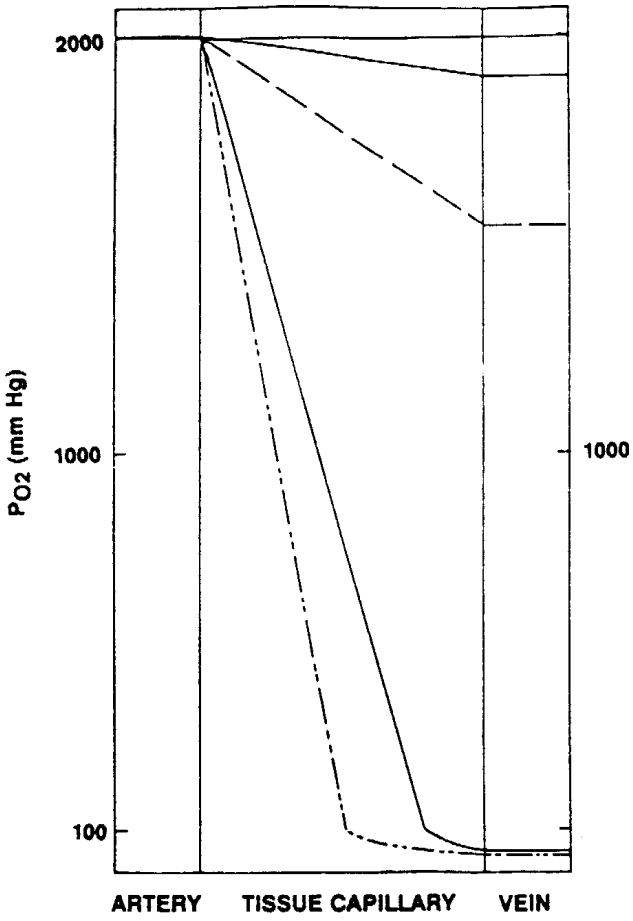

FIGURE 3
EXTREMES OF TRANS-CAPILLARY BLOOD PO_2 AT HIGH INSPIRED OXYGEN PRESSURE

Computed for oxygen breathing at 3.5 ATA. Modified from Lambersten.[1] The alveoli and renal glomeruli have a PO_2 of 2,000, the line at the top of the artery column. The other four lines show the PO_2 of the carotid body (top), kidney tubules (dashed), brain (lower solid) and heart (dots and dashes).

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the tubular cells reduces their mean exposure to oxygen (Fig. 3).

The endothelium of the systemic venous system is probably not exposed to toxic oxygen pressures at useful inspired oxygen pressures.

The red and white cells of the blood are exposed several times a minute to an oxygen pressure fluctuating in a second from arterial to venous, and then back from venous to arterial levels.

The interest in and importance of the many conceptual factors determining true oxygen dose and its broad range is not that such considerations should now limit practical use of hyperoxia in diving, or decompression or therapy. Their importance is that "micro-anatomical dose" of oxygen must be recognised as relevant to any fundamental investigations of *oxygen tolerance* of cells or organs, and any investigations of *extension* of *oxygen tolerance*. In-situ measurement of masked oxygen doses and their effects will not be easy when in any organ, including the brain, only a small fraction of its functioning cell mass is exposed to acutely toxic oxygen pressures. Most biochemical investigations in vitro or in vivo take little account of this physiological reality.

The requirement for definition of organ oxygen tolerance

The described masking of oxygen dose and effect, and the probability of symptomatically unrecognisable forms of oxygen poisoning, have made it sensible to search in multiple human organ systems for adverse effects of oxygen. Prominent functional derangements during severe hyperoxic exposures have been found in cardiovascular, visual, pulmonary and central nervous systems, with other foci of simultaneous investigation not demonstrably affected.[3]

A special finding in exceptionally prolonged exposures of man to different high pressures of oxygen has been the apparent patho-physiological interactions of oxygen effect upon different organs, such as brain and heart, or brain and lung.[3-5] Other powerful interactions may exist to be uncovered in severe oxygen poisoning. They are unlikely in the general present range of oxygen usage.

Physiological modifications of oxygen tolerance

Physiological factors can influence development of the two most grossly obvious components of organ oxygen poisoning (pulmonary effect and generalised convulsion). For example:

Interrupting exposure to hyperoxia extends both pulmonary and central nervous system oxygen tolerance.

Small (physiological) or large increases in alveolar/arterial carbon dioxide pressure hasten onset of the convulsive expression of central nervous system oxygen poisoning. This is related to an increase in oxygen flow and dose in brain. Causes of alveolar/arterial hypercapnia can include subnormal alveolar ventilation at rest or in physical work. These may be voluntary, or due to respiratory gas density, or due to extrinsic factors of life-support apparatus or environment.

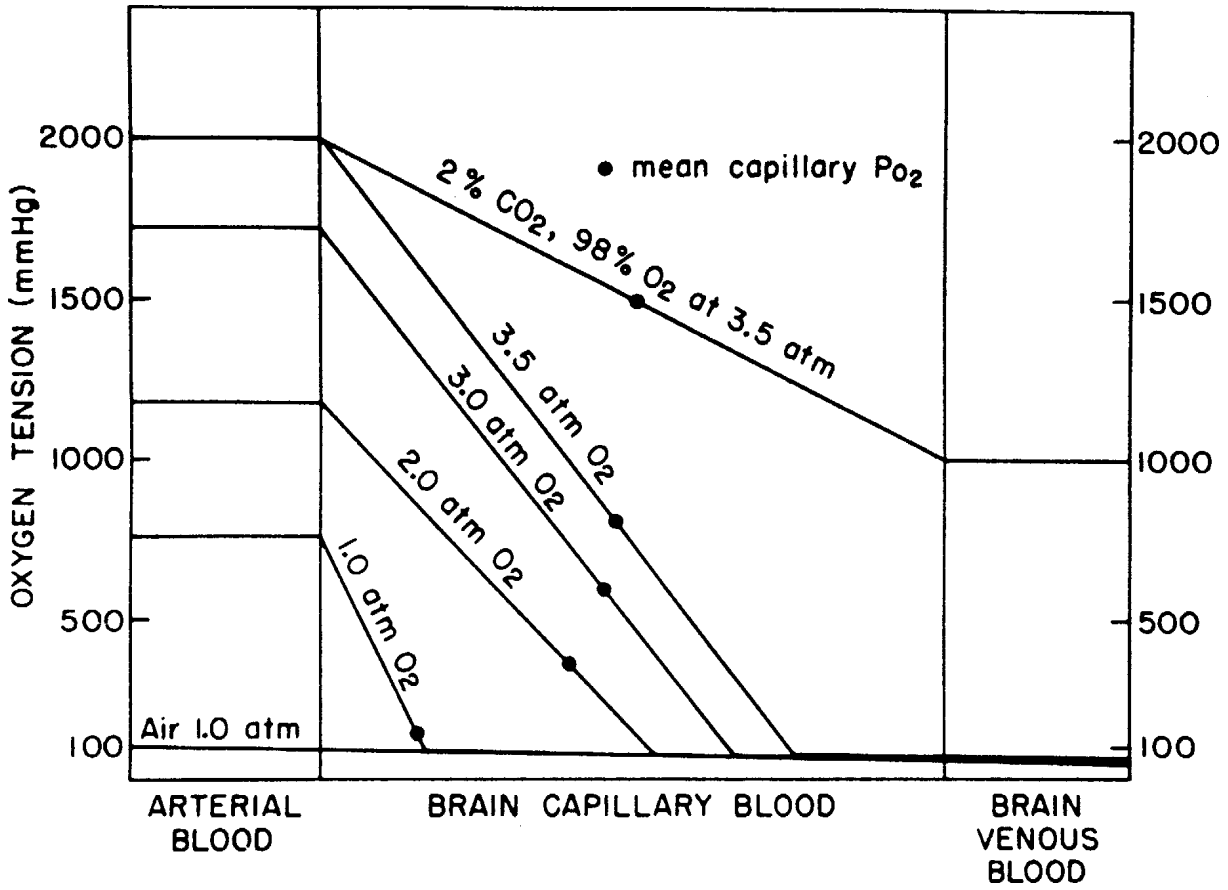

FIGURE 4 EFFECT OF INCREASED INSPIRED PO_2 ON OXYGEN TENSIONS OF ARTERIAL BRAIN CAPILLARY AND INTERNAL JUGULAR VENOUS BLOOD. (AVERAGE VALUES IN NORMAL MEN.)

The graph illustrates, for each of several levels of inspired PO_2, the manner in which oxygen tension across the mean brain capillary is increased by progressive increases in inspired PO_2. The patterns of change in brain capillary PO_2 are calculated from experimentally determined values of oxygen pressure in arterial and brain venous blood on the assumption of uniform O_2 loss. When arterial hypercapnia is induced by administering carbon dioxide with oxygen at 3.5 ATA, brain oxygenation is drastically increased. From Lambertsen.[28]

The rise in tissue carbon dioxide partial pressure induced by oxygen breathing (Fig. 6) is not known to potentiate local chemical oxygen poisoning. It may induce indirect effects through physiological actions within the central nervous system.

The effect of morphine in hastening onset of human central nervous system oxygen poisoning appears due to respiratory suppression with consequent elevation of arterial carbon dioxide pressure (and presumably increase in brain oxygen flow and pressure).

Alveolar/Arterial hypocapnia can transiently abolish pre-convulsive neuromuscular signs of human central nervous system oxygen poisoning.

There is yet no reason to expect that small degrees of alveolar hypercapnia modify local chemical oxygen effect in the lung, even though severely toxic degrees of inspired carbon dioxide can do so.

Exercise and immersion each hasten onset of central nervous system convulsions[6,7] for unknown reasons. There is yet no reason to assume an important influence of exercise or immersion on local chemical effect of oxygen in the lung.

Influences of tolerable degrees of hypothermia or hyperthermia on oxygen tolerance of human central nervous system or other organ functions have not been defined.

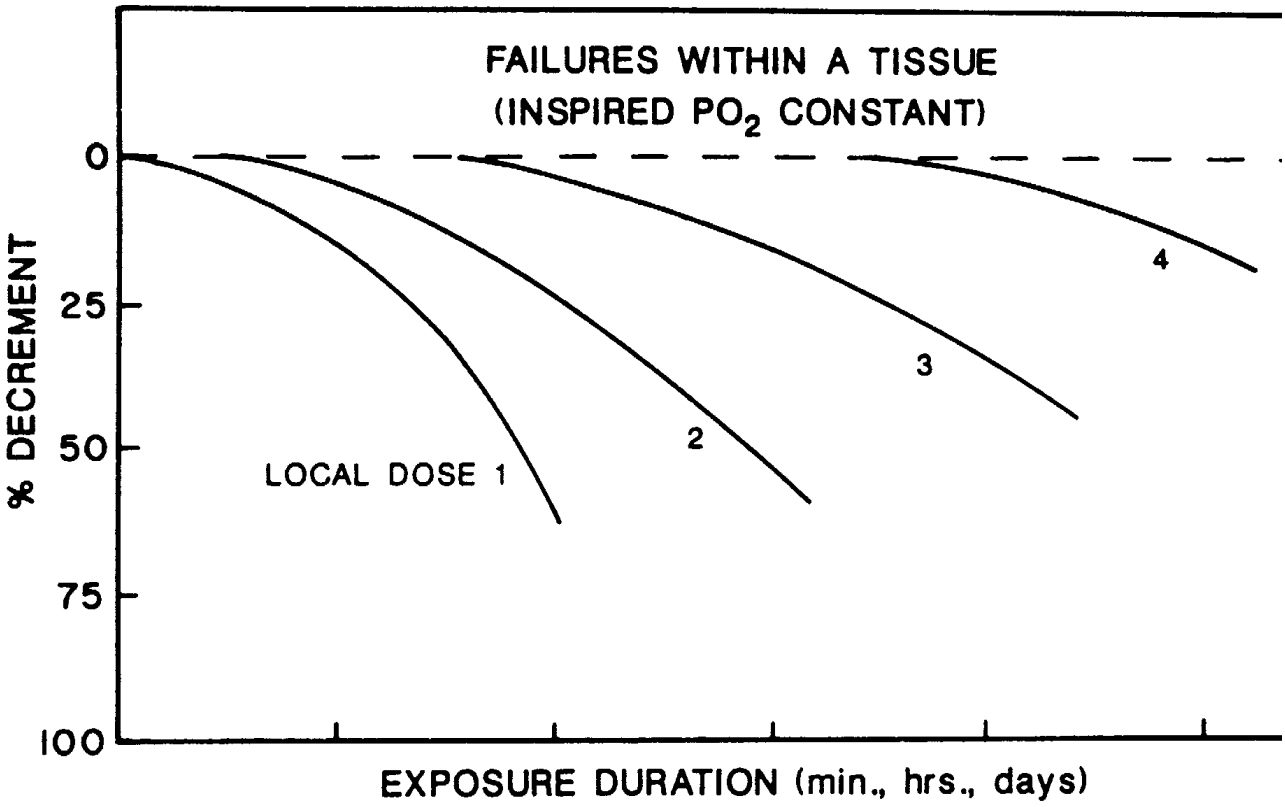

FIGURE 5 CONCEPT OF DIFFERENT RATES OF DEVELOPMENT OF A SINGLE FORM OF ADVERSE EFFECT IN DIFFERENT MICRO-ANATOMICAL LOCI OF A SINGLE TISSUE (E.G. BRAIN CORTEX).

At a constant level of respiratory hyperoxic exposure, large differences in local oxygen pressure should result in different time courses for development of oxygen effect.

Extension of tolerance to oxygen

The goals for practical extension of oxygen tolerance in undersea medicine and human activity are to delay and to limit the degrees of all forms of oxygen poisoning. These goals should be attainable. However, man will not change. Neither will oxygen or the activity of free radicals. The answers to extension of oxygen tolerance will come through the combination of improved fundamental information concerning mechanisms, targets, dose and effects, along with sensible use of harmless physiological measures.

The question of "latent period" for development of oxygen poisoning

Useful safe or "tolerable" periods of hyperoxic exposure are widely employed in operations and therapy. Does a true "latent period" precede the development of any toxic oxygen action? Most probably not, but if evidence indicates only small initial effect sensible practical use of the initial period can be made. "Toxic actions" of oxygen at any target site probably begin immediately, along with the harmless and self-limited physiological effects cited. Because small degrees of effect are "tolerable", the generic "Oxygen Tolerance Table or Curve" indicates a useful range of exposure (Fig. 7). Expressions of poisoning at two atmospheres or more of oxygen pressure appear early in the lung; they appear to be delayed in the visual system.[3] While oxygen poisoning is not degree limited, it does take time to generate detectable or practically important degrees of disruptions. Knowledge concerning the early time courses of the separate and simultaneous forms of poisoning is an essential element for concepts of useful oxygen tolerance and its successful extension.

Relation of degree of oxygen effect to reversibility

Overlapping sequential influences of oxygen poisoning occur, from the fully reversible physiological influences, to initial chemical effect, through reversible disruptions of cell function, to structural damages, to cell death. Overlapping forms of secondary restorative processes occur also, on ending hyperoxia, involving different rates of *reversibility* of specific chemical effect, *recovery* from reversible cell functional change, and *repair* of irreversible structural damage. Only early and rapidly reversible effects can be relevant to useful extension of oxygen tolerance.

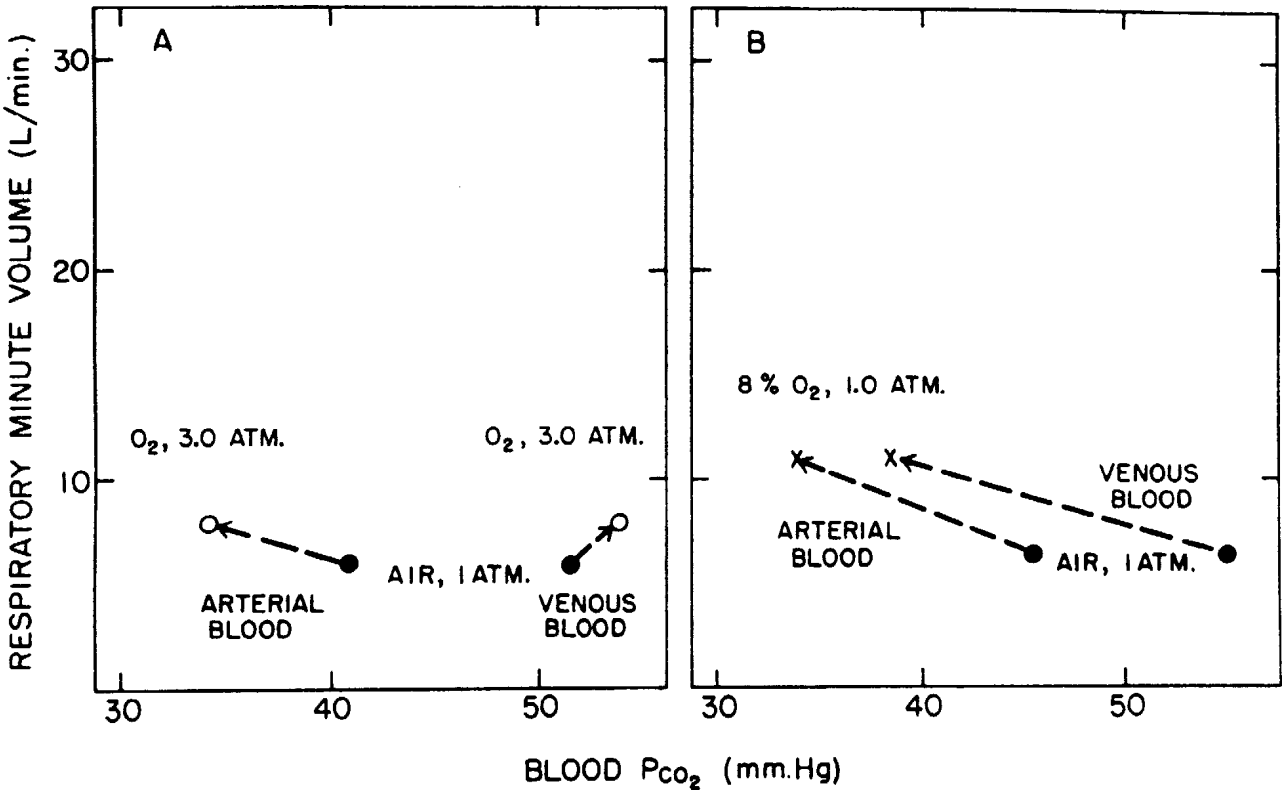

FIGURE 6 INFLUENCES OF O_2 AND HYPOXIA ON ARTERIAL AND CENTRAL PCO_2.

Control values relating respiration to arterial and internal jugular venous PO_2 in air breathing are compared with effects of high inspired PO_2, and with low inspired PO_2. High O_2 pressures (3.0 ATA) *raise central venous PCO_2 while inducing hyperpnea and thereby lowering arterial PCO_2*. Hypoxaemia leads to *simultaneous lowering* of arterial and central PCO_2. Exposure to hyperoxia is inevitably accompanied by exposure of brain and other tissues to effects of hypercapnia.[1]

Rapid reversibility will depend upon slowing or interrupting the toxic process at the early stages of chemical and functional effect, prior to cell structural damage and prior to the inflammatory response or cell replacement involved in repair (Fig. 8). This ideal may not be absolutely attainable, except by slowing the rate of formation of the oxygen free radical itself. Once formed the free radical disappears by doing harm, but the effect may sometimes be on a renewable chemical target such as a scavenging enzyme.[8-10] These events may be normal physiological components of the useful periods of oxygen breathing at various pressures.

Relevance of rates of reversal or recovery to tolerance extension

Reversal or *Recovery* requires time, after terminating a toxic oxygen exposure, and the factors determining rates of reversal or recovery have no necessary qualitative relation to those determining rates of onset. Certainly *recovery from severe degrees of oxygen poisoning is slower than from mild exposures*. The times required for *reversals* of different biochemical consequences of slight degrees of oxygen poisoning may conceivably be very brief, from fractions of a second, to minutes. The time for *recovery* from functional disruptions will be longer, and the time for *repair* of structural damage is too long for practical relevance in undersea operations or therapy.[2] True extension of tolerance to a severe chemical or structural effect of oxygen poisoning is unlikely and, considering the many still undefined functional effects, is not a primary goal for research in extending the usefulness of oxygen in therapeutic or operational activities.

Even severe degrees of effect may be "silent" in some tissues, going unrecognised in research or therapy. Some effects may be serious, but others are unimportant because the tissue is not functionally critical. This means that, for useful extensions of oxygen tolerance, it is necessary not only to investigate what happens, and *how much*, but *how fast does each specific early, detectable effect develop*, and *how fast does it resolve* from an early stage. Study of severe damages, survival times, or post-mortem pathology, with thousands of simultaneous interacting damaging effects, may continue to provide important qualitative guidelines.

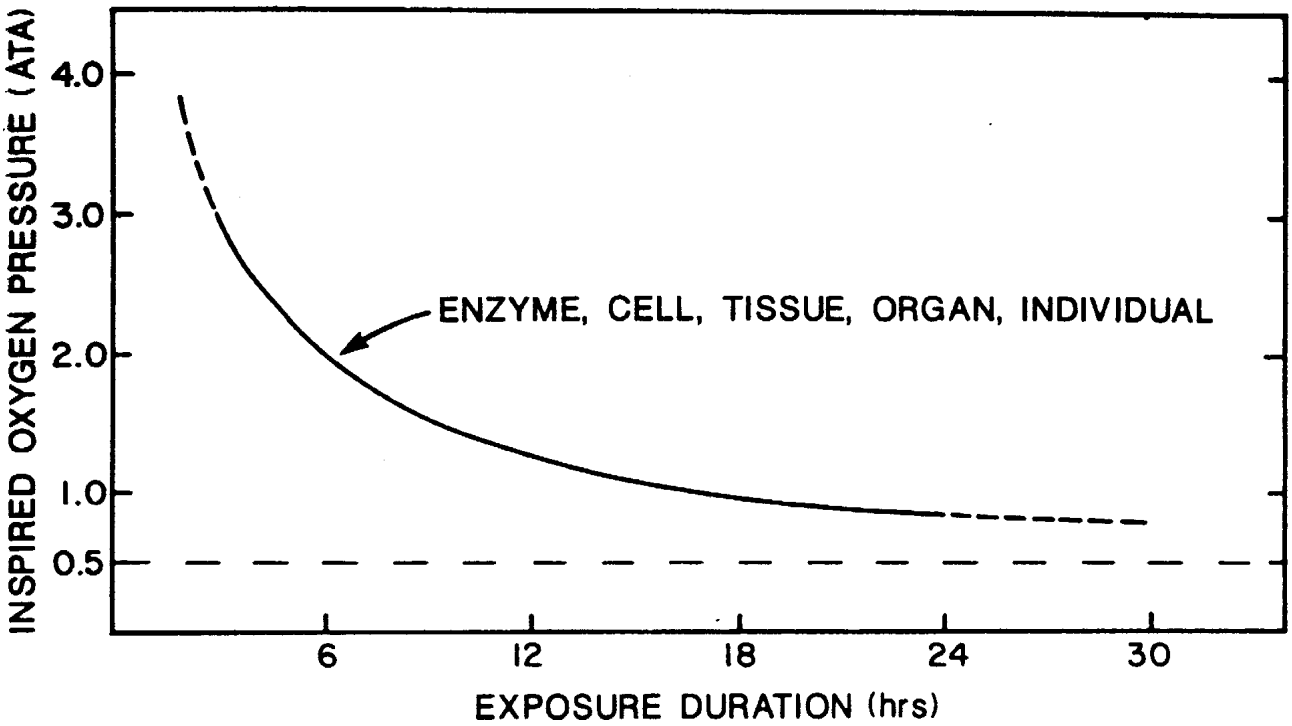

FIGURE 7 THE GENERIC OXYGEN TOLERANCE CURVE

The diagram conceptually illustrates the continuous exposure duration required at different oxygen pressures required to produce a detectable or uniform degree of cumulative oxygen poisoning. It is conceived that at infinitely high oxygen pressures, severe poisoning occurs instantly, and that at a sufficiently low degree of hyperoxia development of detectable effect will require extreme duration, whether for enzyme, cell or organ.[27]

However, these measures are not as useful as indices of success in oxygen tolerance extension as are measurements of early onset and recovery of functional disruptions of enzyme, membrane, cell, or organ systems, in human beings and other species.

Tolerance extension by "adaptation"

At low levels of atmospheric hyperoxia, some forms of true protective adaptation appear to occur, such as those related to changing antioxidant defenses observed in some tissues or cells.[11-12] Some may even prove useful during continuous low level hyperoxia, less than one ATA. At higher oxygen pressures such a process of adaptation could conceivably occur in some cells of the intact human being, concurrently with progressive and severe poisonings in some other cells.[2] At very high useful oxygen pressures rapid rates of development of poisoning damage would make any chemical "adaptation" too little and too late, as well as not universal among different types of affected cells. Processes to complement physiological adaptation are therefore required if truly major extensions of tolerance are to be accomplished.

Tolerance extension by "drugs"

Extension of oxygen tolerance by use of drugs should certainly continue to be investigated as part of an overall improvement in understanding of basic reactions. However, the pharmacologic approach, such as providing ancillary antioxidant or free-radical scavenging mechanisms, will attain truly broad usefulness only where a drug can approach the free permeability of the oxygen molecule, reaching the many right locations, in the right concentrations, at the right times, and remain effective there in the face of continuous hyperoxia, without itself inducing multiple toxic effects.[1] This picture is intentionally pessimistic at this time since most synthetic or natural drugs will face the serious handicap of membrane and other diffusion barriers, not faced by oxygen.

Tolerance extension by interrupted exposure to oxygen

This existing physiological approach, now accepted but still only superficially explored, represents a rational focus upon oxygen tolerance extension at all useful oxygen pressures. Interruption of oxygen exposures at one ATA by

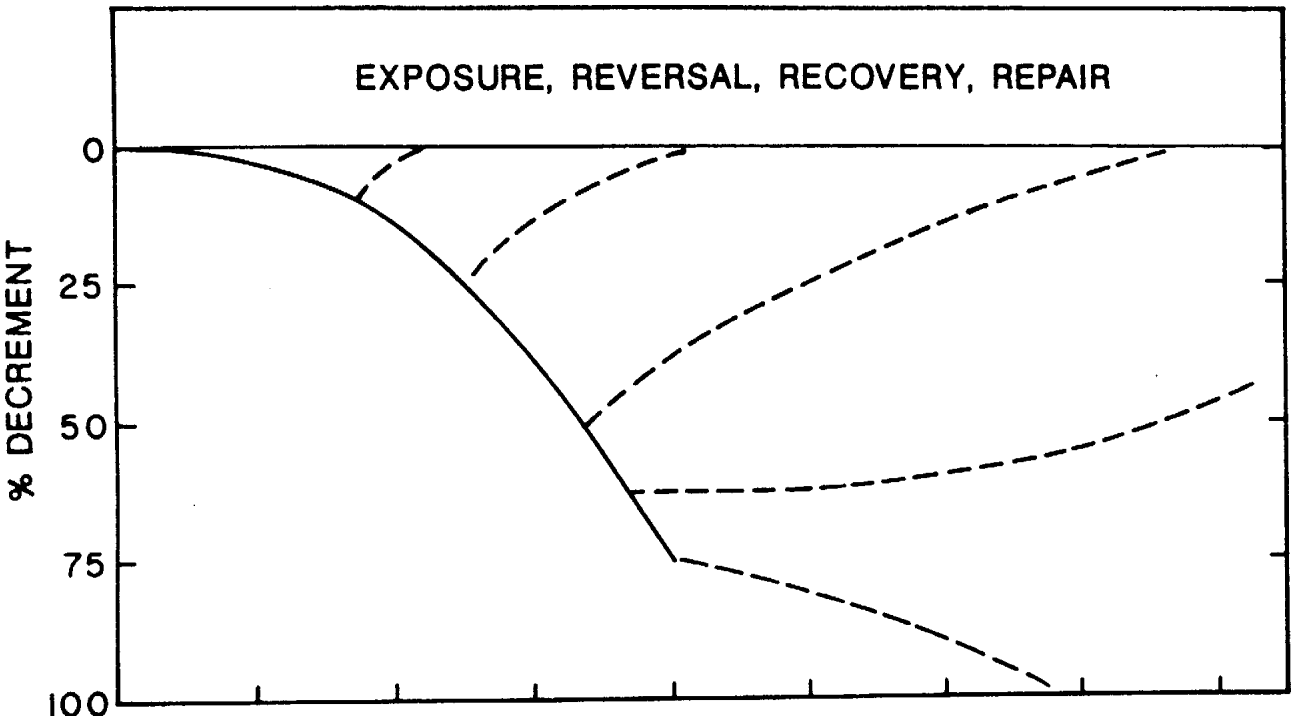

FIGURE 8 CONCEPTUAL RELATIONSHIPS OF EXPOSURE DURATION AND RECOVERY TIME FOR CONTINUOUS OXYGEN BREATHING

Solid curve indicates development of an adverse effect of oxygen during a continuous exposure. Dashed lines indicate various post-exposure "recovery" patterns. Recovery from minor effect can be prompt and complete. Recovery after severe oxygen poisoning, as in extensive hyperbaric therapy, can be prolonged. Extreme exposure can result in failure of recovery.

periods of air breathing was found fifty years ago to delay death in dying animals.[3,4] However, in 24-hour pulmonary oxygen tolerance studies at 1 ATA in man, brief interruptions of oxygen showed no detectable advantage in lessening pulmonary symptoms, or preventing a decrease in vital capacity,[13] and investigation was not followed up in one atmosphere oxygen therapy.

At pressures greater than one ATA it was learned in the earliest stages of the evolution of open-sea oxygen diving that the onset of definite signs of central nervous system oxygen poisoning, such as diaphragmatic and facial twitching, could be immediately abolished (within a minute) by ascending to shallower water (lowering inspired PO_2 without stopping pure oxygen breathing).[14] This allowed further time to be spent again in deeper water. This observation clearly meant that some toxic effects of oxygen had intrinsic rapid reversal mechanisms. Encountered repeatedly in undersea operational activity, this observation generated the simple concept of oxygen tolerance extension by optimised interruption of hyperoxic exposure.[14,15] There are now better ways than open sea diving to explore this critically important phenomenon, and the procedure of repeated interruption of oxygen exposure for oxygen tolerance extension at pressures greater than one atmosphere has now been investigated in successive studies in insects, small mammals, and in man.[16-23]

Factors of duration and pressure in tolerance extension by intermittent exposure

Clearly factors of oxygen pressure, oxygen exposure duration and duration of interruption are all important in efforts to optimise tolerance extension by intermittent exposure. This has led to the terms "effective" and "efficient" patterns of intermittency.[17] The "most efficient", achieved by optimisation of relationships between oxygen exposures and interruptions of exposures, provides the greatest tolerable oxygen exposure duration per day or week (or other practical unit of time). The "most effective" patterns prevent oxygen poisoning even in exposures of very long cumulative duration.

It was recognised from the beginning that success in extending oxygen tolerance by interrupting early stages of hyperbaric oxygen poisoning would depend upon the relative rates of onset and rates of recovery of discrete chemical reactions involved in the phenomena of oxygen poisoning. It was also considered that such a direct procedure could

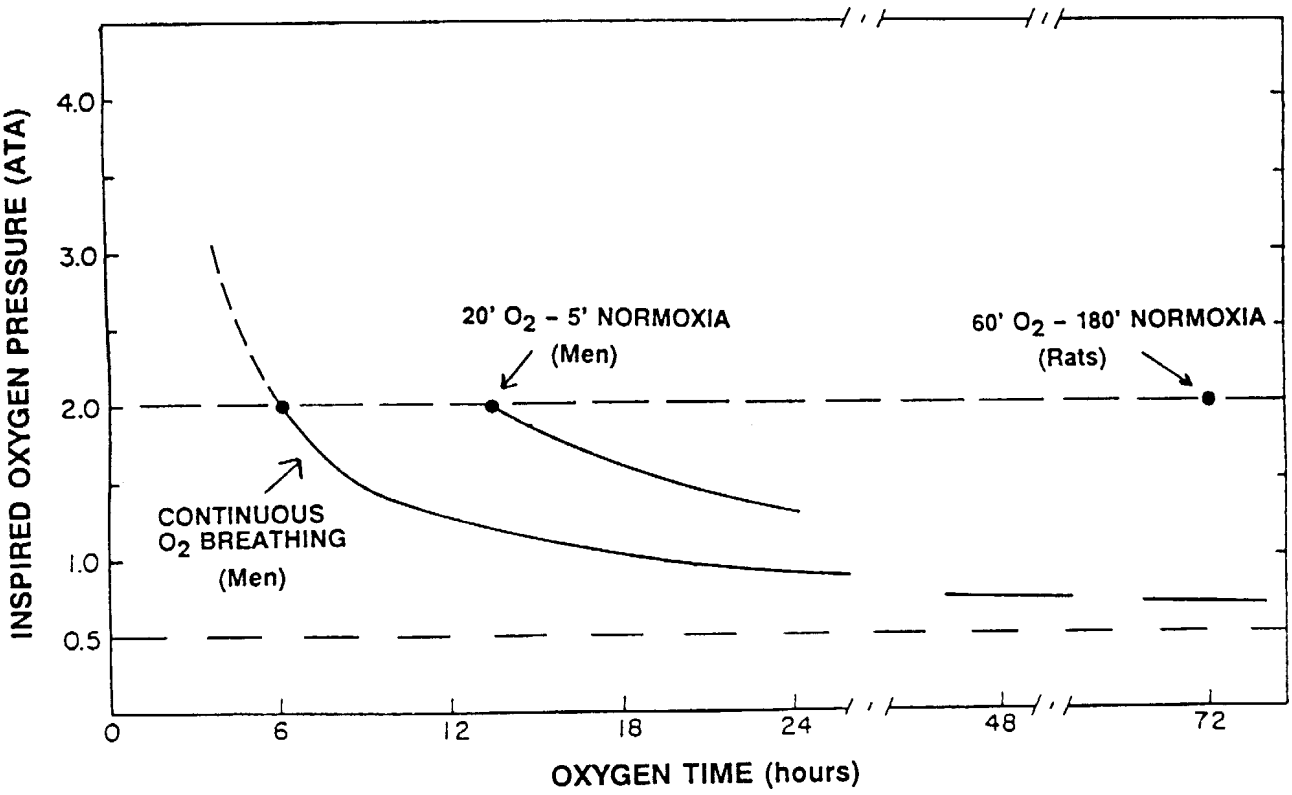

FIGURE 9 EXAMPLES OF OXYGEN TOLERANCE DURING INTERMITTENT OXYGEN BREATHING IN MAN AND ANIMALS

Points for man indicate 5% decrement in vital capacity.[18,27] Potential for practical extension of human oxygen tolerance at 2.0 ATA may lie between the extreme examples of interruption patterns shown: over 12 hours in man[18] and over 10 days in rat.[26] At lower oxygen pressures larger gains should be attainable. Figure modified from Lambertsen.[2]

simultaneously reach and influence reactions in all cells in all organs, since it involved merely the normal delivery of oxygen and its removal by the metabolism of oxygen itself.[1,2] Such considerations have led to wide use but to only very limited experiment.

Early investigations in animals[16,17,21,24] showed that an efficient extension of oxygen tolerance could be accomplished by 10-minute interruptions following 30-minute oxygen breathing periods at 3.0 ATA, or even by 5-minute interruptions between 20-minute oxygen exposures. This pattern was promptly and lastingly applied to the still current hyperoxic therapies for acute gas lesion diseases (e.g. decompression sickness and gas embolism).[25] It has been investigated in human subjects in relation to pulmonary oxygen tolerance at 2.0 ATA. However, essentially no information even now exists concerning other organs. In addition, the information required for optimisation of oxygen tolerance extension in humans at one ATA or at any other useful oxygen pressure is not available. Indications exist that there is large opportunity for multiday oxygen tolerance extension at 2.0 ATA (more than ten days) without evident pulmonary or neurologic oxygen poisoning (Fig. 9), even though cumulative effects in animals are still produced at 4.0 ATA .[26] The large gains at the lower oxygen pressure may in fact combine prevention and adaption in achieving the grossly extended oxygen tolerance. Since demonstration of such gains at oxygen pressures of one ATA or less will be extremely difficult, exploration of oxygen tolerance extension at higher pressures will continue to be desirable, even where practical operational or therapeutic interest is more concerned with lower degrees of hyperoxia.

The philosophy of extremes for physiological extension of oxygen tolerance

A nearly self-evident philosophy for extending useful oxygen tolerance at any partial pressure can aid planning in future basic and applied investigation. Elements of this philosophy include:

Patterns of oxygen effect within chemical, tissue and organ systems, organ systems will inevitably be different at each oxygen pressure.

Optimal patterns of duration of oxygen exposure and interruption for tolerance extension will be

different for each oxygen exposure pressure.

An infinitely short exposure to any useful degree of hyperoxia will produce no residual harm, and require no recovery period.

An infinitely prolonged interruption of non-destructive hyperoxic exposure will allow complete recovery.

Optimal extension of tolerance for clinical or operational use in human beings will involve the combination of the shortest practical oxygen exposure with the longest practical interruption.

Optimisation for rational use of oxygen in various hypoxic states requires equivalent understanding of tolerance to brief hyperoxia, tolerance to brief hypoxia, and the interactions of tolerance to hypoxia and hyperoxia.

Summary - The significance of the opportunity for oxygen tolerance extension

The potential gains through research in oxygen tolerance and its extension, at low and at high levels of hyperoxia, are among the largest in the environmental and biomedical sciences, and in undersea operations. Significance of such gains extends beyond avoidance of lung damage, red-cell haemolysis, and central nervous system convulsion, to multiple other tissues, some not even now examined.

The long deferral of direct investigative attack in human beings upon this large opportunity has here been precautionary, due to the awareness that to extend human oxygen tolerance safely it is necessary first to investigate oxygen tolerance systematically.[2] It is especially important to learn the early influences of oxygen poisoning upon normal functional activity of critical chemical reactions, cells and processes, from pulmonary and glomerular capillary endothelium to cognition and vision. Optimisation studies of intermittent oxygen exposures are also required, using cells and small animals as guides to the large investment involved in attempting oxygen tolerance extension in humans. Validation of success in gross extension of human oxygen tolerance may require precise measurement in man over the extreme duration of multi-week interrupted exposures. Without prior broad examination of continuous exposure oxygen at multiple pressures in multiple organs, investigations of intermittent exposure may encounter unexpected or previously unrecognised consequences of cumulative oxygen effect.

The *"cautionary philosophy"* of long term investigation in any form of oxygen tolerance extension must therefore be that, in proceeding to extend tolerance of particular cells or functions of special interest, we must not ignorantly damage important cells or functions not as simply measured.

The continuing *"positive philosophy"* is that hyperoxia will always be useful as well as toxic, and can become greatly more useful in spite of its persistent inherent toxicity. Oxygen should be employed fully, and not feared where the necessary information is obtained, degree of adverse effect is controllable, and full recovery is to be expected.

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DIVING WITH SAFETY

We reprint below a pamphlet, with the above title, produced by the Division of Accident Prevention of the Queensland Department of Employment, Vocational Education, Training and Industrial Relations. The regulations described came into force in October 1989 and have aroused considerable opposition among some members of the diving industry. The pamphlet has been reprinted to allow all members to form their opinions on the legislation and their attitudes to such rules and regulations. The grapevine has it that other States are considering similar regulations under their Industrial Health and Safety legislation. It seems to the Editor that the main impact on the recreational diver will be showing his or her certification card when getting a refill or boarding a charter vessel, being restricted to 4 dives a day when on a charter vessel and having to sign a record of dive (presumably to show that they have returned

from the dive) when on a charter boat. It is hoped that members will write to the Editor giving their views of the legislation.

With the introduction of the Workplace Health and Safety Act and Regulations on 31 July, 1989, the face of underwater diving in Queensland began to change. The Act introduced direct government regulation of diving practices in the fee-paying recreational and occupational diving areas, and provision for heavy penalties should defined standards be breached.

Diving regulations under the Workplace Health and Safety Act came into full effect on 30 October, 1989. These regulations impact directly upon all involved in underwater diving, with the exception of private sport divers making use of personal equipment.

Queensland is the first state in Australia to legislate diving safety practices for the recreational industry. The legislation has been developed in close consultation with industry bodies to ensure that it meets the current and future needs of Queensland's diving industry.

Duties of care

The primary thrust of the Workplace Health and Safety Act lies in the concept of "Duties of Care". Simply stated, these place legal responsibility for workplace health and safety upon all who come into contact with that workplace.

In terms of the diving industry, this includes owners and employees of dive shops, scuba instructors, divers (recreational and occupational), resort owners, employers of occupational divers, owners and operators of dive charter vessels, dive supervisors and those involved in the import, manufacture and maintenance of diving equipment.

Standards and codes of practice

Regulations concerning the diving industry are outlined on pages 96-100 (Regulations 259-264) of the Workplace Health and Safety Regulations.

These Regulations outline relevant industry standards adopted under the legislation and set the working boundaries for future operations within the Queensland diving industry.

Australian standards adopted for recreational and occupational diving under the new legislation include:

AS 2299
Underwater Air Breathing Operations
(with modification to provision 2.3 "Divers")

AS 2815
Training and Certification of Divers

AS 3519
Training and Certification of Recreational Divers

Students

Prior to commencing a course of instruction, students for open water certificates must now be certified medically fit by a qualified doctor with knowledge of diving medicine. For resort course participation, they must complete a medical questionnaire and be assessed as fit for diving by the scuba instructor.

Open-water students are limited to two (2) training dives in any 24 hour period.

Certified divers

When hiring equipment or refilling cylinders, divers are required to present a diving certificate issued by a recognised scuba training organisation such as PADI (Professional Association of Diving Instructors), NAUI (National Association of Underwater Instructors) or FAUI (Federation of Australian Underwater Instructors). Under no circumstances should equipment be issued without such certification.

Scuba instructors (employed)

Instructors must hold current medical and scuba instructor's certificates, the latter complying with standards established by the Recreational Scuba Training Council (USA) or the Australian Coaching Council Incorporated.

Instructors have a legal duty to ensure that students for resort course and open water instruction have fulfilled the relevant medical requirements prior to acceptance into training programs.

Instructors are further required to ensure that any vessel used for diving instruction holds a current certificate of survey.

Dive shops and self employed instructors

Owners of dive shops or self employed instructors have a duty to:

Ensure that all life support equipment is assembled and checked by the holder of an appropriate current service certificate.

Refuse to hire equipment or provide air fills to any person unable to present a current diver's certificate issued by a recognised scuba training organisation.

Ensure that where diving equipment is hired to a person for instructional use it consists of:

(a) a regulator fitted with an alternate air source
(b) a submersible pressure gauge
(c) a depth gauge and timer
(d) a buoyancy control device
(e) a power inflator device
(f) a submersible air cylinder and harness
(g) a mask, snorkel and fins.

Ensure that a service record is maintained on diving equipment.

Ensure that air used in underwater diving operations is provided clean and free of contaminants in accordance with AS 2299 (subject to modifications outlined in Regulation 262(e)).

Dive charter vessels

A dive charter vessel must be issued with a current certificate of survey by a lawful State or Commonwealth survey authority. It must also be equipped capable of providing oxygen therapy which renders the complete oxygen requirement to the lungs for the duration of stay at a dive site and until medical support becomes available.

A powerful boat must be maintained in the water for pick-up and rescue operations where the primary vessel cannot be readily used, and a communication system maintained for contacting the Diver Emergency Service (DES), should the need arise.

The person in command of a dive charter vessel (other than the pilot) is required to ensure that:

A dive-supervisor is nominated to oversee all diving activity, with one supervisor present for every 14 divers.

A lookout is posted when diving is performed.

A trained operator of oxygen equipment is on board.

A register of diving is maintained and signed by each diver upon completion of the dive and prior to weighing anchor.

Dive-supervisors

Dive-supervisors must hold a current certificate in dive leadership from a recognised training organisation. They have a duty to ensure that all divers hold a current certificate and perform dives only to their level of experience and qualification.

Divers must be informed of the dive destination, level of expertise required and duties of the dive-supervisor.

All dives must be planned as non-decompression dives and conducted in accordance with specified dive tables. Divers are required to keep a record of each dive, to be signed by the dive-supervisor.

Dive-supervisors must ensure that no more than four (4) dives are performed by a diver in any 24 hour period. They are also required to ensure that a DES instruction notice is easily accessible and understood by all at the dive site.

Occupational divers

Occupational divers are those persons engaging in commercial diving, scientific research or search and rescue operations. The Workplace Health and Safety legislation calls up AS 2299 to be applied to all occupational diving activities in Queensland.

Accident reporting

Every serious bodily injury, illness or dangerous occurrence must be reported within 24 hours to the Director, Division of Accident Prevention in the prescribed form.

The term "serious bodily injury" refers to any injury which results in admittance to hospital as an in-patient.

Inspections

A full-time diving inspector is employed by the Queensland Department of Employment, Vocational Education, Training and Industrial Relations to provide advice on general diving matters and assist in clarification of diving responsibilities under the Workplace Health and Safety Act and Regulations.

The inspector has the power to issue improvement, prohibition or seizure notices. When serious breaches of safety have occurred, prosecution action will be taken. Infringement notices (on-the-spot fines) may apply in less serious situations and where no injury has occurred.

Penalties

The penalty for breach of the Act causing serious

bodily injury or death is a fine of up to $120,000 for a body corporate, or $30,000 and/or six months imprisonment for an individual.

Penalties only apply where duties of care are abused and health and safety or human life is put at risk.

Purchase of legislation

Copies of the Workplace Health and Safety Act and Regulations are available from Goprint in Brisbane at the following addresses:

371 Vulture Street
WOOLLOONGABBA
QLD. 4102
Telephone: (07) 896 3360

135 George Street
BRISBANE
QLD. 4000
Telephone: (07) 224 4146

In other areas of the state, copies are available from local offices of the Department of Employment, Training and Industrial Relations, Division of Accident Prevention.

Further information

The Diving Inspector
Division of Accident Prevention
State Law Building
Cnr Ann & George Streets
BRISBANE, QLD. 4000

GPO Box 69
BRISBANE, QLD. 4001
Telephone: (07) 227 4895

The above has been reprinted from DIVING WITH SAFETY, a pamphlet produced by the Division of Accident Prevention of the Queensland Department of Employment, Vocational Education, Training and Industrial Relations.

GLEANINGS FROM MEDICAL JOURNALS

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

A TENTATIVE GUIDE TO THE MANAGEMENT OF MARINE STINGS

Struan K. Sutherland
Commonwealth Serum Laboratories, Melbourne

The following brief notes have proved of some use to medical and paramedical groups and are modified from time to time.

Fish and stingray stings

Pain relief

Pain relief is obtained rapidly by bathing the injured region in warm, but not scalding, water. If necessary, boat-engine cooling-water can be used. Often the pain returns quite dramatically when the heat therapy is ceased.

Local anaesthetic agents sometimes are indicated and, in severe cases, a regional nerve block by means of bupivacaine or lignocaine may be necessary.

Systemic opiate therapy may be required.

Antivenom should be administered with appropriate precautions in significant stonefish stings.

Care of the injured area

Take positive action and remove foreign bodies or dead tissue. Local tissue necrosis is usual with envenomation by stingray spines. Ensure good drain. X-ray examination may be necessary.

Wash well with *fresh* water as sea-water may encourage bacterial growth. The wound potentially is infected so remember that marine bacteria represent a wide range of organisms, many of which are not characterised fully. Most are resistant to common antibiotic agents, and also require special, salinated media for culture. Expert opinion is that trimethoprim-sulphamethazole is the drug of first choice.

General effects

Maintain vital functions (ABC - airway, breathing and circulation).

Shock - note pain relief as above.

Effects of venom - antivenom if indicated

Apply pressure - immobilisation type of first aid.

With the exception of bites by blue-ringed octopuses, conus stings and sea-snake bites, the pressure-immobilisation technique should *not* be used to attempt to hold the toxins at the site of the bite or sting. To do so may increase pain and local tissue damage.

Additional note

Tetanus prophylaxis should be updated as required. Death from tetanus has occurred, especially after stingray injuries.

It may be necessary to rest the injured region for days for satisfactory healing to occur.

Jellyfish stings

Prompt application of domestic vinegar to the affected areas appears to be the simplest and most rational first-aid measure for most jellyfish stings. Methylated spirits should *not* be used.

Vinegar is not recommended for "bluebottle" *(Physalia physalis)* stings and these best are treated with ice-packs.

Box-jellyfish stings often are very serious and the patients may require maintenance of their vital functions and pain relief as well as rapid antivenom therapy.

Acknowledgements

I am grateful to Dr John Williamson for his comments.

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This advice appeared in The Medical Journal of Australia, 1989; 151 (Dec 4-18): 626.

STING ABSTRACTS

Successful use of *Chironex* antivenom by members of the Queensland Ambulance Transport Brigade

Fenner Peter J[1], Williamson John A[2] and Blenkin John A.[3]
Med J Aust 1989; 151: 708-710

ABSTRACT We report the first administration in Australia by ambulance officers of the Commonwealth Serum Laboratories' *Chironex fleckeri* antivenom, in two separate, major, potentially life-threatening *Chironex* envenomations. In both cases, the antivenom was effective in helping to relieve signs and symptoms (including pain). This antivenom still is the most specific part of the treatment of a major *Chironex fleckeri* sting after stabilization of the airway, breathing and circulation. The additional use of verapamil may contribute to the prevention of some of the cardiac complications that result from the direct effects of the *Chironex* venom.

From:
1 Ambrose Medical Group, North Mackay, Qld. 4740.
2 Townsville General Hospital, Townsville, Qld. 4810.
3 Yeppoon Public Hospital, Yeppoon, Qld. 4703.
Correspondence: Dr P.J. Fenner.

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Fatal and non-fatal stingray envenomation

Fenner Peter J[1], Williamson John A[2] and Skinner Ralph A.[3]
Med J Aust 1989; 151: 621-625

ABSTRACT A fatality occurred in a previously healthy 12-year old-boy after a penetrating chest injury from a stingray barb. The injury occurred under freak circumstances. Death was a result of cardiac tamponade which was secondary to venom-induced, localized myocardial necrosis and spontaneous perforation, six days after the direct penetration of the right ventricle by the barb. Three other cases of less serious stingray envenomation are described which illustrate the significant localized morbidity that may occur without immediate wound exploration and toilet after adequate anaesthesia. We also report a study of a series of 100 minor stingray envenomations which, when treated, resulted in no morbidity. It is possible that local infiltration with 1% plain lignocaine may have a direct counteraction against stingray venom that remains in the wound area. Stingray venom has insidious, but powerful, localized tissue necrosing properties in humans.

From:
1 Ambrose Medical Group, PO Box 34, North Mackay, Qld. 4740.
2 Department of Anaesthesia, Intensive Care and Marine Medicine, Townsville General Hospital, Townsville, Qld. 4810.
3 Tangalooma Resort, Moreton Island, Qld. 4004.
Correspondence: Dr P.J. Fenner.

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Cold packs: effective topical analgesia in the treatment of painful stings by *Physalia* and other jellyfish

Exton David R[1], Fenner Peter J[2] and Williamson John A.[3]
Med J Aust 1989; 151: 625-626

ABSTRACT A study has shown that, when applied to *Physalia* ("bluebottle") jellyfish stings, cold packs are as effective as topical analgesia in the relief of mild-to-moderate skin pain. The application of ice also has been shown to be effective for topical analgesia in a number of other jellyfish stings, including by *Cyanea* ("hair jellyfish"), *Tamoya* sp. ("Moreton Bay stinger" or "fire jelly") and *Carybdea rastoni* ("jimble") as well as by *Physalia*. In the current state of knowledge, cold packs or ice are recommended as the first-aid treatment for jellyfish stings with local skin pain.

From:
1 Surf Life-Saving Association of Australia, Queensland State Centre, PO Box 36, Newstead, Qld. 4006.
2 and 3 as for 1 and 2 above
Correspondence: Mr D.R. Exton.

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RECOMMENDED TREATMENT OF STINGRAY INJURY

First aid

If medical aid is not available immediately, place the affected area in water as hot as can be tolerated without burning (test first !). Seek medical aid.

If heat is unavailable or fails to provide relief, then ice or ice-packs are applied to the area for up to 30 minutes and, if they help, are reapplied.

Medical management

If necessary, inject local anaesthetic agent (1% plain lignocaine) directly into the wound and around the site of envenomation to ease the pain.

Perform x-ray examination of the wound area for traces of foreign material.

Explore the wound carefully, following the full length of any tract which may be left by the penetrating barb. Remove all traces of broken barb or integumentary sheath and clean the wound with hexachlorophene in 70% alcohol; if available, use an operating microscope.

Leave the area open to granulate.

Administer tetanus vaccine or adult diptheria and tetanus toxoids, if indicated.

Antibiotic therapy should be considered if the injury is more than six hours old and/or is extensive.

Patients with wounds that are considered to penetrate to the abdomen or chest should be referred immediately to experienced medical centres for further assessment.

Follow-up of all wounds is necessary until they are healed fully.

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This advice appeared in The Medical Journal of Australia, 1989; 151 (Dec 4-18): 623.

ASTHMA AND DIVING

Diving practices of scuba divers with asthma
Farrell PJS and Glanvill P
Br Med J. 1990; 300: 166

Summary by *SPUMS J.*

A questionnaire, circulated in *Diver* magazine, produced 104 replies from asthmatic divers. 16 divers thought that it was safe to dive within 2 hours of wheezing, 27 thought that it was safe after 12 to 48 hours, 7 chose periods between 1 week and 1 month. Over 50% of the respondents (54) answered they did not know. 20% of these had dived within 12 hours of wheezing. The 104 divers had completed 12,000 dives between then without producing a pneumothorax or cerebral arterial gas embolism.

Scuba divers with asthma
Martindale JJ
Br Med J. 1990; vol. 300: 609
Letter

Scuba divers with asthma
Watt Stephen J and Gunnyeon William J
Br Med J. 1990; vol. 300: 609
Letter

Scuba divers with asthma
Glanvill Peter and Farrell Patrick
Br Med J. 1990; vol. 300: 609-610
Letter of reply

NARCOSIS

Memory deficit caused by compressed air equivalent to 36 metres of seawater.
Philp RB, Fields GN, Robert WA.
J Appl Psychol 1989; 74(3): 443-46

Twenty four students from a diving school undertook a hyperbaric chamber dive to a pressure equal to 36 m of seawater. Tests of cognitive function and manual dexterity, performed in the chamber during the 35 minute bottom time and before, or after, the dive included immediate and delayed free recall of words presented as 7 lists of 15 each, recognition of previously presented words, number identification , and a forceps pickup of ball bearings. Delayed free recall and immediate free recall (primacy region) were significantly impaired, whereas manual dexterity and recognition memory were not. These are in keeping with previously reported findings but indicate that significant impairment of memory may occur in experienced divers at operation depths for air diving. Lack of effect on recognition memory suggests that cuing strategies might be useful for debriefing divers.

BUBBLES IN THE LUNG

Fate of air emboli in the pulmonary circulation
Presson RG Jr, Kirk KR, Haselby KA, Linehan JH, Zaleski S and Wagner WW Jr.
J Appl Physiol. 1989; 67(5): 1898-1902

The lung serves an important nonrespiratory function by trapping and excreting venous air emboli. The site of trapping and the mechanism of excretion, however, are uncertain. To observe the behavior of bubbles in the pulmo-

nary circulation, we injected venous air emboli into anesthetized dogs and videotaped their elimination from the pulmonary microcirculation by using in vivo microscopy. Small intravenous bubbles lodged exclusively in pulmonary arterioles and were eliminated from that site. To determine whether the gas was dissolving into nearby blood and then was carried to the capillaries for excretion, the rate of bubble radius change was measured during nonperfused conditions produced by balloon occlusion of lobar blood flow and compared with perfused conditions. Bubble volume decreased at the same rate during perfused and nonperfused conditions and thus was independent of regional blood flow. Molecular diffusion of gas directly across the arteriolar wall into alveolar spaces was the most likely mechanism of elimination because calculations based on the Fick equation for molecular diffusion predict an elimination rate nearly identical with those observed experimentally.

TREATMENT OF PNEUMOTHORAX AT DEPTH

Inherent unsaturation in the treatment of pneumothorax at depth

Daugherty C G
Undersea Biomed Res. 1990; 17(2): 171-177

In discussing the treatment of pneumothorax under pressure, standard diving medicine publications recommend only the insertion of a chest tube before decompression. However, there are marked difficulties in performing surgery in a typical offshore commercial chamber, and present knowledge of chamber bacteriology indicates there is important risk of serious infection. By contrast, the venerable principle of inherent unsaturation and known experimental data permit the rational and safe use of pressure and oxygen breathing to resolve this problem. This should be the treatment of first preference for, even if it is not effective, no other options are closed off. Two representative cases are presented.

BUBBLE FORMATION

Bubble formation properties of hydrophobic particles in water and cells of *Tetrahymena*.

Hemmingsen EA and Hemmingsen BB.
Undersea Biomed Res 1990; 17(1): 67-78.

It is generally assumed that hydrophobic surfaces play a role in bubble formation in vivo, but no tests of this assumption seem to exist. Model systems for both in vitro and in vivo study of the bubble nucleation properties of hydrophobic surfaces were developed. First, aqueous suspensions of particles were exposed to gas supersaturation, and the numbers of bubbles that formed were determined. Although the supersaturation thresholds for spontaneous bubble nucleation in pure water exceeds 175 atmospheres gas tension, gas tensions of only a few atmospheres caused the profuse formation of bubbles with the most effective particles. Some or most of this latter effect seemed to be caused by gas trapped in irregularities on the particles. Second, particles that were especially effective bubble promoters were added to suspensions of ciliates. Upon their ingestion, all of the particles lost their ability to induce bubble formation in the cells with supersaturations equal to or exceeding the threshold for spontaneous nucleation in water. These results indicate that intracellular bubble formation may not occur readily in vivo.

From

Physiological Research Laboratory, University of California, San Diego, La Jolla, California 920093, and Department of Biology, San Diego State University, San Diego, California 92182-0057

IMMERSION

Tissue heat transfer in water: lessons for the Korean divers.

Rennie DW.
Med Sci Sport Exercise 1988; 20: S177-184

The factors which influence tissue heat transfer and temperature gradients from body core to skin surface are reviewed in the context of studies on Korean diving women. The resistance to heat transfer imposed by resting muscle is shown to be 2-3 times as great as that imposed by overlying fat and skin. However, exercising muscle imposes very little resistance to heat flux because of the increase in convective heat transfer. Accordingly, the limiting resistance to heat flow is shifted to subcutaneous fat and skin during exercise in cold water. Hypothetical examples are given of how important the subcutaneous fat can be in maintaining a high core-to-water temperature gradient in cold water and the same validated by examples from the literature. Last, hypothetical examples are given of the role cutaneous blood flow must play in controlling heat flux and temperature gradients across the subcutaneous fat layer.

Lack of diurnal effects on periodic exercise during prolonged cold water immersion

Doubt TJ and Smith DJ
Undersea Biomed Res. 1990; 17(2): 149-157

Diurnal effects on periodic exercise were examined in 8 male divers wearing passive thermal protection during whole body immersions in 5°C water for periods of up to 6 h. Studies were done during the course of 5-day air saturation dives at a depth of 1.61 ATA, with immersions begin-

ning at 1000 h (AM) and 2200 h (PM). During each hour of immersion, leg exercise was done for 3 min each at workloads of 50, 70, and 90 W. Heart rate (HR) at each workload increased uniformly with immersion time, without a change in slope of HR vs. workload. No AM or PM differences occurred. AM resting VO_2 increased linearly, and to the same extent as PM, with exposure time. VO_2 at 50 W also increased at the same rate as resting values. VO_2 at 70 and 90 W were similar for AM and PM and did not vary significantly during the 6-h immersions. Temporal increases in exercise HR may reflect cardiac compensation of diminished plasma volume. Workloads ≥ 70 W generate enough metabolic heat in this specific condition to meet the thermogenic requirement. Lack of diurnal effects on exercise variables may be due to environmental conditions suppressing circadian rhythms.

From
Diving Medicine Department Naval Medical Research Institute Bethesda, Maryland 20814-5055

Prolonged whole body immersion in cold water: hormonal and metabolic changes

Smith DJ[1], Deuster PA[2], Ryan CJ[2] and Doubt TJ[1]
Undersea Biomed Res. 1990; 17(2): 139-147

To characterise metabolic and hormonal responses during prolonged whole body immersion, 16 divers wearing dry suits completed four immersions in 5°C water during each of two 5-day air saturation dives at 6.1 meters of sea water. One immersion began in the AM (1000 h) and one began in the PM (2200 h) to evaluate diurnal variations. Venous blood samples were obtained before and after completion of each immersion. Cortisol and ACTH levels demonstrated diurnal variation, with larger increases occurring after PM immersions. A greater than three-fold postimmersion increase occurred in norepinephrine (NE). There were significant increases in triiodothyronone (T_3) uptake and epinephrine, but no change in T_3, thyroxine, thyrotrophic hormone, and dopamine. Postimmersion free fatty acid levels increased 409% from preimmersion levels; glucose levels declined, and lactate increased significantly. Only changes in NE correlated significantly with changes in rectal temperature. In summary, when subjects are immersed in cold water for prolonged periods, with a slow rate of body cooling afforded by thermal protection and intermittent exercise, hormonal and metabolic changes occur that are similar in direction and magnitude to short-duration unprotected exposures. Except for cortisol and ACTH, none of the other measured variables exhibited diurnal alterations.

From
1 Diving Medicine Department, Naval Medical Research Institute, Bethesda, Maryland 20814-5055
2 Department of Military Medicine, Uniformed Services University of the Health Sciences, Bethesda, Maryland 20814-4799

The resuscitation of immersion victims.

A.D. Simcock.
Appl Cardiopulmonary Pathophysio 1989; 2: 293-298.

This paper reviews the outcome of 150 victims of drowning and near-drowning brought to a district general hospital close to the sea. The pathophysiology of the drowning process is reviewed. Patients were treated immediately on arrival by a resuscitation team. Respiratory difficulties were relieved as quickly as possible. Common problems were hypoxia, hypothermia, acidosis and low blood pressure. The apparently dead were assessed very carefully. The results show an excellent prognosis for those patients who had not suffered cardiac arrest before arrival. There were, however, two survivors from the cardiac arrest group. Survival rates in this group will only improve if the hypoxia is relieved before cerebral damage occurs.

OXYGEN TOXICITY

Biochemistry of reoxygenation injury.

L.Ernster.
Crit Care Med 1988 Oct; 16(10): 947-953.

This paper summarizes current knowledge on the biochemistry of oxygen toxicity in general and ischemia-reoxygenation tissue injury in particular. The superoxide radial, hydrogen peroxide, and the hydroxyl radical in cells can be formed enzymically or non-enzymically. Primary effects of oxygen radicals result in lipid peroxidation, which is believed to be initiated by a perferryl radical. Secondary effects are believed to be due to a disturbance in cellular calcium homeostasis. Reactions and treatment potentials are highly complex and their effects on cells, tissues, and organism are difficult to predict. Treatment potentials include superoxide dismutase, catalase, calcium entry blockers, iron chelators, xanthine oxidase inhibitors, and agents to prevent leukocyte adhesion. Reoxygenation injury mechanisms during resuscitation from clinical death can be studied in animals by evaluating the effects of antireoxygenation injury therapies and by monitoring free radical reactions.

SELECTED ABSTRACTS

Reprinted from the Program and Abstracts of the UNDERSEA AND HYPERBARIC MEDICAL SOCIETY ANNUAL SCIENTIFIC MEETING 1989

CHAMBER PROBLEMS

Temporary Pacemaker Malfunction in the Hyperbaric Chamber.

Brian A. Youn M.D.* and Ken Kozikowski M.D.+
* Geisinger Medical Center Danville, P.A. 17822, U.S.A.

+ Maryland Institute for Emergency Medical Service Systems, Baltimore, M.D., U.S.A.

With the growth of hyperbaric medicine in recent years, the treatment of critically ill patients with temporary pacemakers is becoming more common. Most of these patients have temporary pacemakers only for prophylaxis or "back-up" if their own intrinsic rhythm deteriorates. Few, however, are pacer dependent and lack any intrinsic rhythm. The latter group is potentially the most dangerous and requires special attention for inadvertent lead displacement and primary generator failure. There is surprisingly little reference to temporary pacemaker function in the hyperbaric chamber. In our experience, we have had temporary pacemaker malfunction at 120 fsw during a pressure test. This failure prompted an objective assessment of temporary pacemaker function in the Hyperbaric Chamber. We studied two models of the more popular temporary pacemakers, Medtronics Model 5330 (A.V. Sequential) and Model 5375 (VVI). Both pacemakers failed abruptly, at 120 fsw for the AV Sequential and 130 fsw for the VVI Pacer. Function for both units returned during decompression. Rate and Ma output did not change up to the point of failure. Medtronic temporary pacer models 5330 and 5375 functioned well up to 120 fsw which allows safe use within the chamber for most routine treatment tables up to 3 ATA; for 6 ATA table, a though-the-hull modification will allow safe usage of Medtronic pacemakers.

VENTILATORS IN CHAMBERS

The Penlon Oxford Ventilator - A second look.

Brian A. Youn, M.S. and Rodney Houseknecht, CRTT.
Department of Critical Care Medicine and Hyperbaric Medicine, Geisinger Medical Center Danville, P.A. 17822, U.S.A.

The Penlon Oxford Ventilator is a commonly used ventilator in the multiplace chamber. This ventilator is a compact, volume-cycled, pneumatically driven, control mode ventilator that has been proven to be safe to 46 ATA. Many patients have severely altered pulmonary mechanics including pneumonia, emphysema, obstructive lung disease, and ARDS. These changes in compliance and resistance may significnatly affect the function of the Penlon ventilator. The Penlon was tested serially from surface to 6 ATA with a TTL test lung set on various compliance and resistive settings and were recorded on an 8-channel Hewlett Packard recorder. Measurements included flow, volume and pressures (proximal, machine and intrapleural). Flow, pressure, respiratory rate, and volume changed dependent on depth, resistance, compliance and machine working pressure. Intrinsic positive end expiratory pressure developed as a function of increasing resistance, suboptimal I:E ratios and increasing respiratory rate. Awareness of these changes are important to optimize ventilator function.

RECALLS

TYPO FORCES NAUI TO RECALL DIVE TIME CALCULATOR

In the latest issue of *Sources* , a NAUI publication, there is a splashy full-page ad for NAUI's dive time calculator. It reads: "In the time it takes to read this page, you can plan an entire day's worth of diving with the NAUI Dive Time Calculator...."

Unfortunately, the folks at NAUI should have taken a little more time than that to read their calculator before they put it on the street. On a subsequent full page in *Sources* is an "immediate recall.... as we go to press". You see, the Dive Time Calculator is replete with typos that render too much time for dives beyond 100 feet.

Dennis Graver, NAUI Director of Training, fesses up to the error. In an effort to get the planner out quickly, NAUI staff proffed the data from faxes, which had to be cut up and pasted to read. The numbers did not get aligned correctly, Graver told *UNDERCURRENT* , and some got moved a single space. The error was not detected until after the printer was given the go ahead to print, assemble and ship.

Fewer than 2,000 of the faulty planners have been distributed. They can be returned to NAUI Headquarters for full credit on the new version.

The new model should be available in February, and some improvements are being added as well. If you have a version of the NAUI dive planner (any version in circulation prior to February 1 is faulty) return it to: NAUI Headquarters, P.O. Box 14650, Montclair, CA 91763-1150.

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The address of UNDERCURRENT is P.O. Box 1658, Sausalito, California 94965, USA.

DACOR RECALL

If you own a DACOR M18 or M19 console, stop using it. You could be endangered by a mid-dive loss of depth and bottom time information normally displayed.

Vern Peterson, Director of Manufacturing for Dacor, told *UNDERCURRENT* that "we've had ongoing reports of problems with these units. The M18 battery light would come on when the battery was still in good condition. We went to the M19 to solve the problem, and found an incompatibility between the electronic module and the battery which caused a drain on the battery. We had a lot of complaints from the field, particularly as these batteries cannot be replaced in the field".

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sportdiving
MAGAZINE

Dacor initiated a recall program, notifying its dealers to replace the consoles and to stop selling the older modules. However, some places in the Caribbean were still selling the units, so, Peterson said, "we instituted a recall program to get them off the market".

According to Peterson, Dacor has no reports of accidents stemming from a malfunction. He said that 80 percent of the units have been returned.

Dacor will replace their MI8 or MI9 electronic consoles with the new MI10 console no charge. Contact your local Dacor dealer or Dacor headquarters for information on how to go about returning your unit.

Dacor Corporation is located at 161 Northfield Road, Northfield, IL 60093. Telex 289499. Fax (312) 446-7547. (800) 233-3483.

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The address of UNDERCURRENT is P.O. Box 1658, Sausalito, California 94965, USA.

COURSES AND MEETINGS

DIVING MEDICINE FOR DOCTORS
OCTOBER 29th to NOVEMBER 2nd 1990
HYPERBARIC MEDICINE UNIT
FREMANTLE HOSPITAL

A course will be held at Fremantle Hospital from October 29th to November 2nd 1990.

The aim is to give medical practitioners the necessary knowledge to carry out appropriate medical examinations on divers.

Course fee $ 500.00

For information and bookings contact

The Director, Hyperbaric Medicine Unit,
Fremantle Hospital,
P.O.Box 480, Fremantle,
Western Australia 6160.

Telephone (09) 431 2233
Fax (09) 335 9868

DATAMAX SPORT

How DataMax Sport Works

Activation

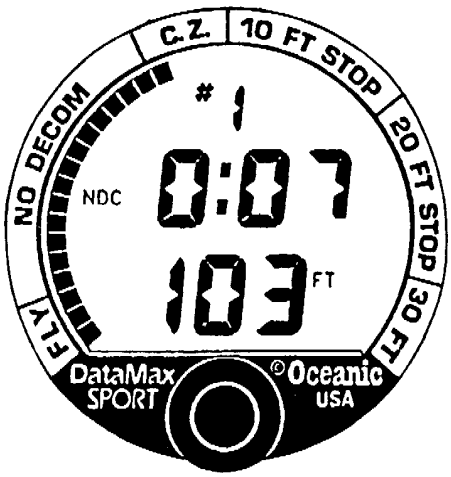
Press the button on the face of the computer and all segments of the LCD display are shown. The DataMax Sport goes through a self-diagnostic check, and if anything is wrong internally, it shuts down. If everything is O.K. the Sport continues with a battery check.

At The Surface

While on the surface, DataMax Sport displays your previous dive number, run ning surface time, current depth, and tissue loading.

Pre-Dive Planning Sequence—PDPS

Every fifteen seconds, your surface display is replaced by the PDPS. This display helps you plan your dive by showing how long you can stay at depths from 30 to 160 feet. This information is based upon your previous dive profiles and nitrogen build up.



While Diving

While no-decompression diving, the DataMax Sport displays dive number, no-decompressions (NDC) dive time remaining, present depth, current tissue loading and in a separate display that comes on screen every 12 seconds running bottom time. Tissue loading is shown by a bar graph that fills the perimeter of the display. The bar graph corresponds to a colour coded dial, showing how close you are to a no-decompression limit.

During a decompression dive the bar graph fills past the yellow Caution Zone, into one of the three decompression stop segments. The graph now shows your safe ascent "ceiling", how far you can ascend given your current nitrogen uptake. The NDC indicator changes to DEC (decompression) and the display now indicates the amount of time necessary at your decompression stop for safe ascent.

After Your Dive

The DataMax Sport logs up to 7 of your previous dives. You can hold each dive profile on screen to log it by pressing and holding the activator button. Your DataMax Sport give you the max. depth, bottom time, end-of-dive tissue loading, and whether you exceeded the safe ascent rate. After logging your information the display shows a new Pre-Dive Planning Sequence (PDPS) for your next dive.

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DIVING MEDICAL CENTRE COURSES

SCUBA DIVING MEDICAL EXAMINATIONS

Courses will be held to instruct medical practictioners in Diving Medicine, sufficient to meet the new Queensland Government requirements for recreational scuba diver assessments.

Courses will be held at

Cairns, Queensland
31st August 1990 to 2nd September 1990

Surfers Paradise, Queensland
5th to 7th October 1990

For further details contact

Diving Medical Centre
132 Yallambee Road
Jindalee
Queensland 4074
Phone: (07) 376-1414

ROYAL ADELAIDE HOSPITAL HYPERBARIC MEDICINE UNIT COURSES IN DIVING AND HYPERBARIC MEDICINE, OCTOBER 1990

BASIC COURSE
Royal Adelaide Hospital, Adelaide.
15 - 19 October 1990.

Concentrates on the assessment of fitness of candidates for diving. HSE-approved course.
Cost: $A 500.00.

ADVANCED COURSE IN DIVING AND HYPERBARIC MEDICINE
Royal Adelaide Hospital, Adelaide.
22 - 26 October 1990.

Discusses the diving-related, and other emergency indications for hyperbaric therapy.
Cost: $A 500.00 ($A 800.00 for both courses)

FURTHER INFORMATION AND ENROLMENT
For further information, or to enrol, contact:
Dr John Williamson,Director, HMU,
Royal Adelaide Hospital, North Terrace,
SOUTH AUSTRALIA, 5000.
Telephone (61) (08) 224 5116).

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JOINT MEETING ON DIVING AND HYPERBARIC MEDICINE
ASSOCIATED COURSES

Dr David H. Elliott is preparing a modular pre- and post-course, meeting the training recommendations of DMAC and NATA on the Medical Aspects of Diving Accidents and Illnesses. The plans are as follows:

Compressed Air Diving. A practical course in diving medicine for doctors, at Den Helder with the Royal Netherlands Navy, 6-9 August 1990. Cost N fl 2,046.

The Medical Management of Decompression Illnesses. An advanced seminar and review, at the Hotel Okura Amsterdam, 10-11 August 1990. Cost N fl 558.

Mixed Gas Diving. An advanced course for doctors at the Cattedra di Medicina Subaquea ed Iperbarica and with the co-operation of the Italian Navy, 20-24 August 1990. Cost N fl 1,860.

For additional information, please contact
Dr. David H. Elliott, Courses Co-ordinator,
Biomedical Seminars, 7 Lyncroft Gardens,
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