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South Pacific Underwater Medicine Society

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

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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

Abbreviations do not mean the same to all readers. To avoid confusion they should only be used after they have appeared in brackets after the complete expression, e.g. decompression sickness (DCS) can thereafter be referred to as DCS.

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PROJECT STICKYBEAK

This project is an ongoing investigation seeking to document all types and severities of diving-related accidents. Information, all of which is treated as being **CONFIDENTIAL** in regards to identifying details, is utilised in reports and case reports on non-fatal cases. Such reports can be freely used by any interested person or organisation to increase diving safety through better awareness of critical factors.

Information may be sent (in confidence) to:
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EDITORIAL

When Cousteau and Gagnan produced the first practical demand valve in 1943 they would undoubtedly have believed that its sole effect would be to liberate divers form the tyranny of the air hose, which tethered them to a small area of the sea floor, and the cost of a surface support team. Indeed it set divers free in the water column. A change from the two dimensional sea floor territory to the free swimmer's three dimensional world. If an environmental impact study had been required it is probable that they would have made reference to the recreational, scientific and commercial applications of their invention. But it is extremely unlikely that there would have been any awareness, let alone mention, of the less tangible effects of this change in diving methods and the introduction of a new type of div er. As Neil Bohum has said "it is hard to predict, especially the future". And indeed in this same year, 1943, Mr Thomas Watson, the then head of IBM, gave it as his opinion that the world market for computers would be satisfied by five machines. The consequences of the introduction of the "Aqualung" are still not fully worked through, indeed the recent sad demise of the too-good-to-be-true National Safety Council of Australia (Victorian Division) has merely accelerated one phase of the revolution in diving which this invention started. It is the way of the world to fail to predict the social consequences of the introduction of any new technology.

Until the adv ent of the "Aqualung" and its clones and rivals there was, by and large, no such creature as a recreational diver. Few had the influence, charm (and presumably the effrontery) to emulate Professor Henry Milne-Edwards, who persuaded the Paris Fire Brigade to loan his their manually operated air pump and the brass helmet diving outfit designed to rescue people from flooded cellars. This was in 1844 and professors were seemingly persons with high social status. He had this apparatus transported to Sicily and boarded aboard a small vessel, taking with him several strong men to operated the pump. His dive to 7.6 m (25 feet) was the reward for all this effort. How different it is nowadays. The social structure of society has changed superficially since then. But the social order in the diving community has changed far more radically. Until 1943 there were, in general, two classes, those who organised the diving and those who dived. The former, gentlemen or engineers, did no diving. This was left to the rather more disposable labouring class which provided the divers. As they were expected to use equipment of uncertain reliability and never to complain of cold or danger it was only an exceptional diver who ventured critical comments on the methods in use. Naturally they had no training, for not only would this have been considered a dangerous innovation taking some of the mystery from the craft but there was no worthwhile theory of diving to impact on them. This comment may seem a little harsh when one considers the efforts of Haldane, Behnke and other naval medical officers to produce safer diving conditions, but it did apply to the non-naval divers.

The influence of recreational divers brought into diving critical minds, persons who could, and did, ask questions and demand answers. A revolution indeed. It was the influence of such persons which produced the snorkel, the modern face mask with its nose pocket, the buoyancy vest, the weight belt with quick release buckles and the wet suit. And of course a questioning attitude to the problems they met. Indeed but for these possibly unwelcomed amateurs in the diving world it is probable that the diving table would still be regarded as sacrosanct eternal truths, with the assumption that every decompression sickness victim, rather than the majority, having caused their own problems. In recent years the existence of the commercial interest in servicing recreational divers has had the unexpected effect of blending some of the original diving establishment members into the civilian diving world. There has been a great reduction in the division between the official experts (usually naval officers) and civilian divers. There was for long and uneasy dichotomy in diving, the Navies treating the recreational diver as inferior rather than as different and having a tendency to sermonize when presented with decompression sickness rather than seeking to learn more about the condition. But with the increasing development of a system of civilian operated chambers in Australia, New Zealand, the U.S.A. and the United Kingdom (and the cost restraints on naval resources) a different and better relationship should develop. And with it an awareness that there remains much that each group can learn from investigating the details of diving related problems.

At the present time the incidence of decompression sickness in recreational divers cannot clearly be sheeted home with any certainty to the limitations of the tables, diver ignorance, imperfect programming of dive computers or other factors. Probably a mixture of all these is involved. There is a need to define the true risk/ safety trade off of multiple dives and multiple depth dives, to mention only a couple of matter which have not received adequate attention up to now.

The papers in his issue require no "commercial" as they are full of useful information and should satisfy the interests and needs of even the most fastidious of readers. The trio of papers from the Hobart meeting about oxygen produced free radicals should give pause for thought to all divers as they will increase theoutput of free radicals every time they submerge and the deeper they go the higher the production.. The lesson from Dennis Graver on how to vomit safely underwater is another item for all divers to note. The two books reviewed are highly recommended, especially for doctors who dive and for those who treat patients with diving related illnesses. On a final, but hardly lighter note, if anyone knows about a particular gravestone on Thursday Island please contact Nick Cooper (page 145).

ORIGINAL PAPERS

SCUBA DIVING FATALITIES IN AUSTRALIA AND NEW ZEALAND

1. THE HUMAN FACTOR

Carl Edmonds and Douglas Walker

Background

The USA Underwater Diving Fatalities Statistics [1,2] have until recently been compiled almost single-handedly by John J McAniff, Director of National Underwater Accident Data Centre (NUADC), University of Rhode Island. These records, which go back to 1970, include more than 2,600 fatalities, and are unsurpassed in their scope.

In Australia and New Zealand (ANZ), the deaths are less numerous but data is more detailed and is comprehensively catalogued. It is collated as "Project Stickybeak", run by Dr Douglas Walker and the provisional reports with case histories are published[3] and made available to instructor and other supporting organisations. All material acquired is confidential and no identifying details are included in the use of this information to promote safer diving operations.

There is national cooperation in the supply of data in both Australia and New Zealand. Copies of depositions, police interviews, witnesses statements and inquest proceedings are supplied by the Law/ Justice / Attorney General's departments in each state. The Water Police, Police Divers and Navy Diving Units' reports include bench testing of the diving equipment used by the deceased, compared with the national standards for compressed air equipment, and often in-water trials where an experienced diver, of similar stature, employs the equipment in a simulation of the dive profile. Gas analyses are routinely carried out at government laboratories.

Because of the excellent publicity achieved by the people and organisations involved, virtually all ANZ diving instructors and dive shop proprietors, as well as most established divers, are aware of "Project Stickybeak". This ensures that few diving deaths are missed, and much more information may be collected than is available to other individual agencies.

This report is the first of three extensions of "Project Stickybeak", and deals with an analysis of the human factors contributing to the death. It encompasses medical information, psychological problems and various diving techniques that imply questionable judgement. The second extension deals with faults and misuse of equipment and the third with environmental factors.

Both authors have had many years (1967-1989) of investigating diving accidents and diving deaths, incorporating both the civilian and Armed Services diving activities.

Survey Material

In this ANZ survey, information was compiled on factors contributing to 100 consecutive scuba diving fatalities in this decade. The NUADC figures quoted for comparison were calculated from the 1980-1987 reports.

For diving fatalities to be included, the following requirements had to be met:
1. Scuba gear had to be worn by the victim in the water, with the intent of diving.
2. All military, large commercial or helium diving activities were omitted.
3. At least three of the following four sources of detailed information
   a. A coroners inquest or enquiry (full transcript including witnesses declarations and cross examination);
   b. Autopsy findings (anatomy, histology and toxicology in full detail);
   c. Official government (Navy, Water Police, etc.) assessment of equipment functioning and in-water trials. This includes gas analysis on scuba tank compressed air.
   d. Detailed written accounts of witnesses, (buddies, other divers, boatman or bystanders, rescuers)

As well as these, additional information was sometimes available from newspaper articles, underwater and diving agencies, including instructor organisations, private sources and rescue services.

Inevitably, judgements had to be made regarding the relevance of much data. Often there were minor or major abnormalities, mistakes, difficulties or problems encountered, which did not appear as if they were related to the incident leading to death. Unless stated otherwise, these have not been incorporated as contributing factors in this paper.

In the ANZ series all factors which were likely to have materially contributed to the sequence of events which led to death, or prevented action being taken which would have led to a successful rescue, were recorded.

The results were categorised as follows

DIVING DATA

This gives an overview of the diver, the type of diving, the behaviour of the diver and observers. Related

statistics from the NUADC data are supplied for general interest. We have not attempted to replicate the basic data of NUADC or Project Stickybeak.

MEDICAL CONTRIBUTIONS

These include psychological (e.g. panic, fatigue), physiological (e.g. vomiting, extreme physical unfitness) and pathological conditions (e.g. pulmonary barotrauma, drowning) leading to death.

DIVING TECHNIQUE CONTRIBUTIONS

Although this survey does not extensively analyse the diving data, certain diving procedures or techniques that involve human judgements and have been perceived as having an influence on diving deaths are recorded. These include diving experience, out-of-air situations, buddy diving, weight ditching and buoyancy problems.

EQUIPMENT CONTRIBUTIONS

These are subdivided into equipment faults and equipment misuse because, for example, it seems unfair to impute fault to the weight belt because it was misused, e.g. by being worn under the buoyancy compensator harness and thus failed to be ditched in the emergency.

If, however, the weight belt has no fault and is correctly worn, but is inappropriately (voluntarily) either ditched or retained so as to contribute to the accident, then this is rightfully attributed to poor diving technique or judgement, and is included in this report.

ENVIRONMENTAL CONTRIBUTIONS

These include both natural hazards (e.g. tidal currents, sharks) as well as non-scuba related man-made hazards (e.g. boats, dam outlets). In this paper the environmental contributions are not further categorised as they are the basis of a subsequent report..

If the diver had attempted to dive under conditions for which he was clearly untrained and inexperienced, then this is seen as an error of judgement and is referred to in this report.

Results and Conclusions

As there were 100 cases, each case represented 1 % of the total. Even where information was not available, the figures still represented a percentage of the 100 cases.

Diving data

Despite the different method of selection of cases, the NUADC and the ANZ series showed remarkably similar profiles of diving activities. In most cases the accident came as a great surprise to all associates of the deceased, but in 9 % the victim had been specifically advised by a diving medical expert, and sometimes by a dive instructor, that they were unfit for scuba diving.

Table 1. DIVER DATA

	NUADC	ANZ
Average age (years)	33.1 yrs.	32.9 yrs
First Scuba dive	5.4 %	8 %
Under training	9.0 %	5 %
Multiple deaths	9.8 %	4 %
Diving alone	17.5 %	21 %
Male/Female ratio	9:1	9:1
Age > 50	9.7%	8 %

Age. The age range was 13 to 65 with majority between 21 and 35, and a small increase around 46-50. The latter was related to the “cardiac deaths” which had their peak in this age group

Table 2. AGE INCIDENCE

Age	No
11-15	1
16-20	8
21-25	21
26-30	19
31-35	21
36-40	9
41-45	3
46-50	10
51-55	5
56-60	1
>60	2

Depths. The depths of the dive, the initiating problem and of unconsciousness (or death) are shown in table 3. A small number never descended at all, and over a quarter first encountered their trouble on the surface. Despite the wide range of diving depths, at least half either died or lost consciousness on the surface. In 14 % of cases the fatal dive was a repetitive one.

Duration. In 17 %, the diver succumbed in the first 10 minutes of the dive. In 56 % the problem developed following an exhaustion of the air supply (either on reserve or out of air). In 8 % it was intermediate between these times.

It would seem reasonable to conclude that, in planning a dive, accidents could be anticipated more often at the start or at the end.

Table 3 DIVE DEPTHS

Depth in metres	(ft)	Dive	First problem	Unconsciousness
Surface 0	(0)	3	28	50
1-3	(1-10)	4	3	2
4-6	(11-20)	13	10	7
7-9	(21-30)	12	3	5
10-12	(31-40)	12	4	4
13-15	(41-50)	9	5	3
16-18	(51-60)	7	1	1
19-21	(61-70)	7	-	2
22-24	(71-80)	4	1	1
25-27	(81-90)	6	3	4
28-30	(91-100)	1	1	1
31-45	(101-150)	5	-	4
46-60	(151-200)	3	4	3
69	(226)	2		
92	(302)	1		

Purpose. The purpose of the dive was recorded, but is not designated as a contributory cause in this presentation.

Table 4. PURPOSE OF THE DIVE %

Recreational dive	30
Hunting	30
Photography	7
Introductory dive	7
Work activity	6
Cave diving	5
Under instruction	5
Instructing	2
Wreck diving	3

Responses. Once a problem has developed, even though the surface was sought in most cases, the weight belt was rarely ditched and the buoyancy compensator (BC) was not inflated, either at the surface or at depth.

Table 5. BEHAVIOUR OF VICTIM %

BC	inflated	21
	not inflated	48
Weights	ditched	9
	not ditched	83

Table 6. BUDDY RESPONSE %

Assisted with air supply	11
Ditched victim's weight belt	12
Inflated victim's BC	10
Surfaced with the victim	12
Rescue and/or first aid	23
Buddy breathing failed	4

When the buddy remained with the victim, or eventually found him, there was usually an appropriate response. Only in one case did the rescuer become a victim.

Buddy breathing seemed to cause some problems, especially during ascent.

Overview of contributing factors

The number of contributory factors increased with the detail available of the dive. A "sole cause", such as a shark attack or an inexplicable burst lung, was a rarity, except in the divers who dived alone, when our records are probably not complete.

In allocating cases , each victim was recorded only once in each major category (Table 7).

As well as these contributory factors, certain diving techniques or activities were likely to have contributed to the final event. These are

Table 8. DIVING TECHNIQUES

Inadequate air supply	56%
Buoyancy problems	52 %
Other equipment misuse	35 %

Medical disorders

Cause of death. Even though an understanding of the events is not obtainable by autopsy findings alone, they

Table 7. MAJOR FACTORS

	NUADC *	ANZ Series		
		Probable	Likely	Total
Medical disorders	55.7 %	59 %	43 %	74%
Equipment faults	9.5 %	23 %	18 %	35 %
Environmental	34.8 %	52 %	18 %	62 %

* The results were not strictly comparable with the NUADC series, which had less information available on each death, did not use identical classifications, and only recorded one contributing factor and then in only 73 % of cases.

are indicative of the final event. The following results were derived from the conclusions from the autopsy findings, more than from the formal coronial findings.

Table 9. CORONER / AUTOPSY . Officially Designated Cause of Death

	ANZ	NUADC
	125 causes for 100 victims	
Drowning	86 %	74.2 %
Pulmonary barotrauma	13 %	24.5 %
Cardiac	13 %	9.1 %
Aspiration of vomitus	6 %	<1 %
Trauma *	3 %	1.5 %
Asthma	2 %	
Marine animal injury *	1 %	
Co-incidental**	1 %	

* Catalogued as environmental in the ANZ series
** The co-incidental cause was a dissecting aneurysm of the aorta.

In the final assessment only the past medical or physiological disorders which were thought to influence the death of the victim were included in table 10. Rarely was the past medical history available. For this reason, the "Pre-existing" figures must be considered as underestimates of the true situation.

In Table 10 cases marked with * were not recorded as contributing factors unless other related disorders co-existed. Excluding these, in 25 % of the cases there was a pre-existing medical contraindications to scuba diving. This compares to an overall "failure rate" of almost 10 %, during the 1980s, amongst recreational scuba divers who attended the Diving Medical Centres in Sydney (7).

Many of these factors were subjective, such as fatigue and panic, and we had to rely on witnesses descriptions. They are fortunately usually associated with other environmental, equipment or technique problems.

Salt water aspiration while the diver was still conscious, was likewise an unverifiable factor and relied on data from others. In most cases it was overtaken and pathologically obscured by its logical extension, drowning.

Patients who have had diabetes, epilepsy and cardiac surgery are, like asthmatics, excluded from diving suitability, both in the customary medical examinations and the signed declarations required by diving instructor organisations. This has not prevented them from settling in these statistics

Stress responses, fatigue and panic. These subjective symptoms are "soft" data that can only be presumed by a detailed description of the diving activities. Nevertheless, they occur frequently throughout the fatality case reports. To dismiss them because of the inability to demonstrate morbid pathology, would be to ignore two of the major contributory causes of diving deaths.[4]

Panic is a psychological stress reaction to anxiety. The threat of death is a reasonable cause of anxiety. Under selected circumstances, anyone will panic. Difficulty in obtaining air is a frequent cause of panic and the inhalation of water was associated with panic in 19 % of the cases.

Table 11. STRESS LEADING TO PANIC n = 39

Salt water aspiration	19
Fatigue	16

Fatigue is a physiological stress reaction to a muscular effort which was often underestimated by the victims. Under sufficient physical stress anyone can become fatigued. Salt water aspiration, panic and cardiac disease all occurred more frequently than would be expected in these cases.

Table 10. MEDICAL CONTRIBUTIONS
(Excluding drowning)

	Pre-existing	Probable	Likely	Total
Panic		31	8	39
Fatigue		23	5	28
Vomiting	1	6	4	10
Nitrogen narcosis		7	2	9
Drugs	*8	1	6	7
Very physically unfit	4	3	1	4
Severe disability	3	1	2	3
Severe visual loss	3	1	2	3
Alcohol		2		2
Motion sickness	2	1	1	2
Gross obesity	*8		2	2
Carotid sinus reflex		1		1
Salt water aspiration		22	15	37
Pulmonary barotrauma		10	3	13
Cardiac disease	3	7	5	12
Asthma	9	6	2	8
Respiratory disease	5	3	4	7
Hypothermia		1	2	3
Hypertension	*8		2	2
Ear problem	2	1	1	2
Diabetes	1	1		1
Others		1		1
Epilepsy	1			
Decompression Sickness				nil
Contaminated air supply				nil

* = not recorded as contributing factors unless other related disorders co-existed.

Table 12. FATIGUE (28 cases)

Salt water aspiration	18
Panic	16
Cardiac disease	9
Nitrogen narcosis	3
Severe disabled	2
Hypothermia	2
Very physically unfit	2

Vomiting. After exclusion of those cases in which vomiting happened after removal of the victim from the water or as a terminal event, it initiated or complicated the event in 10 % of the cases.

Table 13. VOMITING (10 cases)

Salt water aspiration	4
Regulator leaking	3
Sea sickness	2

Nitrogen narcosis. This contributed to the death in 9 % of cases, but was never the sole or major cause.

Drugs. Alcohol, carbon monoxide and narcotics were tested during the autopsy in most cases. Otherwise the information was obtained fortuitously, and therefore be an underestimate. Cannabis was used once, but was not considered a contributor (Table 14).

The relationship between alcohol intake and drowning is well described elsewhere.[5] The higher incidence of cardiac deaths amongst those with hypertension and treated with hypotensive drugs, is probably also predictable.

Salt water aspiration. While still conscious, this was present in 37 % of cases and was a interim factor, following some other event such as using a snorkel in white water or an out of air situation. Problems with the regulator occurred in 12 cases and were therefore unexpected. The result of the inhalation of water is seen in the associations between this and other medical contributions.

Table 14. DRUG INTAKE

Illness	Number	Drug	Cause of death
Asthma	9	Salbutimol	Pulmonary barotrauma (2)
			Drowning (7)
			Cardiac (1)
Hypertension	5	Hypotensives	Cardiac (4)
			Drowning (4)
N/A	4	Alcohol excess	Drowning (4)

Table 15. SALT WATER ASPIRATION
n = 37

Panic	19
Fatigue	18
Cardiac disorder	9
Asthma	6
Hypothermia	3

Pulmonary barotrauma. This was evident in 13 % of cases. In some cases the extensive pulmonary damage was obvious, but in others it was complicated by the effects of subsequent drowning. The clinical presentation of classical cerebral arterial gas embolism was considered adequate to make the diagnosis even without pathological verification.

The suddenness of these cases made other observations more difficult, however some associations were noted.

Table 16. PULMONARY BAROTRAUMA
n = 13

Panic beforehand	5
Nitrogen narcosis	3
Emergency ascent	2
Asthma	2

Cardiac disease. Of the 12 % of divers who died of cardiac disease, there was 2 cases of myocarditis, pathologically demonstrated and in young divers who had intercurrent illnesses. The average age was 43.6 years, (S.D.= 7.6). The mode was in the 46-50 years age group, with 5 deaths, and 3 between 51-55 years. They did tend to die quietly.

Three had a history of heart disease and another four of hypertension requiring treatment. With so many possible trigger factors (previous pathology, exertion, cold exposure, prescription drugs including beta blockers, hypoxia from aspiration of sea water, etc.) for both myocardial ischaemia and ventricular fibrillation, it would be hard to incriminate one specific aggravating factor.

Table 17. CARDIAC DEATH
n=12 *

Salt water aspiration	9
Fatigue	9
Drugs	5
Hypothermia	2

* In accepting this diagnosis we have required very gross pathology or an excellent clinical description. If we were to accept all autopsy and clinical diagnoses of cardiac disease, the number would have risen to 21.

Asthma. In no case was the diagnosis of asthma made purely on the basis of histological findings. There is a 24 hour delay in the production of the characteristic eosinophilic infiltration and desquamation changes with an acute attack of asthma.[6] This could well have reduced the apparent influence of this illness, but it was somewhat compensated for by the ability of some of the pathologists to detect and record the chronic signs of asthma.

Of the 9 % who had asthma, the following information was found:

Table 18. ASTHMA
n = 9

Autopsy cause of death	
drowning	7
pulmonary barotrauma	2
Medical contributions	
salt water aspiration	5
fatigue and/or panic	5
Technique problems	
compromised air supply	6

When the factors for asthma provocation in scuba diving are considered, namely ;

exertion
inhalation of cold, dry air
hypertonic saline inhalation

and it is realised that each of these stresses are used clinically to initiate asthma as a diagnostic provocation tests[6] the high incidence of this disorder is understandable.

It is not known whether breathing against an increased inspiratory resistance (low on air) is also a provoking factor, but this should now be considered in the light of these figures.

Respiratory disease. Four cases of the seven had respiratory infections and two had pleural adhesions (one from a thoracotomy, and one who died with pulmonary barotrauma, pneumothorax and a large haemothorax). Dyspnoea on walking on the flat and a Peak expiratory flow rate of 320 litres per minute, were considered evidence of probable respiratory disease.

Diving techniques

An assessment of certain dive procedures was made as regards both the frequency of these in fatal diving accidents and their contributions to this. As we have no idea how often they have saved lives in other circumstances, these figures should not necessarily be used to condemn any practice, but at least to review it.

Experience. The fatal dive being undertaken was compared in complexity to the diver's training and previous experience.

Table 19. EXPERIENCE

Nil experience	8 %
i.e. never scuba dived before	
Inadequate	39 %
Consistent	43 %
Excessive	6 %

Air supply. As 56 % of the problems developed after the air supply had reached reserve levels (low on air and out of air), it could reasonably be concluded that the divers found it more difficult to handle problems under those conditions. This tallied with the observations on the number of "surface" deaths, and the problems of coping with surface swimming conditions.

Table 20. AIR SUPPLY

Snorkel breathing on surface	8 %
out of air or low on air noted on surface	7 %
out of air or low on air noted at depth	49 %

Most problems develop from the time the victim became aware that the air supply was compromised. To avoid unnecessary air consumption, snorkelling on the surface was employed and coincided with the development of problems in 8 % of cases. One of the other ways of producing a low on air situation was by the victim using either a cylinder smaller than normal or a cylinder with less than customary air pressure (9 %). Most had contents gauges.

In the case of the small cylinder, not only was there less air supply than that available to the other divers, but when the low on air situation developed the actual amount of reserve air was much less than usual. In some of the cylinders, holding only 28 cu ft, there was only a few breaths of air once the low on air situation was reached at depth.

Buddy diving. The buddy system, which has universal support amongst recreational diving groups and instructors, appeared to have more verbal than factual acceptance. The divers were therefore assessed, not according to their statements, but according to what happened during the dive. Many alleged buddies were divers who only shared the same boat.

Over a third of the victims were either diving alone or separated voluntarily before the problem developed. One quarter voluntarily separated afterwards!

Table 21. BUDDY BEHAVIOUR

Nil. Solo from start.	21 %
Voluntary separation	
before any problem	13 %
Voluntary separation	
after a problem commenced	25 %
Separation by the problem	20 %
Not separated	14 %

By far the most common reason for the separation was that one diver (the subsequent victim) ran out of air or low on air, and the buddy decided not to interrupt his diving activities because of this. Occasionally the buddy accompanied the victim to the surface and then deserted him.

The problems that sometimes separated the buddies were uncontrolled ascents, underwater and surface currents, sometimes sudden and unexpected. In only 14 % did the buddies remain together.

Table 22. BUDDY DIVING VARIANTS

Two or more buddies	15 %
Victim follows the buddy	5 %
Victimisation of buddies	2 %

Amongst the small numbers that were classified as buddy divers, there were some practices which seemed to detract from the buddy concept. In 15 % there was not one

buddy, but two or more. This led to considerable confusion as to who exactly was responsible for whom.

In 6% the victim was following the "buddy". Once a problem developed under this system, any observation by the lead diver would have been be fortuitous. To attract the lead divers attention required energy, air and time consuming behaviour on the part of the victim, who could ill afford these commodities once the problem developed. The experienced diver was invariably the one who took the lead, and therefore had the luxury of a buddy observing him at all times.

In two instances there were groups of people being led on a dive. The procedure used was that the first diver to exhaust his air supply would inform the dive leader that he was now "on or near reserve". The dive leader would then take time to determine who else was in or close to a low on air situation. These two divers were then buddied, to surface and return to base.

Thus the dive leader managed to select the two heaviest air consumers, and usually the two least experienced divers, and buddied them together into a situation in which either one was likely to develop a complete out of air situation during ascent , while performing a safety stop, or on the surface. This seemed to be an accepted practice in "resort " areas.

Weight belts. As in previous surveys it was found that very few of the victims, only 9 %, successfully ditched their own weight belts.

Table 23. WEIGHT BELT CONTRIBUTIONS %

Too heavy	45
Not ditched by victim	40
Fouled or unreleasable	6

In 83 % the weight belt was not ditched by the diver and in 40 % this probably contributed to the victim's death. In 3 % it was fouled by being worn under other equipment harnesses. It was unreleasable in 3 %, because of, entanglement with lines, the weight slipping onto the quick release buckle, or the strap being too long and jamming the release on the belt .

Failure to ditch the weights, when in difficulty, presumably reflects on training techniques.

Buoyancy / BC. Many of these problems came under the equipment category, but an appreciable number were clearly errors of judgement and were therefore included as faults in diving technique.

The wet suits available for most of this decade required; 1 Kg weight for each 1 mm thickness, 1 Kg extra for "Long John" extensions and a hood, 1Kg for aluminium tanks, and an extra 1 to 2 Kg for individual variation in buoyancy. In excess of this, the diver was considered to be overweighted and to require extra effort, hyperventilation or reliance on the BC, to remain buoyant on the surface.

Using these criteria it was found that 40 % of the divers were overweighted on the surface. At depth the problem of overweighting was compounded by the loss of buoyancy from the wet suit and body spaces. There is then a much greater effort required for surfacing.

Reliance on the BC inflation then becomes not just a convenience, but an essential.

Apparently many divers have replaced the skills of buoyancy control with heavy reliance on the BC. They are purposely overweighting "to get down", and the BC is inflated to return to the surface. In these cases the BC is relied on not to trim buoyancy with depth, but to return the diver on the surface. Such a procedure introduces the potential for accidents.

Table 24. BUOYANCY CONTRIBUTIONS

Negatively buoyant	
>2 kg on surface, without BC use	40 %
Negatively buoyant	
>2 kg at depth, without BC use	7 %
Positive buoyancy due to BC usage	8 %
TOTAL	52 %

The BC problems included;
- accidental inflation,
- confusion with use (two victims repeatedly confused inflation with dump valves),
- overinflation during ascent (Boyles's law and the Polaris effect),
- inadequate and very slow inflation at depth (especially in a low on air situation)[7],
- mechanical failures and malfunctions,
- ditching problems with some types (involving inflator hoses and harnesses),
- effort required to overcome drag when swimming underwater and on the surface.

Discussion

BACKGROUND

Recently, the purported low death rates in the 1980s were shown to be based on overly optimistic figures and creative statistical interpretations.[8-10] So also was the alleged improvements in safety amongst scuba divers.

These death rates of 16-20 per 100 000 now being proposed, together with the increased death rate per dive increasing to 1 in 95,000, have compelled both the NUADC and the instructor organisations to review and appreciably modify their claims of safety.

The NUADC and Project Stickybeak have conscientiously recorded the number of diving deaths, and this survey extends that effort to understand why such deaths occur.

The ANZ series requires extensive detail of each death. The NUADC records only one cause of death (usually drowning) and, sometimes, one initial contributing problem.

The ANZ series defines all the known contributing adverse factors. It differentiates medical disorders, diving techniques, equipment faults and misuse, and environmental factors.

Consider the case in which a diver descends to 50 metres (165 ft), becomes narcotic and uses all his air. Attempted buddy breathing results in his face mask being accidentally displaced. He then panics. As he commences his ascent he decides to not ditch his weight belt but to rely on his BC. The air in the BC expands rapidly and causes a totally uncontrolled "polaris type" ascent during the last 10 metres. As he hits the surface he gets run over by his safety boat, which could not swerve in time. The diver is knocked unconscious and his BC is damaged in the collision. As he retained his weight belt, the diver sinks to die of drowning.

To record this as "Drowning", even if complemented by one "probable starting cause", is a gross oversimplification of a complex series of interactions, and ignores the many contributing factors, which may have implications for diving safety and instruction.

There are a variety of contributing factors in this example; depth, narcosis, out of air, buddy breathing during ascent, loss of face mask, panic, uncontrolled ascent due to air expansion in the BC, injury from the boat, and the decision not to ditch the weights, are all relevant. These would all be included in the ANZ survey.

Due to the changes that have taken place in scuba equipment and techniques, during the 1980s, this paper is restricted to diving deaths during this decade. The NUADC statistics are similarly restricted to scuba deaths in this period.

DATA COLLECTION

The ANZ cases demonstrate that although diving may be safe under most circumstances, when a number of adverse factors combine, the diver is often unable to cope with the complexities of his equipment and environment.

Although comparisons to the NUADC surveys are inevitable, the populations and the survey data are not really comparable. Press clippings provide the greatest number of cases for the NUADC, and although this may be adequate for deriving gross morbidity figures, it is not adequate to explain the deaths.

The NUADC had coroner findings or autopsy results in 64%. These appear to refer to the official statements or summaries, as opposed to the full transcripts. We have not found these summaries to be sufficiently informative for our purposes of identifying contributing causes.

There is a wide difference of knowledge and expertise amongst officials. Coroners' and other government inquiries are also frequently characterised by naivety in their tendency to oversimplification of "the" cause of the accident, instead of an understanding of the dynamics of the events. They are also influenced by possible criminal responsibility, litigation implications or liability of their statements.

Sometimes autopsy observations are misinterpreted. As a common example, air embolus and decompression sickness have been diagnosed because of the presence of air in the heart and blood vessels. If this air is in the right ventricle, as well as the left, the diagnosis must be questioned. Air can develop in the heart and vessels as a post mortem artifact[5] in divers who have been breathing compressed air at the time of their death ("post mortem decompression sickness").

In most cases of unconsciousness or disablement and and subsequent death while diving, drowning is a common sequel to the loss of the air supply. The pathology of drowning may then dominate the autopsy findings, even though it is not the initial cause of the problem, but only "the final event". For this reason, drowning is not considered an adequate explanation for death in divers, but a common sequel to loss of consciousness underwater.

MEDICAL CONTRIBUTIONS

The fact that some divers were known to have been specifically told by diving experts that they were unfit to dive, suggests that sometimes good advice goes unheeded.

Despite the absence of comprehensive medical examinations in most, and the absence of any premorbid medical data in more than half the cases, it was evident that at least 25 % of the divers were medically unfit to undertake scuba diving, on history alone.

A large number of asthmatics and hypertensives on treatment, as well as the cardiac patients, a diabetic on insulin and an epileptic, are represented in this series. Their presence is incomprehensible, considering that the candidates are required to pass special medical standards for diving, as well as complete a screening questionnaire issued

by the diving instructor organisations. The physicians and the instructors are not applying these standards.

A recent report[11] suggests that the failure of Australian physicians to apply the medical standards, is due to ignorance of these standards and a failure on the part of the physician to appreciate the problems of scuba equipment and the demands of the ocean environment. The reasons for the instructors not applying the standards is not known.

In either case, the current system has not succeeded in selecting out the high risk patients. Physicians and dive instructors are still confusing physical fitness (needed for many sports) and medical fitness (a freedom from medical diseases incompatible with safe diving). Both are required. Many of the deceased divers were said to have been very fit physically, despite having such medical diseases.

If drowning is excluded as only the final event in a sequence of adverse happenings, then the stress problems of panic and fatigue dominate the medical contributions. Because these do not feature in autopsies, they are not fully appreciated in some series. They are interwoven with faults in technique (or training), and with many equipment and environmental provocations.

The importance of these stress factors is contrasted with the great rarity of the high profile diving diseases of decompression sickness and gas contamination, which were absent in this series and noted in less than 1 % of the NUADC series.

The importance of other major contributors that leave little or no evidence at autopsy, such as salt water aspiration, nitrogen narcosis, drug intake, vomiting and asthma, can only be comprehended by a detailed dive history. These do not show up as much in the NUADC series, because of the limitations of the data collection and the decision to only include one contributing factor in most cases.

The NUADC and ANZ series show reasonable agreement on the importance of pulmonary barotrauma and cardiac disease. The latter seems to be an increasing problem. The importance of astute medical selection and then adequate training of divers is axiomatic in the prevention of these.

DIVING TECHNIQUES

In an assessment of diving techniques that imply questionable judgement, we are encroaching on diver training more than diver selection.

The inexperienced and overconfident male was overrepresented in both the NUADC and ANZ series. Diving well within the limitations of the diver, and the equipment, was not a well practiced activity amongst these divers.

The majority who die do so after voluntarily inducing a compromised air situation. They are then forced to surface to breath, or to conserve their emergency air supply. Returning with plenty of air was not common.

The traditional admonition that the surface is the danger area for divers, was supported by the figures showing that at least half the cases lost consciousness and died there. Nevertheless the surface was unavoidable in 56 %, as the diver was in a compromised situation as regards his air supply.

The surface problems were frequently aggravated by the decision not to ditch weights. This also contributed to many of the cases that developed at depth, where a failure to appreciate buoyancy factors resulted in excessive exertion being required.

The training technique of older experienced instructors to require trainees to practice removal and replacement of the weight belt on each dive, could well be resurrected. This practice alone may have prevented the deaths in which the belt was eventually unreleasable.

Instruction to unbuckle the weight belt and hold it at arm's length in all demanding situations, was either not taught or not applied in any of these cases. Yet, had this been done and the situation deteriorated, the belt would have been dropped successfully and the diver made positively buoyant, assisting slow ascent and permitting surface swimming without being overweighted. If the situation had not deteriorated, the diver could have replaced his belt without penalty.

The extreme effort in swimming on the surface with scuba gear, heavy weights and an inflated buoyancy compensator[12], seemed not to be widely appreciated amongst this diving population.

The technique of overweighting, "to get down", and the subsequent strong reliance on the inflation of the buoyancy compensator[7] to ascend and remain on the surface, presumably makes instruction much easier. The failure to learn the skills of buoyancy control[13], without an over-inflation/over-weight trade off, is an expensive lesson not to learn. Dependency on equipment may well be related to the failure to ditch it in an emergency.

Buddy diving, as envisaged in the manuals, is a rare event in these cases. The majority of divers dived alone, and died alone.

Even in the NUADC reports, less than half dived as a buddy pair, and only a quarter stayed together. The ANZ series shewed that a third claimed to try to stay together, and only one seventh actually did. The relatively slight differ-

ences in the numbers probably is explained by the greater dive detail in the ANZ cases.

It seems as if the buddy concept, if used at all, was mainly employed when it was not needed. More buddies voluntarily separated from the victim at the start of problems (usually when low on air) than actually stayed together.

Even when it is applied, the less experienced diver, or the one who will consume more air, is initially given the task of following the more experienced divers until he runs out of air and he is then sent to the surface to swim back alone! Or he is buddied with another low on air diver.

Traditionally, companion diving was recommended and the need was self evident because of the recognition that diving was a hazardous activity. As diving is now promoted as being a safe sport, perhaps the need for companion diving is less appreciated. For uneventful dives this attitude may be adequate. For others it is not.

The observations in both the NUADC and ANZ fatality series for the 1980s, should emphasise the need for buddy diving, in which the divers do genuinely take responsibility for each other for the whole time, until they return to shore or safety. It needs to be taught, understood and practiced.

Conclusion

The real tragedy of this survey was that it shows that the lessons and teachings of yesterday, are still not sufficiently appreciated today. The requirement for a high standards of physical fitness as well as a freedom from many medical diseases, together with training in accident prevention and management, an appreciation of the limitations of equipment and a healthy respect for a potentially hazardous environment, are as important for safe diving now as they ever were.

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PROVISIONAL REPORT ON DIVING-RELATED FATALITIES AUSTRALIA 1987

Douglas Walker

Summary

There were four breath-hold and four scuba diving deaths identified as having occurred in Australian waters during 1987. A common finding in all was that the victim was either diving alone or was separated from others at the critical time, though this was not a invariably a factor which determined the course or outcome. Three of the breath-hold

fatalities involved overseas visitors to the Great Barrier Reef, a statistical quirk of no relevance to the critical factors but of possible importance for other reasons. If it was not that these deaths occurred in association with outings on which people were invited to snorkel dive they might have been regarded as simple misadventure drownings and not included in the present review. However the circumstances of these accidents show how difficult it can be to watch over a group of swimmers who are not firmly under the control of a supervisor. The fourth fatality in this group was unfortunately typical of competition spearfishing breath-hold divers, a post-hyperventilation blackout which was followed by the drowning of the unobserved diver.

The four scuba diving fatalities resulted from four very dissimilar circumstances and each case had some singularity which differentiates them from the general run of scuba diver deaths. There was a shark attack, (the first shark caused scuba diving death recorded in Australian waters), an apparent acute myocarditis death, a gross pulmonary barotrauma (which the pathologist called decompression sickness), and a sea cave death which was probably as the result of water surge which arrived unexpectedly causing the victim temporary but fatal problems.

Case Reports

BH 87/1

The victim was apparently healthy and had decided not to go to view the reef from a glass bottomed boat as he wished to go for a quiet snorkel to view the reef, here close to the beach. His wife was on the beach watching him until he called to her that he had seen a fish he wished to photograph and asked that she go and fetch his camera from their room. When she returned she could see him floating quietly face down a little off the beach, his failure to respond to her return being ascribed to him having become very interested in watching the reef below. It was only after the wake from the returning boat washed over him without him responding in any way that the first suspicion arose that something was wrong, a suspicion which led the boatman to return after disembarking most of his passengers. The victim was unconscious and did not respond to resuscitation efforts. His wife was unaware of anything out of the ordinary until the boat returned to her husband.

The autopsy revealed that he had an enlarged heart, which was mainly left ventricular hypertrophy, and the coronary arteries showed gross atheroma and calcification. Some myocardial fibrosis was noted in the postero-septal area. His wife did not report him as being unfit or on treatment and noticed nothing to indicate he was unwell that day.

SOLO. SEVERE SYMPTOMLESS CORONARY ARTERY DISEASE. CALM WATER. NO INQUEST.

BH 87/2

During the trip out to view the Barrier Reef there was an opportunity for passengers to attend a talk on the correct manner to snorkel dive at their destination, a pontoon moored over one of the reefs. Attendance was optional, the presence of a bar on board being an alternative way to spend the time. The victim was noted as not attending the talk. On arrival at their destination a meal was provided and the victim requested, obtained, and consumed, twice as much or more than most others. He had snorkelled for a time before this meal and returned to the water after eating his fill. There was a person keeping watch over the area close to the dive platform, the same person who had given the safety talk, and passengers who were uncertain of their swimming abilities were offered buoyancy vests to provide them with confidence and safety. It is unknown whether the talk included instructions that swimmers and snorkellers remain in the supervised area but it is unlikely any such a restriction was made as the water conditions were good. The victim was noticed by chance by another crew member, face down at the surface around the side of the pontoon out of sight of the safety man. Both these men entered the water and swam to the victim immediately he was seen. His mask, which contained some blood and vomit, was removed and two quick breaths of EAR were given before taking him to the pontoon to start resuscitation efforts and call the emergency service helicopter.

The autopsy showed hyperaemia of the bronchial mucosa but no vomit was observed. Death from drowning was diagnosed, with the assumption that his large meal played a part in this incident. He may have been experiencing abdominal discomfort then felt that he was about to vomit, inhaled some water, and drowned. There was some coronary atheroma but this was not thought to be of significance.

SEPARATION/SOLO. SNORKEL EXPERIENCE NOT STATED. EXCESS DRINK AND FOOD THEN SNORKEL SWIM. CALM SURFACE. SILENT DEATH. FOUND FLOATING. NO INQUEST.

BH 87/3

This group of overseas visitors were making a day trip to the Barrier Reef and were taken out to a pontoon moored there. It was suggested they could swim with snorkels off the pontoon, there being a supervised area for this purpose, with luncheon and a trip in a glass bottomed boat to follow. The victim asked one of those with her in the group to watch her belongings while she snorkeled and this was agreed. Although she was not seen to enter the water or with either mask or snorkel it is assumed that she had done as she had stated because a search of the boat and the pontoon later failed to find her, this search resulting when her failure to come to reclaim her belongings after half an hour began

PROVISIONAL REPORT AUSTRALIAN DIVING-RELATED FATALITIES 1987

CASE	AGE	TRAINED/EXPERIENCED VICTIM	BUDDY	DIVE GROUP	DIVE BASE	DIVE PURPOSE	WATER DEPTH M (FEET)	INCIDENT DEPTH M (FEET)
BH/1	74	Not stated	Not applicable	Solo	Beach	Recreation	Not Stated	Surface
BH/2	48	Inexperienced	Not applicable	Crowd	Boat Solo	Recreation	Not Stated	Surface?
BH/3	57	Not stated	Not applicable	Crowd Solo	Boat	Recreation	Not Stated	Surface?
BH/4	25	Trained Experienced	Experienced	Group Solo	Boat	Spear-fishing Competition	15 (50)	Not Stated
SC/1	34	Trained Inexperienced	Trained Some Experience	Trio Separation Pair	Boat	Recreation	9 (30)	Surface?
SC/2	47	Trained Inexperienced	Trained Inexperienced	Four Separation	Boat	Recreation	21 (70)	Ascending
SC/3	47	Trained Experienced	Not applicable	Solo	Boat	Scallops	13 (43)	Not Stated
SC/4	31	Trained Experienced	Trained	Group Separation	Boat	Recreation	33 (100)	33 (100)

to worry and annoy the person in whose custody they were. The body was never found.

SOLO SWIMMER CALM WATER. POSSIBLY HAD SNORKEL. NO FINS. NOT NOTICED IN GROUP OF SWIMMERS. SILENT DEATH. BODY NEVER RECOVERED. NO INQUEST.

BH 87/4

During an inter-club spearfishing competition the members of one club's team were in two boats anchored about 50 m apart as the divers hunted separately. All appeared to be normal until the comment was made by a child that one of the orange surface marker buoys had not moved for a long time. Until then it had been taken by the divers to be an unused one floating free. The float was now recognised as belonging to the victim. When its line was drawn up the victim's loaded speargun was still attached. After the divers had searched for about 10 minutes they found him on the sea floor in 50 feet deep water and brought him to the surface and attempts were made to resuscitate him but there was no response. About one hour had passed since he had last been seen at the surface.

Although death was due to drowning it was found there had been a small subarachnoid haemorrhage and it was thought this was the reason why this very experienced spearfisherman drowned. There was no history of ill health. Naturally the possibility has to be considered that this was a post-hyperventilation diving situation in which the subarachnoid leak was an additional adverse factor.

SEPARATION/SOLO. SPEARFISHING COMPETITION. VERY EXPERIENCED. ABSENCE NOT NOTICED TILL CHILD'S COMMENT. SURFACE MARKER BUOY WITH LINE TO SPEARGUN NOT DIVER. NO SURFACE COVER. NO BUOYANCY VEST. WEIGHT BELT NOT DITCHED. SUBARACHNOID HAEMORRHAGE THEN DROWNED. NO INQUEST.

PROVISIONAL REPORT AUSTRALIAN DIVING-RELATED FATALITIES 1987

BELT ON?	WEIGHT	CONTENTS GAUGE	VEST	REMAINING AIR	EQUIPMENT CHECK	EQUIPMENT OWNER	WET SUIT	SIGNIFICANT FACTORS
No	Not Applicable	Not Applicable	No	Not Applicable	Not Applicable	Own	No	Heart attack. Calm sea.
No	Not Applicable	Not Applicable	No	Not Applicable	Not Applicable	Not Stated	No	Excess food and drink before swim. Day trip to reef
No	Not Applicable	Not Applicable	No	Not Applicable	Not Applicable	Own?	No	Day trip to reef. Solo swim. Body never recovered.
Yes	Not Stated	Not Applicable	No	Not Applicable	Not Applicable	Own	Yes	Spearfishing competition. Subarachnoid haemorrhage. Post hyperventilation, blackout? Drowned.
On	Not Stated	Yes	Inflation by buddy	Low/nil	Satisfactory	Own	Yes	Failed to follow diveplan. Unrecognised predive illness. Myocarditis.
On	Not Stated Inflated	Yes	Not	Nil	Satisfactory	Own	Yes	Open-heart surgery as child. Solo out-of-air ascent. Then sank. Post mortem. Haemothorax. Torn lung.
On	Not Stated	Yes	Not Inflated	Not Stated	Satisfactory	Own	Yes	Shark attack. Body never recovered.
On	Not Stated	Yes	Partly Inflated	Low	Satisfactory	Own	Yes	Sea cave. Water surge. Separation. Found drowned. No head injury.

SC 87/1

The dive shop owner, an instructor, agreed that he would be willing to take two brothers out in his boat to scuba dive in the afternoon because they wished to spearfish in the morning when he was taking other divers out. The instructor decided that he would join them, making a trio of divers, and after reaching their chosen dive site, a rocky islet, he outlined the dive plan he proposed for them to follow. However when they were still only about 20 metres from the anchor they heard it bumping over the rocky sea bed. The dive leader, the instructor, indicated to them to remain where they were while he returned to reposition the anchor but on his return they were not there. He made a quick search underwater and of the surface, then repeated this without success despite swimming along the length of the proposed dive. Realising that to make a surface search using the dive boat might result in problems if the divers returned during his absence and panicked on finding no boat there so he resolved to remain in the boat till they returned.

After about 1 hour had passed he was starting to consider what action he should now take, then saw coming round the northern tip of the islet what looked like two divers so he drove the dive boat there to pick them up. He found the objects were the ditched backpacks, then observed one of the missing divers signalling from the rocks. He learned that the other diver had become unconscious and had been pulled up onto the rocks. As it was not possible for the boat to come safely into the rocks and take the victim aboard he made a rapid return to land to obtain assistance, then returned to the islet and swam ashore with a rope. Resuscitation (EAR) had been started by the victim's buddy and was continued during their return journey, but without success. The instructor was criticised later for his failure to radio for assistance and in rebuttal was able to show that his action resulted in the most rapid rescue of the victim and any of the emergency services would have taken far longer to reach the islet and recover the victim. Suggestions on incident management made by persons who were not present often show a lack of appreciation of the problem and a tendency to believe that by-the-book must be the only correct response.

The victim's buddy described how they had continued their dive around the islet until aware that their air was down to 50%, at which time he had surfaced to check their position. As the boat was not in sight he descended to rejoin his companion. He decided to continue swimming in the same direction around the rocky islet rather than retracing their course. He offered no reason why they had not waited the return of the instructor, their dive leader, nor why they failed to follow the agreed dive plan, which was that the divers would remain south of the islet. They continued underwater until the victim became low on air, the buddy then sharing his air with him using his octopus regulator. The victim had been quite unaware that they were out of sight of the boat until he surfaced and seemed to panic when he realised this fact and failed to obey a suggestion to inflate his buoyancy vest and appeared to be both slow and inefficient in starting to use his snorkel, inhaling some water. The buddy inflated his vest, calmed him down, and got him to resume use of his regulator as his tank still contained some air. The buddy started to tow him, hopeful of rounding the northern tip of the islet and seeing the boat, but had to hurriedly change this plan when the victim became unconscious and the regulator dropped from his mouth.

Faced with this crisis the buddy decided it was essential to get the victim out of the water and attempt EAR resuscitation. He ditched both his and the victim's backpack and weight belt (he realised their wet suits gave buoyancy) and it was these floating backpacks which were seen by the dive leader. This made him more agile, better able to get the victim up onto the rocks despite the one metre swell breaking on the islet. He managed to avoid the waves washing him back into the sea and commenced EAR resuscitation. It should be noted that he was forced to sacrifice the buoyancy vest at the same time as the tank as they were a single backpack unit.

The heroic efforts made by the buddy were unsuccessful as there was a factor he could not control. He had noticed when they had been snorkelling that morning that his companion became tired when they had been swimming for only 5 minutes and had assumed this to be an effect of the victim's obesity and general poor fitness, but the pathologist found changes in his heart which showed him to be suffering from myocarditis and this would have caused him to have a greatly reduced exercise tolerance. A viral cause was believed to be probable. The stress situation after surfacing had resulted in his suffering an acute cardiac failure.

SEPARATION OF TRIO GROUP AFTER ONE LEFT TO SECURE THE ANCHOR. FAILED TO FOLLOW AGREED DIVE PLAN. FAILED TO RECOGNISE EXTENT OF ATTEMPTED DIVE. CONTINUED SWIMMING AWAY FROM DIVEBOAT WHEN ONLY 50% AIR REMAINING. VALUE OF OCTOPUS REGULATOR. TANK ABLE TO PROVIDE AIR AFTER SURFACING. VALIANT BUDDY RESPONSE. DITCHED WEIGHT BELTS AND BACKPACKS SO ALSO UNAVOIDABLY DITCHED INFLATED BUOYANCY VESTS. WATER POWER PROBLEMS EXITING ONTO ROCKS. ACUTE MYOCARDITIS. ACUTE CARDIAC FAILURE.

SC 87/2

Although the four members of this family had successfully completed a scuba diving course about seven months before, and had dived during a one week holiday following this, the victim had not dived again since then while the others may have made a boat dive so obtained a little more experience. The dive boat was owned and run by the dive shop where they had been trained and there was on board in addition to this family group and the boat man, one other diver, but he dived solo and was not involved in the incident. The four divers were admittedly a little apprehensive and made errors during their kitting up for the dive. One of the children had ear equalisation problems and the victim had to borrow two additional weights, which were placed in the pocket of his buoyancy vest with the desired effect of reducing his buoyancy. Eventually they were all successful in reaching the sea bed, 20 metres depth.

After about 20 minutes the victim's wife saw she was down to 50 ats on her contents gauge, though the others still had twice that amount, and indicated that she was going to ascend and the others should remain till they had used up more air. After surfacing she sat in the boat talking to the boat man until the sudden surfacing of one of her children who cried out that her father was in trouble. The victim floated to the surface before any serious search could be organised. There was no response to resuscitation attempts.

Nobody was attempting to practice buddy diving procedures so when his wife ascended she assumed that he was remaining below and the two others assumed he would surface with her. When he was next seen he was slowly descending, making no attempt to clear his ears (which fact drew the attention of one of his children to the strangeness of the situation) but was seeming to be attempting to swim towards the surface. One of them took the two weights out of his buoyancy vest pocket, and when he still failed to ascent tried to ditch his weight belt but he then held onto the belt so firmly that it could not be removed. His eyes were starting, his face was blue, and his contents gauge was seen to read EMPTY. Very naturally they panicked and surfaced to seek help. The body floated up when he lost consciousness and the weight belt dropped from his hands. They were unable to inflate his vest because it was supplied from his tank, which was empty.

The most singular fact in this tragedy, beyond the act of the survivors seeking to blame the dive shop for allowing them to dive at this location (they were all trained, intelligent, and knew the rules for safe diving), was the autopsy. This was conducted by a forensic pathologist who carefully

followed the advised methods for a diving-related death but who evidently had no understanding of "diving pathology" and lacked awareness of the disasters which await an expert witness in court when subject to a rigorous cross examination and is found to have missed obvious findings. In this case the right pleural cavity was found to be obliterated by very dense fibrous adhesions, there was emphysema of the parietal pericardium with adhesions joining the parietal to visceral surfaces, a left sided haemothorax of about 1 litre, and a laceration (6 cm) in the base of the left lung. This was diagnosed as decompression sickness, hardly an intelligent finding.

The victim had been medically examined and also completed a medical history form before being accepted for training. He had failed to mention that 35 years previously he had been one of the first to have an operation to repair a "hole in the heart" and it is remarkable that the operation scars on his chest were noted by neither the doctor nor the pathologist. There is no evidence that he had ever had a chest X-ray taken. It is possible that this was not a medical history which should have precluded scuba diving. A point to note is that it was the left lung which tore and not the right (which was protected by the adhesions?). It is probable the lethal damage occurred as he was making a solo low air/out-of-air ascent. As he had a contents gauge there was no necessity for him to find himself in an out-of-air situation. A torn lung is a very unusual finding and there is nothing to suggest that air embolism occurred, death being the result of internal haemorrhage and shock with drowning as final factor. No inquest was thought necessary.

TRAINED. INEXPERIENCED. GROUP FOUR. ONE SOLO ASCENT THEN VICTIM SOLO ASCENT OUT-OF-AIR. HAD CONTENTS GAUGE. BUOYANCY VEST INOPERATIVE AS TANK EMPTY. REFUSED TO RELEASE GRIP ON WEIGHT BELT UNTIL UNCONSCIOUS. CHILDHOOD HEART OPERATION. LEFT HAEMOTHORAX AND LACERATION LUNG BASE. HIGHLY INACCURATE PATHOLOGY DIAGNOSIS. NO INQUEST.

SC 87/3

The victim was a careful and experienced diver who on this occasion was alone, diving for scallops from his anchored boat at a scallop bed often visited by local divers. At first his failure to return home at the expected time was thought to indicate that, due to tide or weather conditions, he had avoided such problems by returning to another harbour, but a check showed that this was not the case. When his radio was found to be unanswered a friend went out in his boat to investigate. The victim's boat was located but was empty. Searchers found a bag of scallops, the backpack with a damaged buoyancy vest, and a weight belt, but no trace of the body. The damage was consistent with that a shark would cause. Later it was reported that a fisherman some distance away had witnessed agitation of the surface at the probable time of the shark attack and possibly saw the fin of a shark.

SOLO EXPERIENCED SCUBA DIVER. SHARK ATTACK. BODY NEVER RECOVERED.

SC 87/4

The dive was to be at a rock which had a cave entrance to a passage which passed through it. There were three divers in one of the boats, one of whom had dived through the passage on several previous occasions, and four in the second, two of whom had brought underwater cameras with them. One person remained in each boat as a safety precaution. The group met at the cave entrance, which was at 30 metres depth, and the experienced diver offered to lead them through the passage. Only one diver actually followed him through though they had expected the others to come after them. There was some surge apparent in the cave entrance and for fear of damaging his camera one of the divers quickly retired to open water, though as he was adjusting his buoyancy there the camera washed from between his knees and he never recovered it. The second camera-carrying diver evidently penetrated further into the cave and the returning pair of divers found his body there on their return, lying on the floor of the cave with his regulator out of his mouth. There is nothing noted concerning the actions of the other three divers.

They dragged him out of the cave and were there joined by the diver who had been trying to find his lost camera. He ditched the victim's weight belt and backpack (which were retrieved at a later time) and assisted them bring the victim to the surface and to one of the boats. Their resuscitation efforts were unavailing. The mask was in position when the victim was found but one of his fins and that bootee were missing. His buoyancy vest was noted to contain some air and his tank contained some air though it became empty before it was formally checked later by the police.

Although some abrasions were present on his nose and both hands there was no evidence of any head injury. It is possible he was tossed about by a surge of water, lost his grip on the demand-valve mouthpiece, and drowned. There was a piece bitten out of the rubber of the regulator's mouthpiece, damage which apparently took place during the incident. The damage made it difficult to retain a grip on the regulator.

GROUP. SEPARATION/SOLO. SEA CAVE. POSSIBLY LOST REGULATOR FROM MOUTH WHEN EFFECTED BY WATER SURGE. VALIANT RESCUE EFFORTS BY OTHERS IN DIVE GROUP. BITTEN MOUTHPIECE. NO INQUEST.

Discussion

It is fortunately possible to discuss the four fatalities which involved snorkel divers because the police investigated the incidents and the statements they obtained were retained when the respective coroners decided that no inquest was necessary. In two instances the victims were members of tourist groups making a day trip to view The Reef and the fatalities occurred despite attempts to supervise people when they were in the water. These deaths are a warning to those running such trips of the ease with which some serious incident can occur. There are public relations reasons in such cases for a formal public examination of the circumstances, a consideration not necessarily apparent to coroners concerned with reducing delays by concentrating on cases requiring more detailed investigation of the facts.

There was no way in which the unexpected cardiac death of the victim in the first case could have been prevented because no intimation of his cardiac condition was apparent even to his wife and his death can fairly be described as happening when he was in the water rather than because he was snorkelling. Nevertheless the incident is a warning that however careful the planning, emergency situations can arise "out of the blue", and this man could just as easily have been in the launch or snorkelling with the other members of his group as being alone when he was taken ill. In any discussion of the remaining fatality in this group, which took place during a spearfishing competition, there is always the presumption that any such death is a consequence of pre-dive hyperventilation followed by a determined pursuit of a fish. The finding of evidence that a subarachnoid haemorrhage had occurred does not prove this was the cause of death. Both factors may have combined to disable him and the fact of him being alone and unsupervised added to the adverse factors influencing the outcome.

There were four scuba diving fatalities, in three of which the victim was alone at the critical time, although in the case of the shark attack this cannot have effected the outcome. There may have been a chance for survival in the cave death as had the victim been located immediately following the (presumed) loss of his regulator, or damage to the rubber mouthpiece which made it unusable, he could have been assisted out of the cave, if necessary utilising the "octopus" second regulator of one of the group. The risk in any sea cave of being helplessly tossed about and hitting the rock walls should be recognised by all who venture into these places.

Fatality reports frequently make adverse reference to the separation of divers, or their solo diving, as a significant factor influencing the course of the incident. Similarly inexperience is noted. In the second case both factors were present. Although the victim was trained he lacked experience. He carried from his past a possibly forgotten additional adverse factor, the residual scars of an open chest operation on his heart. It is evident that there was no inevitability of this scarring proving fatal as he managed the training diving without medical problems. Unfortunately there was, as events showed, a reduced safety margin when pulmonary over-pressure occurred during his ascent. Although an attempt was then made to assist him he was then suffering the effects of a serious lung tear and may also have had some cerebral arterial air emboli effecting his responses. The autopsy in this case was notable for failure to regard a torn lung and haemothorax as significant when reporting the cause of death.

In the first case there were breaches of the correct dive procedures but when the incident occurred the buddy showed he was fully competent in the management of a difficult situation he now faced. The instructor also showed an ability to respond in such a manner as seemed most appropriate in the situation rather than in the "text book" manner. Unfortunately, the medical condition which affected the victim was unsurvivable, but the buddy's response was one which would have saved him had this possibility existed. This death might have happened even had he not dived but his chances would have been improved if he had taken notice of the ill health which was apparent during his morning swim.

Acknowledgements

The production of this report would not be possible without the interest and support of the Departments of Attorney General/Law/Justice in every State. The willing assistance of the Police in cases where the Coroner has considered it not necessary to hold any Inquest is noted with appreciation. Thanks are also due to those in the Diving Organisations, as well as those who correspond directly, for their support.

PROJECT STICKYBEAK

The objective of this project is to collect reports on all types of diving-related misadventures which range from the fatal to those so well managed that there was no "incident" to report. Medial Confidentiality is at all times afforded such reports. This means that the reporting of asthma or diabetes, etc., will NOT result in the affected diver losing his or her diving certification. It is only through having accurate, adequate, and up-to-date information that diving can reach and maintain acceptable levels of safety. Reports are urgently required to enlarge the scope of the project.

Reports should be sent to:-

Dr Douglas WALKER,
P.O. Box 120,
NARRABEEN,
NEW SOUTH WALES 2101.

DECOMPRESSION TABLES, THEIR USE AND PROBLEMS

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Introduction

The decompression of divers, caisson workers, aviators and astronauts is occasionally complicated by dysbaric illnesses, including barotraumata, decompression sickness (DCS) and arterial gas embolism. To limit the occurrence of DCS, these decompressions are usually performed in accordance with a set of depth-time rules, a decompression table. It is hoped that by using these tables the rate of excretion of inert gases from the lungs will prevent gas bubbles from forming in tissues and in venous blood, and hence that DCS will be avoided. The frequent occurrence of DCS in all of these groups demonstrates that either available decompression tables cannot prevent DCS or that none of these groups can adequately comply with available tables. It is most likely that virtually any decompression can generate gas bubbles, that the response to such bubbles varies in an individual considerably from day to day and is a major determinant of the outcome of any decompression, and that conservative decompression practice only reduces the probability of DCS and can never totally prevent it. A risk-benefit approach to activities such as diving is obviously then the most appropriate one, and the concept of a "safe" decompression table is almost certainly naive.

The History of Decompression Tables

Although diving is an ancient occupation, the first recognised decompression table was only prepared for the British Admiralty in 1908.[1] This table was based on experiments performed on goats using an end point of symptomatic DCS. The significant probability of DCS associated with use of this original table for deep long dives was in part due to limitations with the experimental design, but largely due to the insensitivity of the chosen end point, clinical DCS. It would appear that bone, brain, and spinal cord damage can occur without overt focal symptoms.[23] The sensitivity of decompression testing has subsequently been increased by the use of ultrasonic bubble detection which has been able to detect mobile venous bubbles before symptoms of DCS emerge.[4] However, the Doppler apparatus used in these experiments only detects moving bubbles, cannot identify their source, and both the identification of bubbles and the determination of bubble frequencies is subjective. There is also an increasing belief that stationary tissue bubbles may be pivotal to the development of both tissue damage and dysfunction after diving. The simple problem is that the characteristics of the critical bubble whose formation has to be avoided in a decompression are yet to be described. Similarly, the pivotal role of complement protein activity in an animal model of DCS,[5] the cycling of blood vessels through open and closed phases,[6] the inability of single function exponential statements to describe gas kinetics,[7-9] and the extremely slow elimination of inert gases in comparison to their uptake,[10] all explain why existing "physiologically based" decompression tables, which do not take these phenomena into account, cannot describe actual events in a diver. The science of decompression table development has consequently become empirical, and since 1908, although they have been hidden behind physiological theorems, the modification of the original decompression table and the development of new tables has been a pragmatic exercise. It follows that testing of decompression tables in the field to a level of statistical significance should be the yardstick by which tables are measured and not the attractiveness of the underlying theory.

The original 1908 decompression table theory[1] incorporated the concepts that uptake and elimination of inert gases were mirror images of each other, that both of these processes were primarily influenced by the blood flow to a tissue and the solubility of the inert gas involved in that tissue, and that gas bubbles did not form in tissues until a critical super-saturation of tissue inert gas was reached. Almost certainly none of these assumptions are valid, but nevertheless, with the exception of some British decompression tables which were based on diffusion-limited uptake and a set of thermodynamic equilibrium tables,[6] similar assumptions are intrinsic to those tables being developed currently. What has been changed in these calculations is the number of tissues thought to be critical in the development of DCS, and the nature of the tolerable inert gas supersaturation.

Problems in Decompression Table Development

In two separate experiments,[11,12] gas bubble formation has been shown to significantly inhibit inert gas elimination. There are two immediate consequences of this observation. Firstly, the ideal decompression is that which creates the greatest possible gradient for inert gas elimination from a tissue without causing bubbles to form. Secondly, repetitive diving, multiples ascents within a single dive, and surface decompression procedures must be (and are) significant risk factors for DCS.[13] The fundamental problem in decompression table design is that the rules that govern a single dive and ascent are not applicable for circumstances when some tissue bubbles exist, as inert gas elimination will be slower and smaller decompressions will result in DCS. Surface decompression procedures (when a staged decompression is interrupted by decompression to the surface with subsequent recompression in a recompression chamber and then resumption of the original decompression, usually from a slightly greater depth than that from which the decompression was interrupted) in particular are thought, with some justification, to be "semi-controlled accidents".

Although considerable attention has been given, in both recent decompression table development and decompression-meter manufacture and marketing, to increasing allowable diving exposures by measuring the real depth-time exposure, rather than assuming the entire dive is spent at the maximum depth of the dive, it is the consequences of bubble formation that are critical to future decompression table design. In addition, this increased allowance for diving exposures by real depth-time monitoring must increase the frequency of DCS for any given decompression table.

A common practice now is to perform "safety" stops at 3 msw (10 fsw) even when the decompression table being used does not require any decompression stops. Such stops will have little benefit if bubbles have already formed, but should on their own do little harm. A more acceptable procedure is to perform those stops required of the first time interval for the greatest depth of the dive which does require a staged decompression to the surface. This is especially relevant for dives beyond 30 msw, where available risk data show that it is probably impossible to do a no-decompression dive (no decompression stages) and still have a subsequent probability of decompression sickness of less than one percent.[14]

The final difficulty in decompression table development is establishing the probability of decompression sickness associated for the individual depth-time formats and in each of the special procedures (repetitive diving, surface decompression). To do this with 95 percent confidence is practically impossible for all of the potential combinations, so a series of selected dives should be tested across the range of possible exposures to this level of significance. A sequential analysis[15] (35 dives without DCS needed before a DCS rate of less than 2 percent can be claimed) or a binomial distribution approach[16] (60 dives without DCS needed before a DCS rate of less than 5 percent can be claimed) are suitable techniques for such testing. With the possible exception of the Canadian Defence and Civil Institute of Environmental Medicine Tables,[17] none of the available decompression tables have been tested to this degree. For example, a common procedure has been to consider a table safe if 10 dives were performed for a particular depth-time combination without incident. In fact such an outcome only determines that there is a 35 percent chance that the associated probability of decompression sickness is less than 10 percent. In addition, it is essential that this testing be done in the ocean and involve real work, as both will significantly increase the rate of DCS in comparison to dives in recompression chambers of any sort or resting dives.[15,16,18,19]

Summary

It follows then, that although thee has been considerable experience in the use of decompression tables since 1908, that there has been little or no advance in real understanding. It also follows that until a new understanding is developed by the application of modern pharmaco-kinetic principles to gas-kinetic studies that decompression tables should be selected on the basis of their proven efficiency in avoiding DCS, and that procedures such as repetitive diving, multiple ascents within a single dive and surface decompressions should be avoided if at all possible.

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ARCHAEOLOGICAL DIVING IN AUSTRALIA A MEDICAL PERSPECTIVE

David Millar

Introduction

Why do students, a Qantas pilot, a customs agent and others want to spend two weeks training in archaeological diving? For some amateur (defined here as one who loves a field[1]) divers there is more than the lure of treasure hunting or the sport of wreck diving. In common with the underwater archaeologist, they share a fascination with the past and its reconstruction. The exploration of Australia has left our coast littered with hundreds of underwater time capsules. Until now, no formal training in scientific diving (archaeologist, biologist, oceanographer) has been available in Australia. Eight amateur divers and one professional underwater archaeologist recently undertook the first NAUI/CMAS Divemaster-Scientific Diver Training Course conducted by Sci Dive Australia in Far North Queensland from March 31st to April 15th, 1989.

This article will discuss aspects of such training and the role of the diving physician on such an expedition, which culminated with work on what is probably Australia's most important shipwreck, HMS Pandora.

Underwater archaeology is a relatively new discipline. Pioneering work in Australia commenced in 1969 with members of the Western Australian Maritime Museum inspecting, and later excavating the Dutch East Indiaman, Batavia, (wrecked on the Abrolhos Islands, West Australia, in June 1629)[2,3]. Artefacts raised from this vessel have included sufficient timbers to allow partial reconstruction of the vessel and many items of her extraordinary cargo. These have included a selection of silverware, stoneware jugs, smoking pipes, astrolabes, a pocket sundial and even a portico facade destined for the gateway to the company's castle in Batavia (now Jakarta, Indonesia).

Since then many thousands of archaeological dives have been undertaken on shipwreck sites around Australia, the Indian Ocean, south east Asia and the Pacific Ocean. Also many dives have occurred on wreck sites by sport divers and treasure hunting divers. Archaeological diving will be emphasised in this article.

As in other scientific diving disciplines (biology, oceanography, etc.) formal archaeological training is required for a systematic approach so that the maximum amount of information can be uncovered. The reconstruction of our past through maritime archaeology is a precise, time consuming discipline. Thus the 30 or so professional archaeologists now working around Australia have required hundreds of enthusiastic volunteers to help in their work. Lured by the romance of underwater archaeology, they are then faced with the reality of long arduous days in remote sites, diving at times in hazardous conditions, as shipwrecks are not noted for occurring off calm, balmy white beaches. On top of this there is new equipment and techniques to master. Despite this much valuable work has been done by amateur divers working with professional archaeologists with an excellent safety record.

In Western Australia over 10,000 archaeological dives have taken place[4] over the last 30 years at various sites along the coast. These include the wrecks of the Rapid, Batavia, Lively, Trial, Zuytdorp and Zeewijk to name a few. These sites are usually in less than 18 m, mostly in remote locations and many are exposed to surf (Lively, Trial and Sirius).

A medical officer has accompanied all major field trips in WA. So far no fatalities have occurred. No cases of decompression sickness (DCS) have been diagnosed. One case of cerebral arterial gas embolism (CAGE) has been successfully resuscitated, evacuated and rehabilitated. Three cases of severe marine stings, one stingray and two jelly fish, have occurred. While a number of minor illnesses and injuries have occurred, for example, a salt water aspiration syndrome like condition has been common in new divers to some sites, the safety record has been admirable.

The wreck of HMS Pandora, which sank in 1789, lies in 31-37 m of water on the outer Barrier Reef, at approximately 11°S 144°E. Surface conditions are relatively calm though the local reef configuration produces frequent and unpredictable changes in the ocean currents, even on the sea-bed. Water temperature is 14-16°C (average sea-bed), and

Figure 1. Divers surveying the HMS Sirius site under the surf on Norfolk Island. Photographer Pat Baker.

the visibility, 10-40 m. Expeditions to this site have been mounted on varying scales over four seasons. Considerable sections of the hull remain and the site is rich in artefacts. The lower sections of the vessel have settled largely intact into the sand, still holding the original contents. An early find was medical implements, including ampoules and syringes, from the ship surgeon's cabin, and what is believed to be his watch.

Table 1 shows the incidence of decompression sickness diagnosed to date against the number of dives on the Pandora site[5]. There were 4 cases of joint pain and one with cerebral symptoms and signs. All responded to surface oxygen, or recompression when a chamber was present, in 1983 and 1986. The combined incidence of DCS on the Pandora site then is 0.16% or 1.6 cases per 1,000 dives. No fatality, pulmonary barotrauma or other significant diving related injury has occurred. These figures are similar to those in a report being prepared by the author on six expedition seasons in the Gulf of Thailand diving to similar depths. The estimated risk of DCS using the US Navy tables at this depth (120 feet) is 1.8%6, placing these figures at an acceptable level for a working site. Other figures for scientific diving7[,8,9] also indicate very low mortality and morbidity rates.

While the efforts of various expedition leaders, divemasters and medical officers are commendable, we can further build on these beginnings. A rigid training program will further enhance the safety, productivity and enjoyment of both professional and amateur alike. Consideration of the scientific diving community as a special group is not new and a working group on scientific diving is attempting to set guidelines for training and operation[10]. The UNESCO Code of Practice for Scientific Diving is an extremely comprehensive document on which a draft standard could be based.

Sci Dive NAUI/CMAS Scientific Diver Course

The forerunner of future training programs was conducted recently by NAUI instructors, Jacques and Capkin Van Alphen and their team at Sci Dive Australia. This course was conducted over five intensive days on board the 25 m MV Kanimbla, in Barrier Reef waters while on route to the Pandora wreck site. Guided by three instructors and the author, nine trainees, who were all advanced divers, completed the NAUI Divemaster, Rescue Diver and Deep Diver courses. This prepared the trainees for the next phase, six days of survey work on the Pandora site under the supervision and instruction of Queensland Museum under-

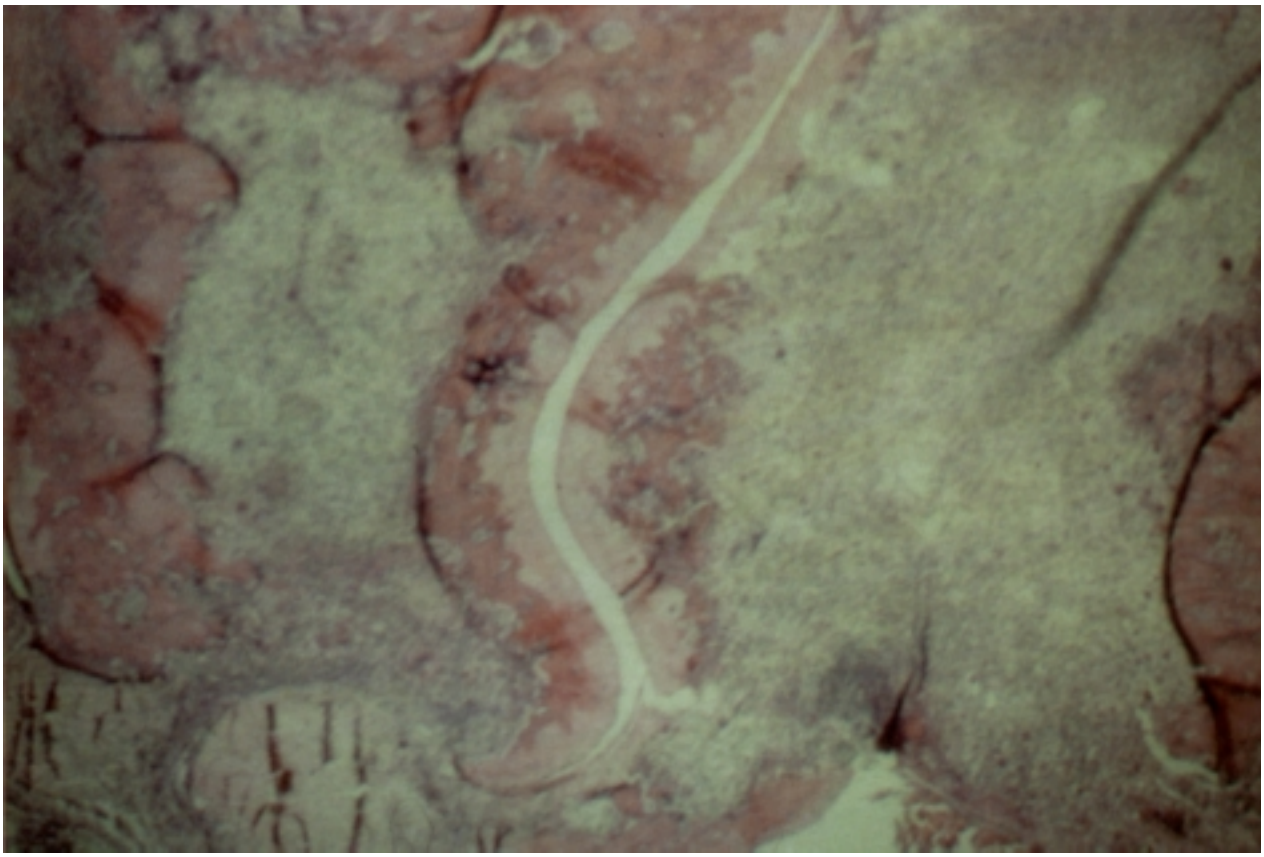

FIGURE 5. A typical haematoxalin and eosin stained section cut through the hind ankle joint of a control rat 28 days after adjuvant injection. **Both illustrations on this page are from the paper by Dr Fiona Andrews which starts on page 125**

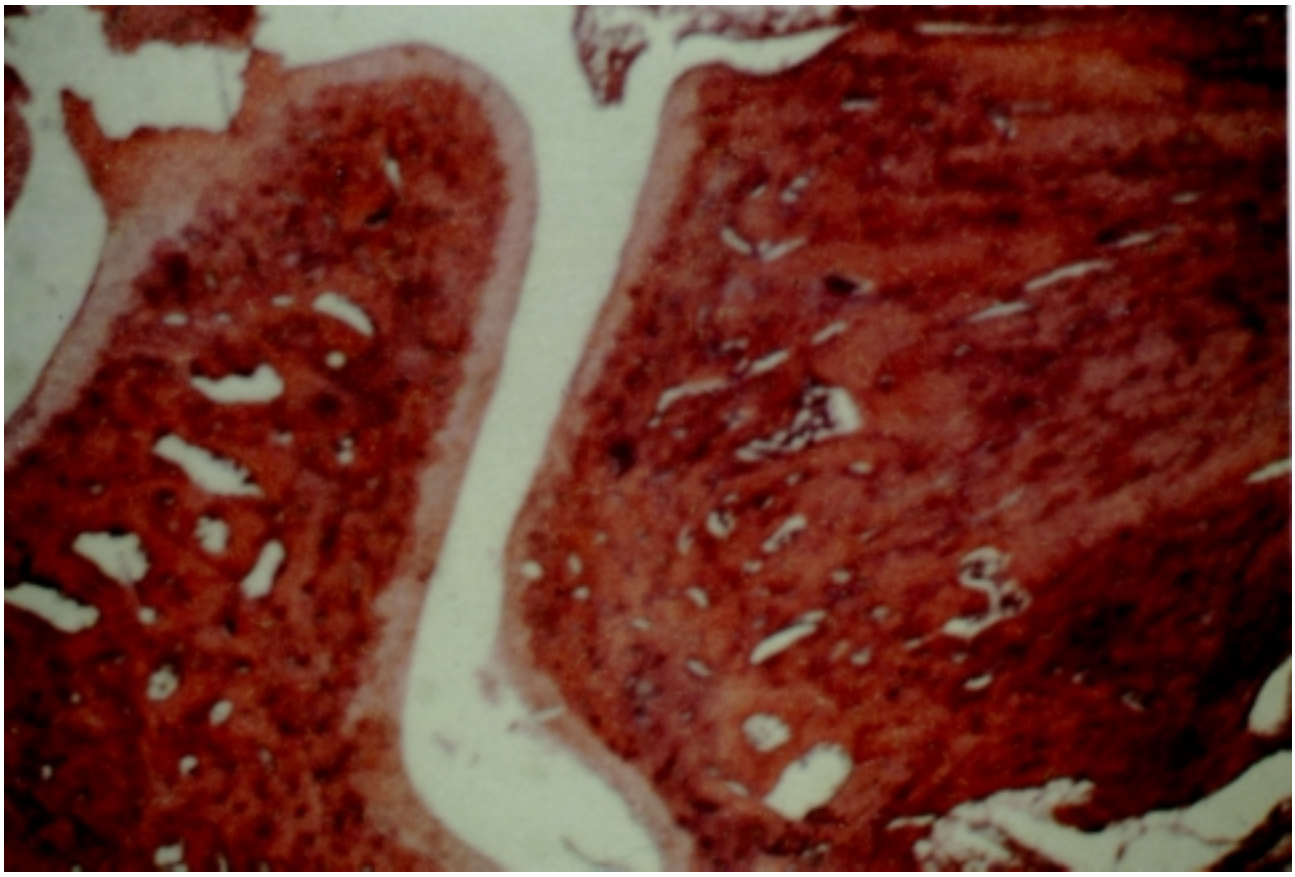

FIGURE 6. A typical iron deficient rat joint section 28 days after adjuvant injection.with very little inflammation.

TABLE 1

Season	Total Dives	Underwater Hours	Cases of Suspected DCS
1983	686	275.9	1
1984	800	364.94	2
1986	1435	568.5	2
1989	160	60.6	0
Total	12	3081.94	5

water archaeologist, Peter Gesner. All objectives of the fieldwork were completed without incident and in a very professional manner.

Scientific Diver Training

One aspect of this article is to examine the major components of such training in the light of this recent course.

DIVER SELECTION

Medical Fitness

All participants had been cleared by approved diving doctors, prior to departure. This is essential for a uniform standard.

Physical Fitness

Working with various equipment underwater and in the hazardous conditions that wreck sites often present, surf, currents, low visibility, requires an above average level of cardio-vascular (endurance) fitness. While this is generally accepted, no assessment of cardio-vascular fitness at the time of the medical or prior to an expedition occurs in most instances. Minimum levels have been suggested for sport divers[11] such as13 METS, which is equivalent to 12 minutes of the Bruce Protocol using a treadmill or bicycle ergometer. While such formal testing may not be practical or popular, a pre-set water fitness test could be established for participants to pass before being permitted to dive.

The Sci Dive course water skills test consisted of a 50 m underwater swim, with a maximum of 2 breaths allowed during the swim, a 400 m swim in 10 minutes, a 50 m tow of a "victim" and a 812 m swim in 18 minutes in scuba gear. All participants completed this test. Strict adherence and enforcement of a set standard of fitness is essential for continued safety. This was certainly so on the Pandora site, where heavy exertion at depth will greatly increase nitrogen uptake. Over exertion and resultant carbon dioxide build-up produced occipital headaches on two occasions, both rapidly responding to 100% oxygen over 5 minutes.

Psychological Fitness

Some degree of psychological stress is inevitable on an extended trip such as this and indeed is part of the selection process. The aim on an expedition is to minimize stresses wherever possible.

Adaptation to a new environment, equipment, and techniques are major stresses. These were well addressed on the course as Phase I allowed a gentle and progressive adaptation.

Long hours of theoretical and practical sessions followed by an examination on all aspects of these were other causes of stress. Well defined goals, a positive, caring attitude by the instructors and the trainees' enthusiasm, were key factors in minimising this. Structured free time is essential for extended field trips. The opportunity to simply sit and do nothing is a real luxury.

Food quality and quantity, sleeping arrangements etc. greatly influence attitude and group interaction. In this case a comfortable boat with good catering an a competent and enthusiastic captain and crew greatly contributed to group harmony. Fresh water showers backed up by an effective desalinator and calm safe anchorages at night, enhanced comfort and sleep.

The ability to have minor ailments promptly attended to minimise the stress of such physical ailments is essential in remote locations.

The combination of all of the above, together with open communication in the form of regular briefings and debriefings, achieved the goal of forming a cohesive working group.

EQUIPMENT

Progressive familiarisation with the diving and scientific equipment in Phase I, gave trainees ample opportunity to feel comfortable with their equipment. Of great concern was the marked discrepancy between some of the depth gauges used. There were variations of up to 10 m on

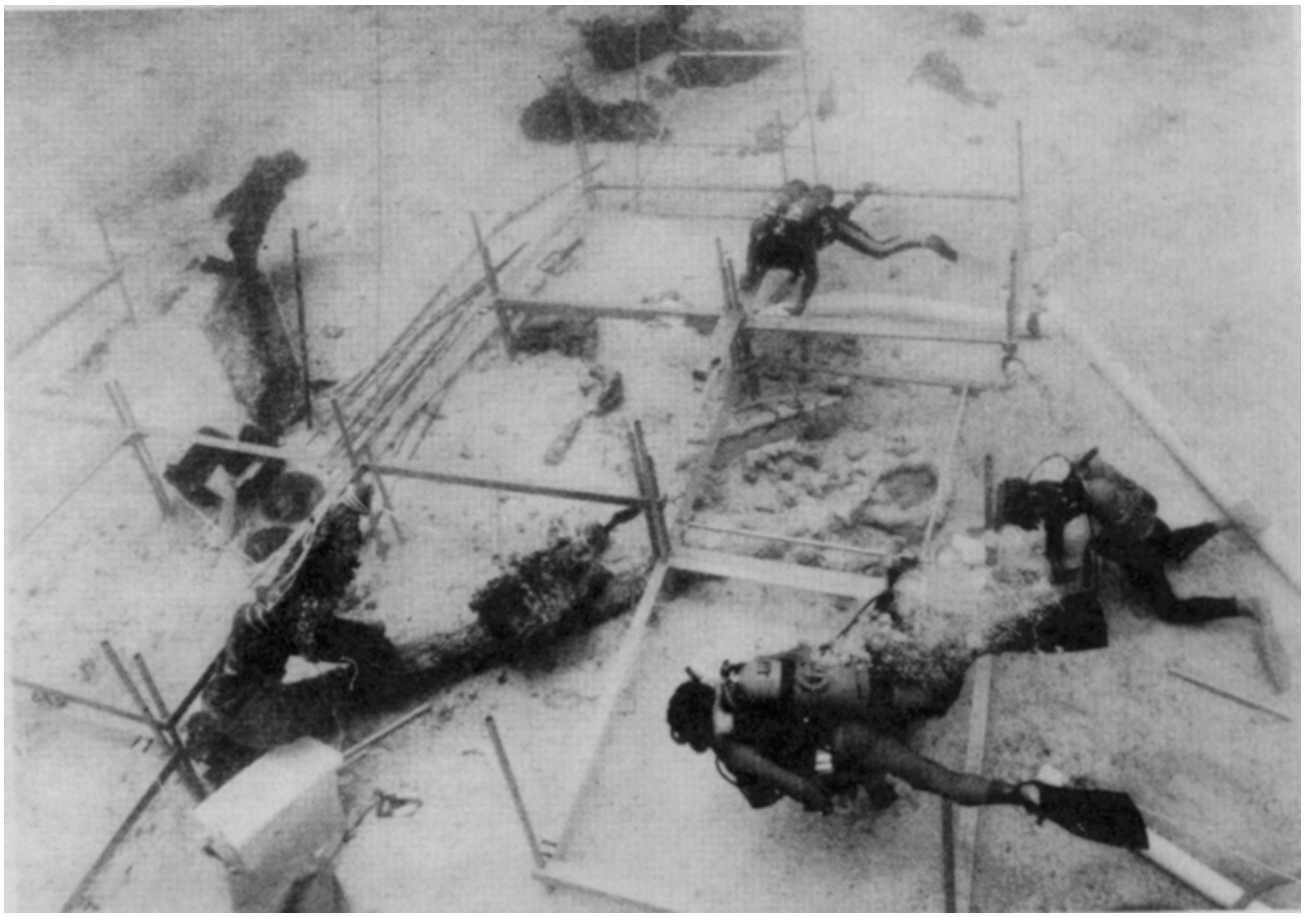

Figure 2. Divers working on the HMS Pandora site. Photographer Pat Baker.

the bottom at 30 m. On an ever changing deep site, such as this, constant accurate depth assessment is essential to diver safety. Completing set tasks, meant using either the 33 m or the 36 m limits where applicable. We wished to keep the safety stop time to not more than 5 minutes for diver comfort. We had sufficient accurate gauges for our work. A shore based depth gauge testing facility would certainly be useful for gauge checking and calibration prior to expeditions.

ENVIRONMENT

Transposing individuals from around the country, or worldwide, to a new diving environment is a stress which is often under estimated. In most of us, there is some fear of the unknown as each dive site is unique in respect of water temperature, sea conditions, underwater topography, marine life, etc. On this occasion, this stress was minimised by the slow gentle work-up to deep diving, taking 5 days to reach 36 m, giving ample opportunity for acclimitisation to the Pandora site. Nitrogen narcosis was watched for and was not a major problem. On one occasion, a team of divers did become fixed on the idea of completing a set task despite insufficient time. This emphasised the need for setting realistic, well rehearsed tasks and the allocation of a time keeper on the bottom for each dive.

TECHNIQUES

Diving Techniques

Some of these techniques, for example emergency procedures, are relevant to all scientific diving expeditions. Considerable refinement of the methods and training on advanced diving techniques has been achieved in recent years. Basic and advanced sport diving training in Australia, by the major organisations, (FAUI, NAUI, PADI) is of an exceptionally high and uniform standard. In this instance the NAUI Divemaster and Rescue Dive modules were well presented and completed by all trainees. Essential techniques covered were emergency ascents, rescues, first aid, treatment of marine injury and illness, and evacuation procedures. The reality of practicing rescues in a remote location was disturbing for some but a useful learning experience for all of us. Overlearning and problem solving are key aspects of this training.

This expedition used deep diving techniques, based on the NAUI training module. The United States Navy (USN) decompression tables were used as a basis for dive profiles with added safety factors consisting of:

1. Bottom times not exceeding the USN no-decompression limits (NDL).

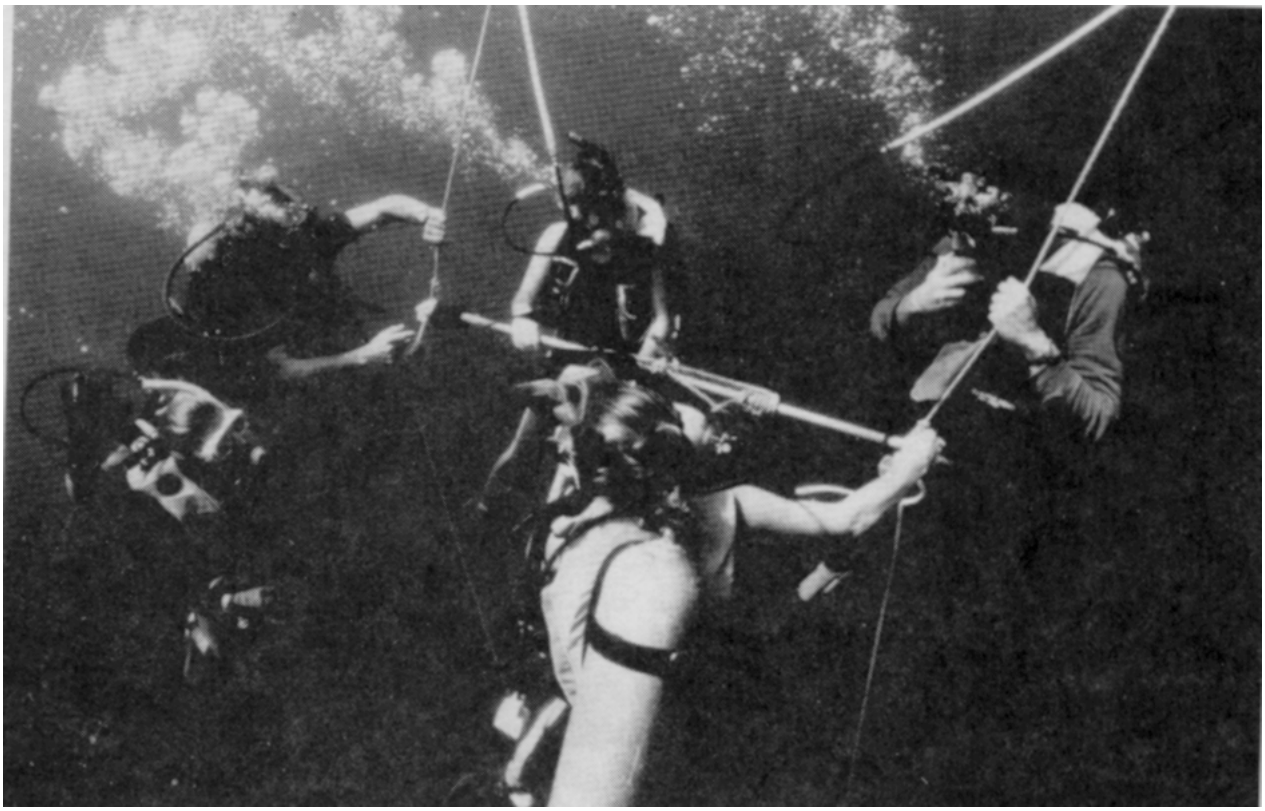

Figure 3. Divers on the decompression bar during a safety stop at the HMS Pandora site. Photgrapher Ian Hodson.

2. A slower ascent rate of 9 m/min, up a buoyed line attached to the stern anchor of the Pandora.
3. A compulsory safety stop at 3 m for 5 minutes on all dives. A heavily weighted decompression bar was slung from the midship section of a Zodiac inflatable which was attached to the stern anchor buoy. This proved quite stable. Each diver breathed 100% oxygen for 3 minutes of the 5 minute stop. This was delivered from an 'E' size cylinder (7 'E' size oxygen cylinders were carried), using Harris regulators with hookah hoses and demand valves. While this may be considered controversial by some, it must be emphasised that this procedure was used strictly to enhance nitrogen de-gassing. It is not being advocated for sports diving or for therapy. Provided that the bar depth is stable at 3 m, as in this case, and that divers on the stop are closely monitored, by the divers in the next team in this instant, using oxygen in this manner is a safe procedure.
4. The use of reduced bottom times when heavy work was expected e.g. strong currents.
5. Strict dive timing and close observation of the stop with extension of stop times when arrival to the decompression bar was late. This occurred on one occasion only.
6. Five hour surface intervals between repetitive dives and more conservative bottom times than allowed by the USN tables. I used an Aladin Pro Dive Computer to compare with our profiles. This unit uses the Buhlmann Tables for its basis and on this limited trial, matched our chosen profiles closely. These tables use shorter NDLs for rectangular dive profiles, a slower ascent rate 10 m/min, and compulsory stops. A dive to 36 m for 15 minutes using this table requires a 3 min stop at 3 m compared with our more conservative 5 mins[12]. The repetitive dive system of the Buhlmann tables includes a large safety margin to allow for the problem of reduced desaturation during the surface interval, caused by bubbles in the lungs. Further evaluation of this and other dive computers is warranted and is to be accompanied by Doppler ultrasound studies on future expeditions.
7. Adequate pre and post dive fluid intake was actively encouraged. The risk of DCS from diuresis and dehydration was thus minimised.

The 15 divers completed 160 dives on the Pandora with a combined dive time of 60 hours 36 minutes with no detectable decompression sickness. The safe exposure of the 9 trainees to working in one specific underwater environment was accomplished. Further experience in other situations, e.g. in shallow surf, poor visibility etc. will round off their training.

Scientific Diving Techniques

While some skills needed by different disciplines (biologist, oceanographer, archaeologist etc.) differ widely, others are similar. Aspects covered in this course were:

The use of basic scientific equipment such as tapes and ropes.

Underwater bottom and reef searches of the areas adjacent to the initial site of the grounding of the Pandora.
Setting up a grid reference system.
Close plot magnetometer survey to ascertain the extent of material beneath the substrate surface.
Surface communications using a hand-held signalling device.
Underwater photography.

All of the above were well handled by the group. Other skills such as sampling techniques could be added to broaden the course base.

The Role of the Diving Physician

On an expedition these duties primarily consist of general health care of all participants, diving clearance of divers, prevention and treatment of diving related problems, and involvement in lectures and practical sessions.

General Health Care

Prevention or early treatment of ailments is vital on an extended expedition and a comprehensive medical kit is essential to meet all contingencies. Problems encountered were minor cuts and abrasions, minor sprains and many upper respiratory tract infections on the last few days. These were not diving related and may have been influenced by the prolonged work schedule. Sea sickness occurred in 5 members of the team, mostly in the first 48 hours and responded well to the use of "Acubands", and/or "Scop" transdermal scopolamine patches. Minor skin rashes from heat, salt water and wetsuit, occurred towards the end and were minimised by adequate fresh water for showers. Three cases of neck or lower back pains responded well to acupuncture.

Diving Clearance of Divers

This consisted of pre-dive assessment initially and then daily with respect to specific dive profiles, underwater tasks and repetitive dive limitations. Working in harmony with the expedition leader and divemaster, as occurred on this occasion, the diving doctor can greatly enhance accident prevention.

Prevention and Treatment of Diving Related Problems

Regular ear checks were performed. All divers did ear toilet using "Aqua-ear" or a mixture of 95% methylated spirits to 5% vinegar. Three divers had problems early on with slow middle ear equalising, but no episodes of barotrauma occurred. The use of "Spraytish" nasal spray prior to steam inhalation in a hot shower at night is a potent remedy. No significant marine injury occurred on this trip.

The one significant incident was a case of decompression sickness that occurred on Day 4 of Phase I, two days before starting work on the Pandora. This occurred in a diver who was not subject to the course requirements, fitness assessment, acclimatising to deep dives etc., undertaken by all other divers.

Case Report

DH the deckhand of the vessel, asked to accompany two of our divers on a dive to free the vessel's anchor while at Martha Ridgeway Reef. The depth according to the depth sounder was 33 m. Aged 27, DH is a qualified divemaster with 500 previous dives. The dive profile was:

depth	37 m
bottom time	6 mins (NDL 10 mins)
slow ascent	6 mins
safety stop at 5 m for	3 mins

Within 5 minutes of surfacing, the 3 divers were breathing 100% oxygen on the surface for 6 minutes. The divers slowed their ascent because of the extra work involved in clearing the fouled anchor chain and the surface oxygen was added as a further precaution. Two hours after surfacing DH presented with left elbow pain, parathesiae and weakness of the left hand. He was anxious and hyperventilating. Examination revealed ill defined deep joint pain of the left elbow, and a glove and stocking distribution of reduced light touch sensation to the left forearm and hand. No actual weakness was demonstrated and full examination revealed no other abnormality.

He had three risk factors for DCS. He was more than 10 kg overweight. He smoked more than 20 cigarettes a day. He was short of sleep from being anchor watch the previous night. A diagnosis of DCS involving the left elbow with a possible spinal bend.

He was treated with 100% oxygen on the surface via a scuba demand valve using a cycle of 25 mins on oxygen followed by a 5 minute air break. He drank 500 ml immediately, then 1000 ml per hour.

Adequate re-hydration was demonstrated by a good urine output.

Reassurance and slowed diaphragmatic breathing while on oxygen was followed by complete resolution of the parathesiae and full return of sensation over 60 minutes. The elbow pain responded more slowly but after 2 1/2 hours of treatment, DH was asymptomatic apart from slight fatigue. He rested for 24 hours and has been well since. No diving was allowed for 5 days. On his return to diving more conservative dive profiles were adopted and no repetitive dives allowed.

DH was the only diver over whom full control was not exercised from the outset. Such control is essential to ensure safety. An on-board recompression facility is obviously desirable in remote locations.

Lectures and Practical Sessions

The presence of a competent diving doctor is undoubtedly reassuring on isolated work expeditions. The psychological benefits are enhanced by an active role in relevant theoretical and practical sessions.

Future Directions in Scientific Diving Training

The requirement for careful diver selection and training in scientific diving is established. Efforts by the Working Group on Scientific Diving and standards such as the UNESCO Code of Practice for Scientific Diving support this. Standardised training courses will further improve safety standards and working ability, with the training procedures themselves exerting a strong selective force on candidates. Apart from medical aspects the principal disqualifying factors on candidates on an intensive course such as that conducted by Sci Dive, are psychological and physical. Only through experiencing what it is like to work in the subsea environment at first hand can the potential scientific diver be ultimately and correctly selected. On this pioneering course, all of the candidates completed the theoretical and practical assessments to a high standard. From advanced divers they have progressed to a level that would make each of them a valuable asset to future scientific diving expeditions. Application has been made to CMAS in the real hope that appropriate recognition of their achievement can follow, i.e. the CMAS Scientific Diver Qualification. The course itself was a model from which others may develop.

As no program can cover all situations, it is important that all scientific divers maintain a detailed, validated, up-to-date log book. This should include data concerning the divers training and qualifications, dives and medical checks, details of equipment used, environments worked in and techniques performed. This would facilitate diver selection by expedition leaders and divemasters.

Australian divers are at the forefront in many areas of underwater scientific endeavour. With further education and acceptance of a valid set of standards, we can build on these beginnings. Future training courses, such as this one, will further promote diver safety and productivity. For the diving doctor, this arena provides a unique and rewarding opportunity to be actively involved in the field with many research possibilities.

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PAPERS FROM THE JOINT SPUMS AND ROYAL HOBART HOSPITAL MEETING, NOVEMBER 1988

OXYGEN PRODUCED FREE RADICALS

Christian Narkowicz

This is a quick resume of oxygen produced free radicals because what one really does in the chamber is expose people to high concentrations of oxygen. Oxygen is known to be toxic. At least some of this toxicity is due to the radicals formed which can include superoxide which will dismute to hydrogen peroxide. Hydrogen peroxide can be removed by disproportionation catalysed by catalase or through peroxidation with various peroxidases. However if all the hydrogen peroxide is not eliminated and there is Fe^{++} or Cu^{+} present a highly reactive hydroxyl radical is formed which is potentially very damaging. What we wanted to know was whether hyperbaric oxygen resulted in an increase of oxygen radicals, or free radicals, and whether the body was able to cope with increased free radical levels, whether we actually do damage to the patients that we are trying to treat.

We have been very lucky to get the assistance of the Tasmanian Police divers. They have given us great co-operation and without them we could not have done these studies. In all we put 10 Tasmanian police divers into our chamber. We gave them 3 consecutive 20 minute periods of breathing oxygen at 3 ATA and we took a venous blood sample after each of these periods, and a final sample back at the surface. We froze the blood samples in liquid nitrogen, and analysed them for free radical levels by ESR, which stands for electron spin resonance. The unpaired electron in a free radical has magnetic properties and one can exploit these magnetic properties to directly measure free radical levels.

To perform an ESR experiment we put the sample between the poles of a very strong magnet that polarises the unpaired electrons into a high energy level and a low energy level. We can induce transitions from the low to the high energy level by irradiating with microwaves at an appropriate frequency (figure 1) We irradiate with microwaves and sweep the magnetic field, and as we hit the resonance condition, where the difference in energy levels is equal to the energy of the microwaves, there one gets absorption. We plot the first derivative of the absorption curve versus increasing magnetic field (Figure 2). The second signal is due to copper in caeruloplasmin and if we were to extend this spectrum there would also be a signal from iron.

A typical trace from a normobaric venous sample has an absorption band, the height of which reflects the concentration of free radicals in the blood (Figure 2). After hyperbaric oxygen treatment there is a significant increase in the level of free radicals. In the post hyperbaric oxygen graph, the peak appears to be 3-4 times the size of the pre-hyperbaric oxygen. Can one make a quantitative statement about that and say there is 3-4 times as much free radical? One can. The integral of the signal is directly proportional to the free radical concentration, assuming identical running conditions for the instrument. The integral is actually proportional to the height of the signal, so in hyperbaric oxygen there were roughly 10 times the concentration of free radicals that were in the baseline data.

FIGURE 1

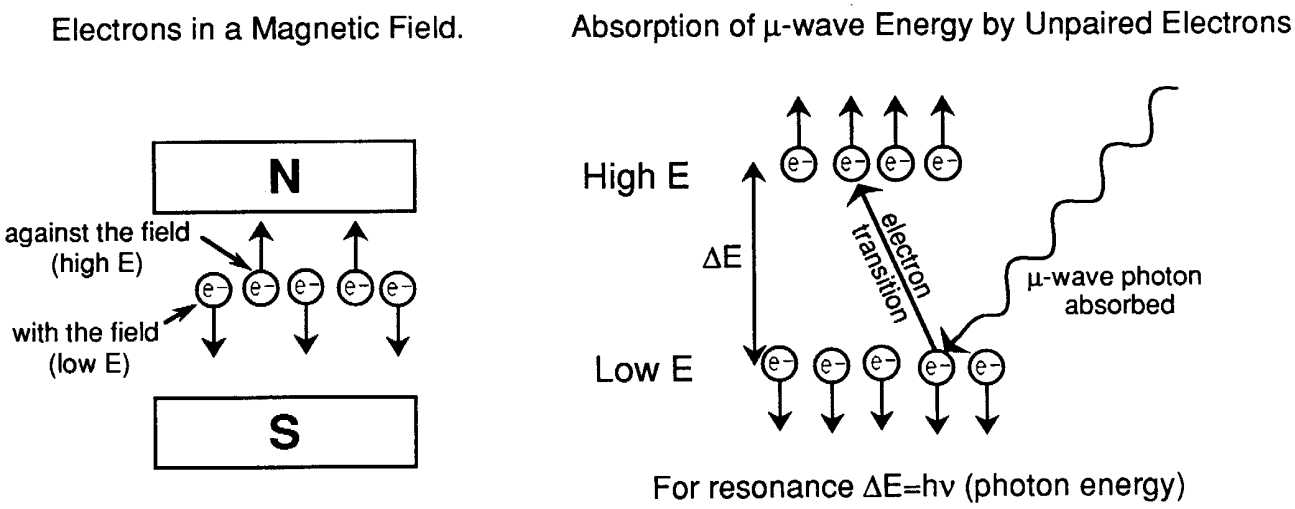


FIGURE 2

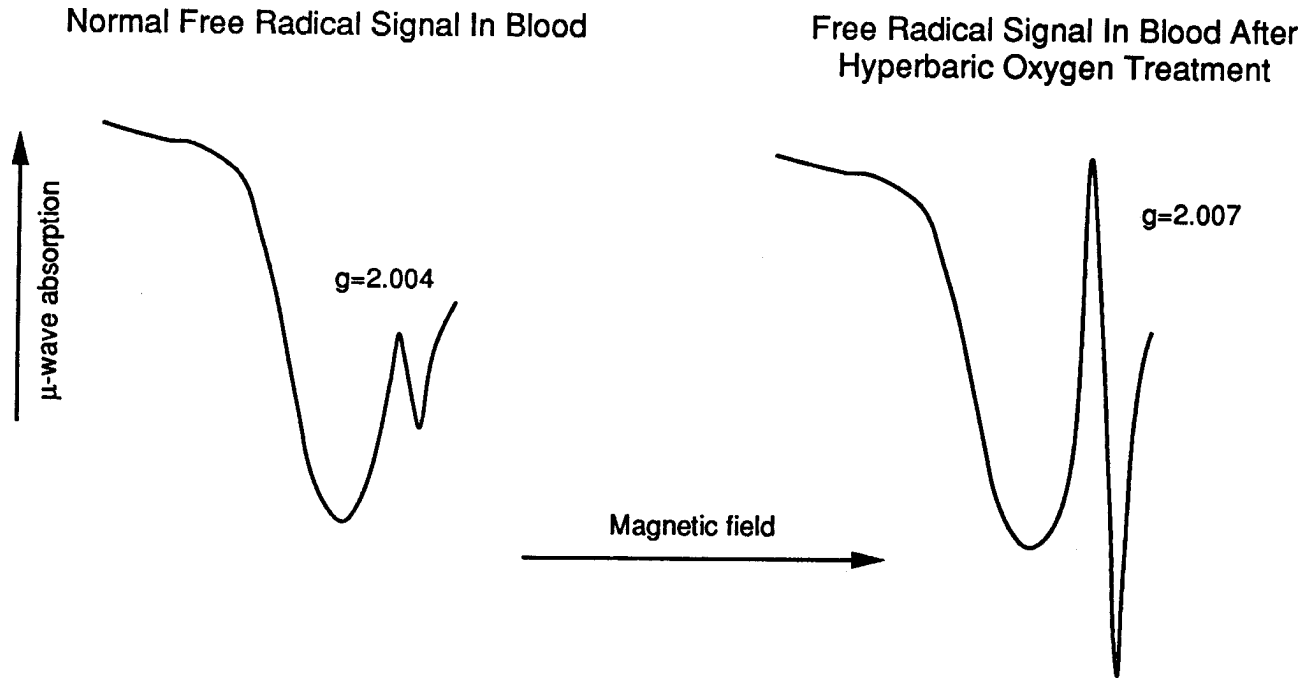


FIGURE 3

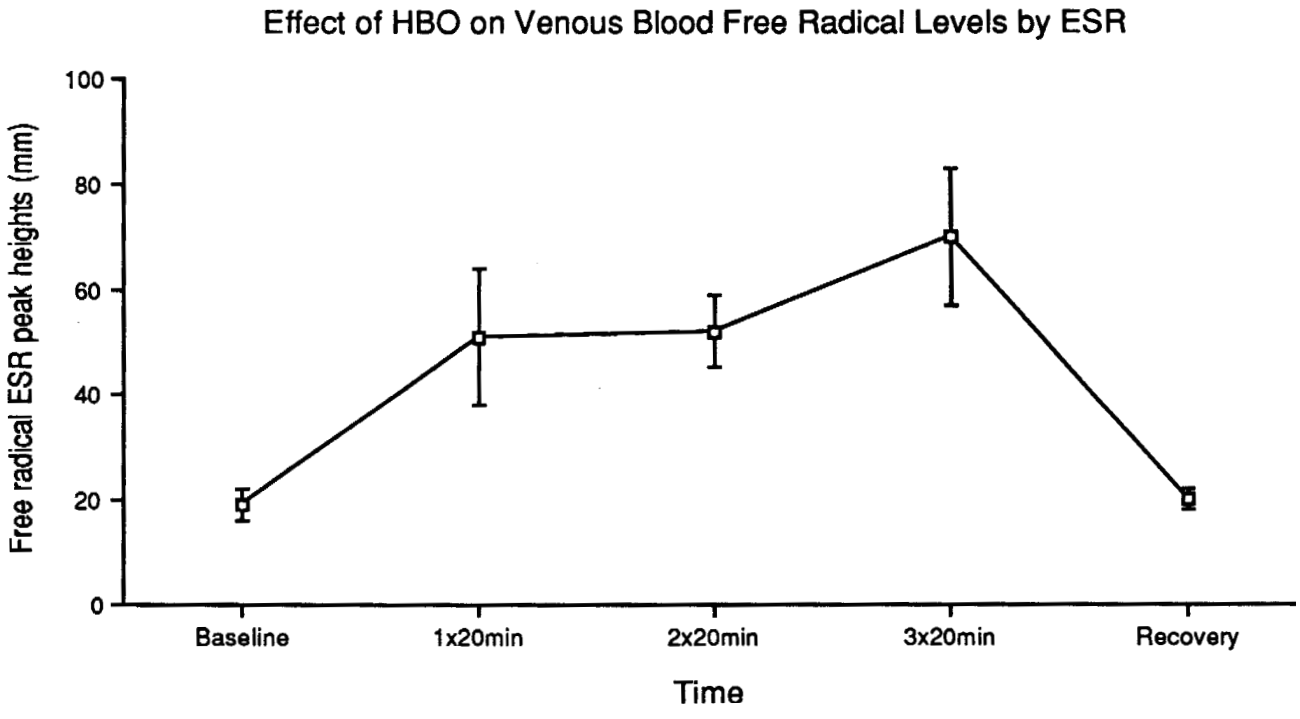
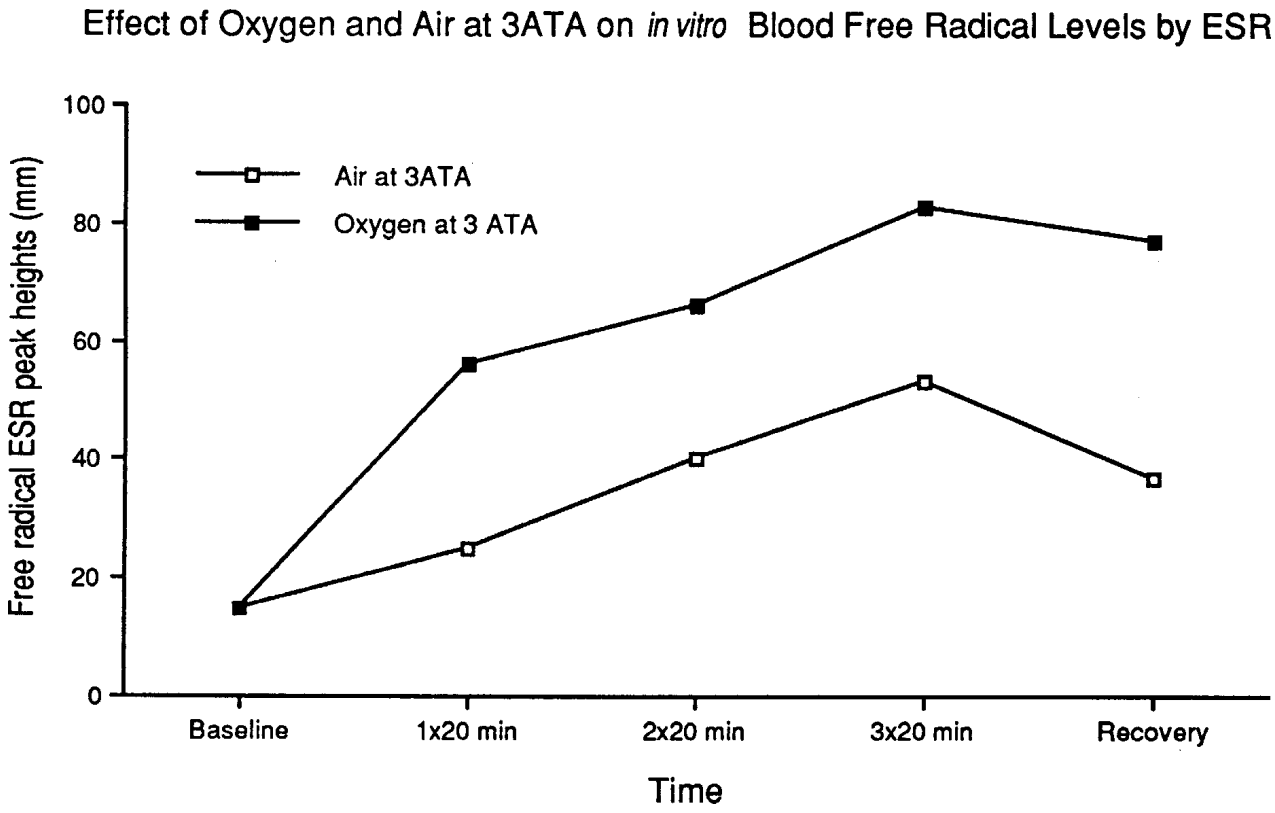


FIGURE 4



The average response in our 10 police volunteers after three 20 minute periods in oxygen is shown in Figure 3. On average it is around a 400% increase after 60 minutes, which is Dr Peter McCartney's normal protocol for hyperbaric oxygen therapy. Now to compare these results with an in vitro sample of blood bubbled with oxygen. If blood in vitro is bubbled with oxygen and blood in vitro bubbled with air, there is a similar sort of response (Figure 4), except one does not get the drop off at the end of the exposure which occurs in vivo because in vitro there is less oxygen consumption. So the oxygen levels remain high at the end of the run. There is some relationship between the concentration of oxygen and the degree of free radical production.

We found that the hyperbaric signal was different in form to the signal in ordinary, normobaric blood. By increasing the power of the microwaves used to obtain a spectrum one gets a linear increase in the height of the signal, with an increase in the square root of power, until one reaches a time where one starts getting a decrease in population difference between the high energy and the low energy levels, which is referred to as power saturation. The actual strength of the signal depends on that difference in population levels. If the high energy electrons can not lose energy quickly enough to make up for the energy that is coming into the system, the population difference decreases and there is a drop off in signal. We found that the hyperbaric signal dropped off a lot earlier than the normobaric signal indicating that it is a different free radical involved to the the free radical in normal blood. For free radicals it could be that several radicals contribute to each signal. The baseline free radical signal is the signal that appears under normobaric conditions and with an increase in oxygen load that signal does increase to some extent but it is a second signal which increases to the greatest extent and that tends to drop off to virtually non significant levels when breathing air after the hyperbaric treatment.

From further experiments we believe that the baseline signal may be due to semi-reduced glutathione reductase which is associated with the pentose phosphate pathway which is responsible for maintaining cells in the reduced form, for overcoming oxidative stress in cells. The baseline signal comes from the red cells. We are not sure exactly where the second signal comes from. With increasing oxygen concentrations one would expect to get increased oxidation of hemoglobin to methemoglobin with loss of superoxide, and the superoxide then has to be scavenged which requires energy for the system to be reduced back to its reduced state. That is where the pentose phosphate pathway comes in by providing the substrates for maintaining the reduced state of the cell. The second signal we have not been able to assign to a particular radical as yet but it could be due to a scavenger or perhaps associated with lipid peroxidation, though the power saturation is not consistent with being an oxygen radical nor an alkoxyl radical nor a lipid peroxyl radical.

At this stage all we can say is that exposure to limited periods of hyperbaric oxygen does cause an increased level of free radicals in blood. The healthy human appears to clear this increase rapidly on re-exposure to room air. But as yet

we have not assessed any possible damage, such as lipid peroxidation in the blood, by other chemical means. There is certainly room for further experiments.

Edited comments from the question and answer session appear below

Dr Ian Unsworth

This is fascinating to me. If there is a rise of radicals during a period of high pressure oxygen it would seem to suggest that the body's normal mechanisms for dealing with radicals have been overcome, and so the radicals are allowed to build up. But it also suggests that when one brings the subject back to one atmosphere radicals very quickly stop forming. Presumably this is due either to automatic dissipation of the radicals or to an extremely quick build up of the body's scavenging. Have we any idea which it might be? As the radicals fall so quickly one can hypothesise that the scavengers which have been knocked out under pressure regenerate very quickly.

C.Narkowicz

The pentose phosphate pathway will provide substrates for regeneration of the antioxidants of the free radical scavengers. The signal that we see could actually be a free radical scavenger because they exist as a free radical once they have scavenged the radical. They take the free radical character from the scavenged radical and become radicals themselves, so it could be something like vitamin E which is scavenging and as yet has not been reduced via the ascorbate pathway and NADPH (nicotinamide-adenine dinucleotide phosphate [reduced form]) back to the reduced form. It will take further experiments, perhaps with our own spin trapping agents which, so to speak, interact with the radicals as they are produced, and trap the radicals in a stable form before they can be reduced by other antioxidants. Spin traps can enable one to identify the actual structure of the radical being formed.

Dr P. Chapman-Smith

Policemen remain fairly docile and blood is easy to get at. Have you any comments on tissue free radicals? Obviously it is a lot harder to chip bits out of the local policeman.

C.Narkowicz

There has been a lot of work on radicals in tissue but not as far as I know concerned with hyperbaric oxygen. They certainly found increased levels of radicals under certain pathological conditions and after exposure to various carcinogens and drugs. Various drugs increases the free radical levels in the tissue. As for our experiments I guess one would have to get a pig or a sheep into the chamber, or a rat.

Dr Janet Vial

There are some problems with looking at tissue samples with ESR. The problem that bedevils this area is artifacts, especially when you are using ESR to measure tissue radicals. Just cutting tissue in itself can generate radicals. One has to be very careful about the way the tissues are handled. It is obviously important that it be frozen immediately to lessen this problem of artifact radicals.

Dr David Davies

Have you pushed your times under pressure any further than 60 minutes? Do you plan to?

C.Narkowicz

We have not gone beyond 60 minutes. To go longer is up to Dr Peter McCartney, and up to the policemen too. Certainly treatments go beyond 60 minutes. For instance Dr Ian Unsworth is treating carbon monoxide poisoning with 90 minutes of 100% oxygen at 2.4 atmospheres. It would be interesting to see whether there was any further increase in free radicals at the end of that 90 minutes.

There is a possibility that there is a point at which one overwhelms the body's own defences and one will get a massive increase in the free radical levels, but that could be 60 minutes, 90 minutes, or it could be 2 hours. We really do not know. It will also depend on the individuals and their diet and fitness and age, etc. It is certainly something worth doing. It would be interesting to see what happens at different exposures.

Dr David Davies

Another thing to consider is doing something similar on saturation divers at pressure. They can be at 200 m pressure for 3 weeks.

C.Narkowicz

However they have their oxygen supply diminished proportionally. But it might show whether it is an effect of pressure or an effect of oxygen. It does seem to be an effect of oxygen rather than of pressure because if we use nitrogen in vitro there is not the increase that occurs with oxygen. Of course we can not use only nitrogen with our police divers, nor with abalone divers.

Divers, from my experience anyway, seem to age quickly. I do not know if this is backed up by statistics but they seem to have a lot of medical problems, arthritis, and premature aging. I do not know if it is their very hard lifestyle or drinking and smoking.

Dr Chris Lourey

Apart from the incidence of osteonecrosis the punch drunk diver abalone diver is no different from the punch drunk crayfisherman. I think it reflects their after aquatic lifestyle and probably their alcohol intake rather than their diving.

C.Narkowicz

There is certainly an increase in free radicals just with breathing air. So at 18 m on air one is also increasing the oxidative stress.

Dr Peter Chapman-Smith

Are there specific tissues that have been shown to be damaged by this release of free radicals?

C.Narkowicz

The eyes are very susceptible. The retrolental fibroplasia of premature infants is caused by oxygen. The red cells are very vulnerable simply because they carry a lot of the oxygen and actually produce a lot of the radicals. As for other tissues, brain and marrow and the lungs.

One of the problems is that most of the evidence so far is circumstantial. We can show experimentally that if one gives free radical protecting agents one can reduce the damage but most of the evidence is circumstantial because of the difficulty of actually measuring free radicals in these situations and of showing that there is an increase in free radicals associated with the damage.

It would be interesting to put someone with a 6-glucose phosphate dehydrogenase deficiency into the chamber and see what happens to them. They have a slower rate of free radical inactivation, with a decreased pentose phosphate metabolism. So they might be suceptible to oxidative stress a bit earlier than someone with a fully functional pentose phosphate pathway.

Question

Does microwaved food generate free radicals?

C.Narkowicz

Not that I know of. I do not think it should but food irradiation is known to produce to hydroxyl radical which is where a lot of the concern comes from because if those radicals are not scavenged by the antioxidants in the food they can affect the proteins, the vitamins, and really affect the quality of the food. Another thing is that ultrasound is known to produce hydroxyl radicals as well. So there is increasing concern over use of ultrasound, or the excessive use of ultrasound, and of course exposure to radiation. Most of the damage is by free radical mechanisms.

This is an edited transcript of a recording made at a Free Radical Workshop during the joint SPUMS and Royal Hobart Hospital meeting on Hyperbaric and Diving Medicine in November 1988.

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IRON, OXYGEN RADICALS AND THE JOINT

Fiona Andrews

Iron has been associated with joint inflammation for a number of years. As long ago as 1674 Hochsletter described an arthritis associated with excessive bleeding in haemophilia patients. This was reproduced in experimental animals by injecting blood into the joints. Later on it was also shown that patients with oral iron overload or haemochromatosis also had iron deposits within the synovial membrane, and this was associated with inflammation of an otherwise normal joint.

Interest in iron and joint inflammation was rekindled by the work of Muirden[1] who showed that iron deposition occurred in the synovial membranes of patients with rheumatoid arthritis. It was speculated that perhaps these iron deposits had some role in the pathogenesis of the disease. This leads us to the question, how does iron deposition arise in the rheumatoid joint?

In normal situations iron is carefully conserved and recycled within the body and very little is lost. But in chronic inflammatory conditions such as rheumatoid arthritis many patients become anaemic. This does not appear to be due to an increased loss of iron but to a sequestering of iron within the reticuloendothelial cells so the iron is not let out back into the recirculating pool. How does this relate to the joint? Ultrastructural studies have shown that the synovium consists of reticuloendothelial like cells and it was proposed that iron deposits, derived from periods of micro bleeding in the joint are sequestered in the reticuloendothelial-like cells of the synovium. In support of this Muirden[2] found that levels of iron within the synovium correlated with an increased activity of joint inflammation.

So how might iron enhance inflammation in the rheumatoid joints? One theory is that iron is involved in oxidative tissue damage. The environment of the inflamed rheumatoid joint is highly suited to the production of reactive oxygen metabolites such as superoxide, hydrogen peroxide and the hydroxyl radical. The major source of reactive oxygen metabolites are the infiltrating inflammatory phagocytic cells. Phagocytosis induces increased cellular aerobic activity which leads to the formation of relatively unreactive superoxide and water. In the presence of iron however hydroxyl radicals, the most toxic reactive oxygen metabolites can be produced. A further factor that leads to an ideal environment for reactive oxygen metabolite activity is an insufficient reactive oxygen metabolites scavenging ability which has also been demonstrated in the rheumatoid joint.

What evidence have we that reactive oxygen metabolite reactions are occurring in the joint? Several studies have demonstrated the presence of lipid peroxidation prod-

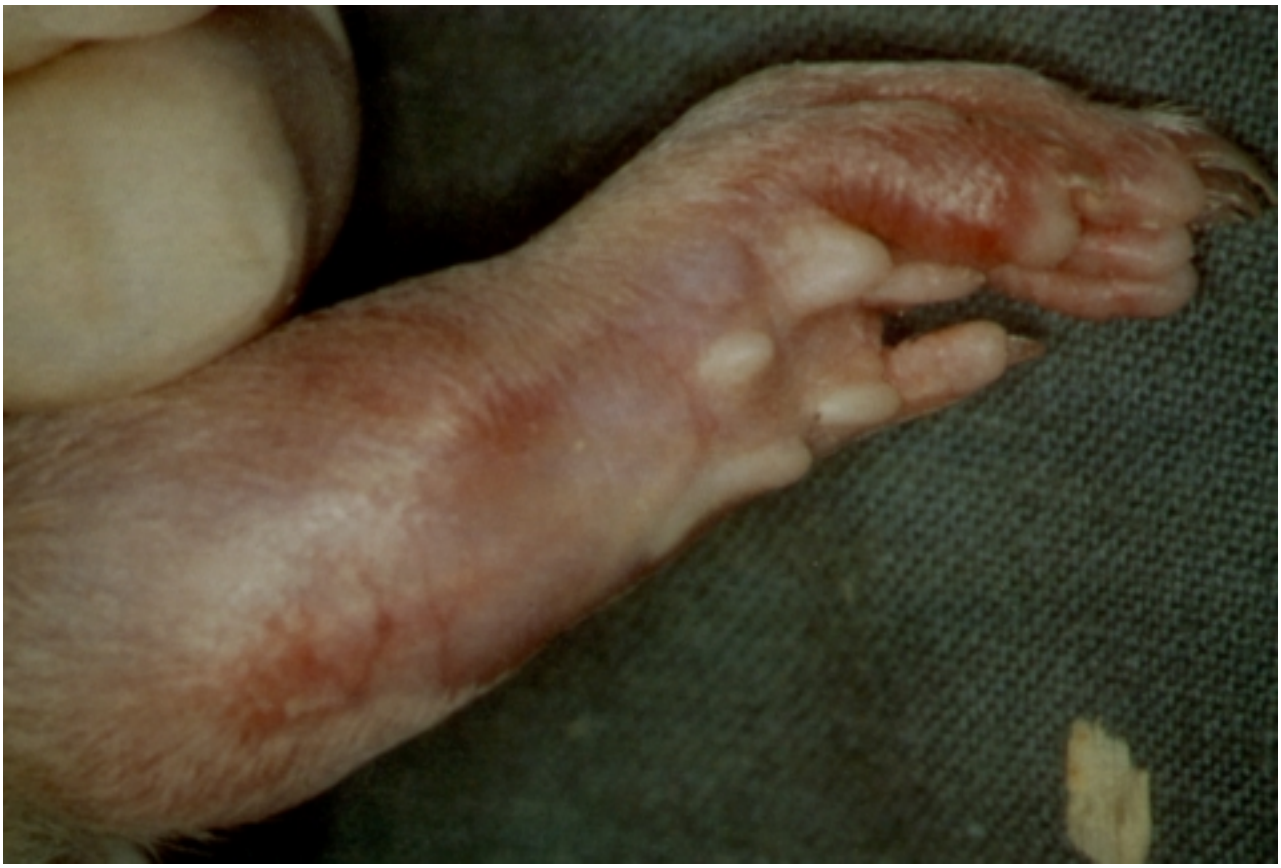

FIGURE 1. A typical arthritic ankle joint from a rat with normal iron levels, 28 days after adjuvant injection showing extensive inflammation.

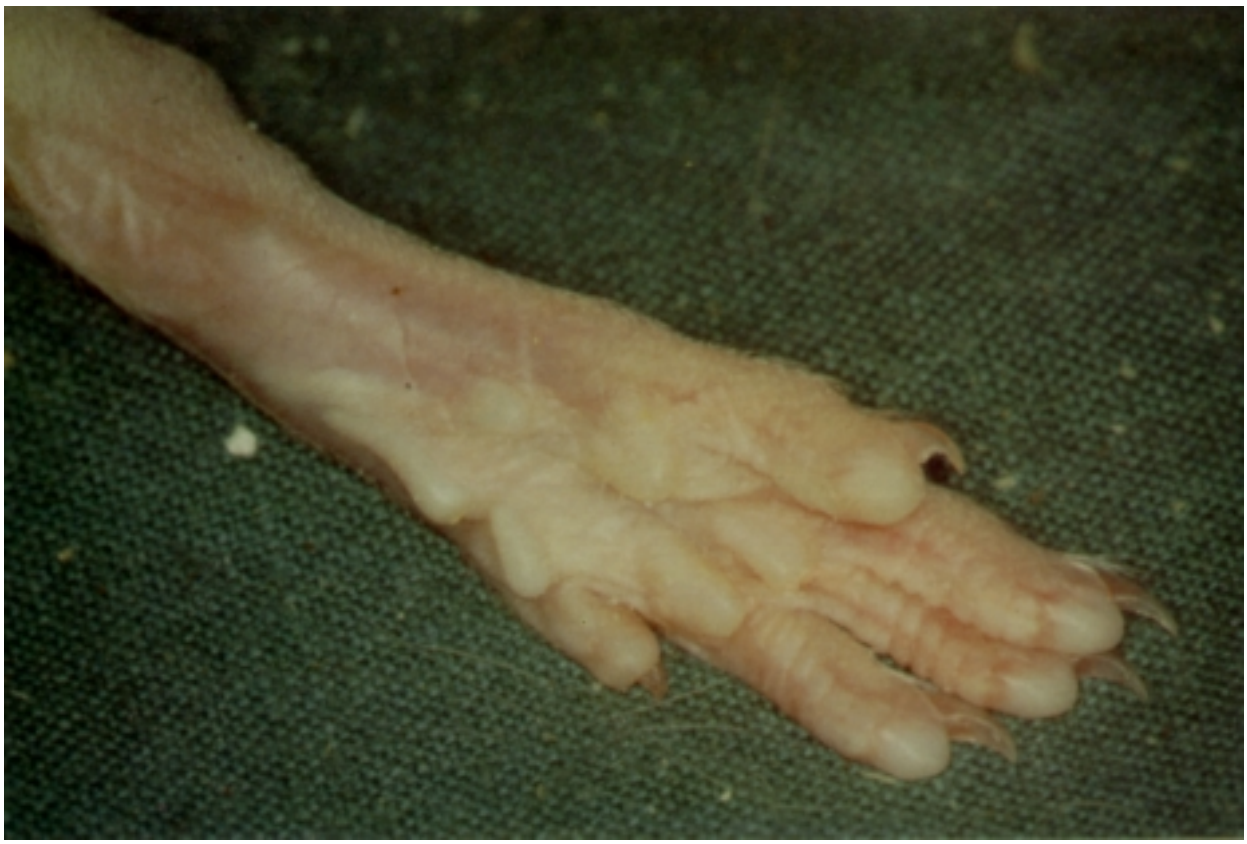

FIGURE 2.
A typical iron deficient rat ankle joint 28 days after adjuvant injection. There is only very mild inflammation.

ucts (products of the reaction of reactive oxygen metabolites and lipid) in rheumatoid joints. Firstly Lunec et al.[3] reported that peroxidation products occurred in 90% of rheumatoid synovial fluid samples tested. Wynyard et.al.[4] found that when rheumatoid patients were infused with iron dextran this led to an exacerbation of synovitis and an increase in lipid peroxidation, suggesting that iron enhanced inflammation via the formation of the hydroxyl radical and its subsequent reaction with cellular lipid.

Based on this evidence, we decided to study this concept further and to look at the possible anti-inflammatory effects of iron deficiency in an animal model of arthritis. The model we chose is adjuvant disease, which is a well used model for testing anti-arthritic drugs. It was first documented by Pearson in 1956[5] and has the advantage of being a well documented and reproducible model to use in the laboratory. The pathology of the joint differs from that of rheumatoid arthritis although one factor which is consistent is the presence of iron deposition within the synovium.

The protocol we used to produce our iron deficient rats was to feed male post weaning rats with an iron deficient diet containing 20 parts per million of iron. Control rats were fed the same diet, supplemented with ferrous sulphate. Using this level of iron deficiency in the test group there was no significant difference in weight gain compared with controls on the supplemented diet and hemoglobin levels were only mildly reduced, although iron stores were significantly reduced. Interestingly the levels of other metals such as copper were not affected by the level of iron deficiency in this experiment.

Having produced iron deficiency we then went on to induce adjuvant disease. This was done by injecting mycobacterium butyricum intradermally into the tail. Twelve days later rats developed the characteristic components of adjuvant disease; firstly local inflammation at the site of the injection and then joint symptoms and a systemic response to the adjuvant. I will just concentrate on the joint symptoms. Suffice to say that iron deficiency had no effect on local or systemic responses.

Iron deficiency did have profound effect on the joint symptoms. Figure 1 (page126) shows a typical arthritic ankle joint from a normal rat with normal iron levels, 28 days post adjuvant injection showing extensive inflammation. Figure 2 (page126) however shows a typical iron deficient rat ankle joint also 28 days post adjuvant injection where one can see only a very mild inflammation. We assessed joint inflammation in three ways. Firstly by visually scoring redness and swelling from 0-4 for each hind ankle joint, with a maximum score of 8 per rat and the results can be seen in Figure 3 (page128). At the beginning of the experiment there was a high percentage of animals with a score of 0, as one would expect. As the arthritis progressed in the control group more animals had a higher score. However in the iron deficient group we demonstrated the anti inflammatory effects of iron deficiency by the significantly higher numbers of rats that had a score of 0 from day 16 to the end of the experiment.

We also assessed joint inflammation by scoring typical histological features of arthritis; inflammatory cell infiltration, synovitis, fibrin deposition and bone erosion, new bone formation and repair of the joint tissue. Again these were scored from 0-4 on sections cut through the right ankle joint of each rat. We obtained similar results to the gross scoring with a significant reduction in histology score in the iron deficient rats towards the end of the experiment (Figure 4). Figure 5 (page115) shows a typical haematoxalin and eosin stained section cut through the hind ankle joint of a control rat 28 days post adjuvant. The joint is almost totally destroyed. The joint space and a large proportion of the bone has been replaced by inflammatory cell infiltration. Thus bone has been eroded away and eventually the joint will collapse.

Figure 6 (page115) shows a typical iron deficient rat joint section at the same time point with very little inflammation. All the iron deficient rats did show some histological signs of inflammation, but at the most it was a mild synovitis.

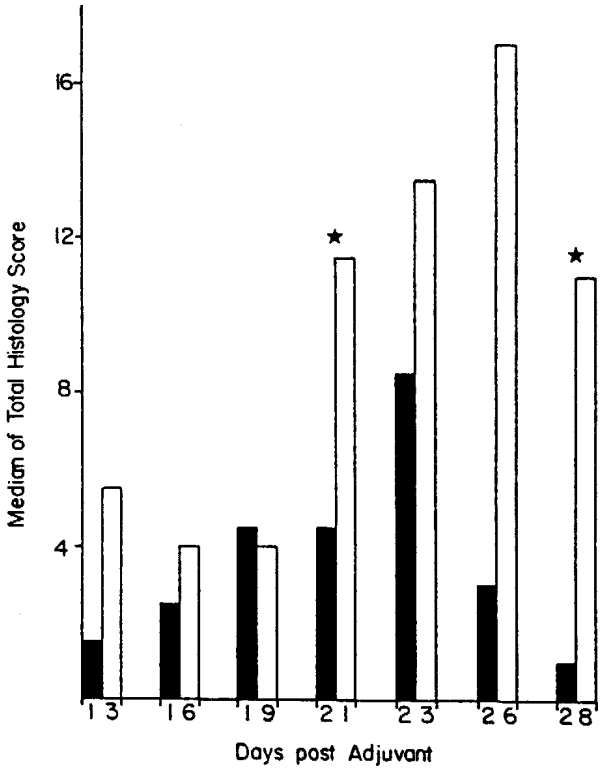

FIGURE 4. A comparison of the medial total histology scores during the course of adjuvant diseas in iron deficient and iron supplemented rats. Solid colums = iron deficient, hollow columns = iron supplemented. * $p < 0.05$ (Mann Whitney Test)

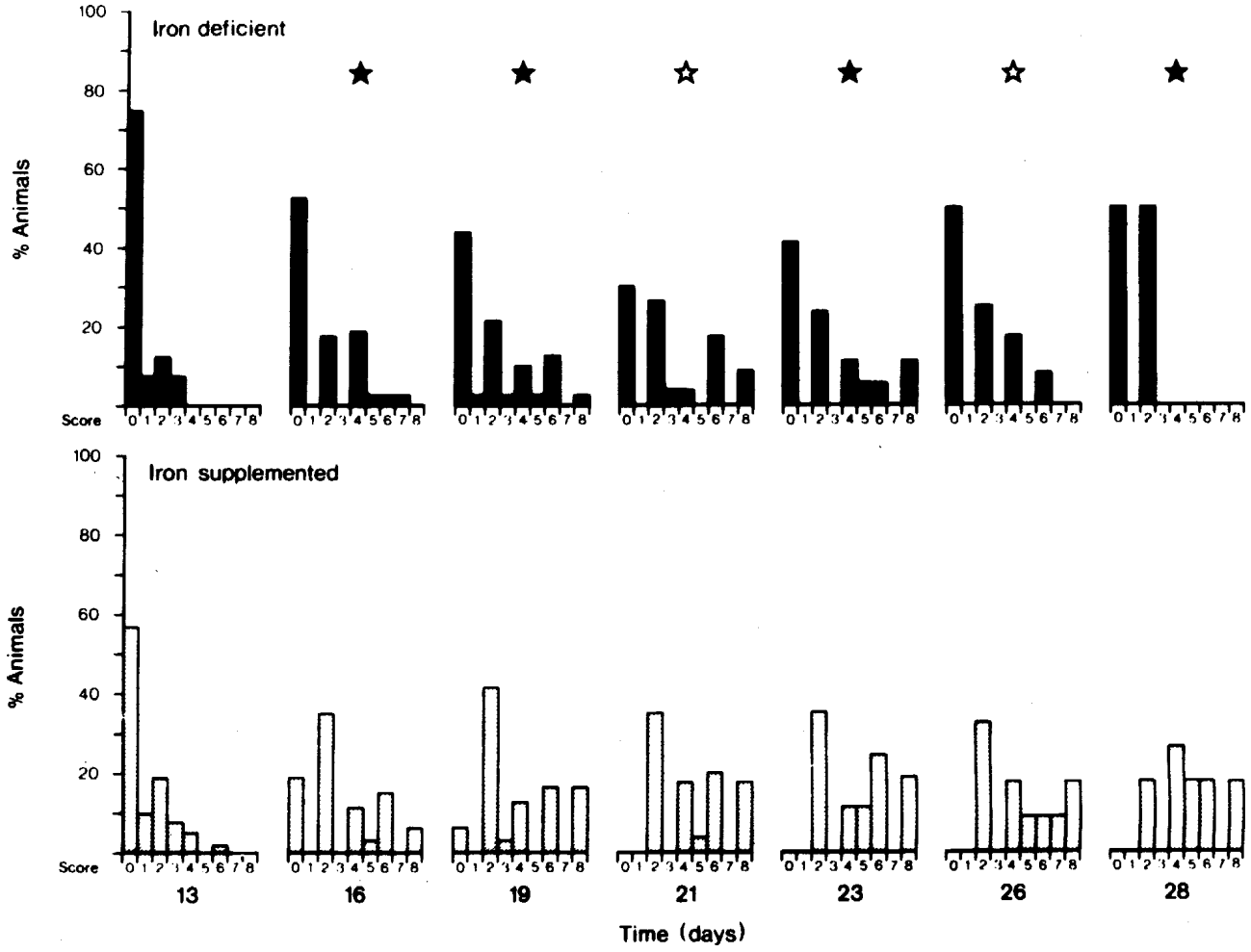

FIGURE 3. A comparison for the visual scores of redness and swelling, from 0-4 for each hind ankle joint with a maximum score of 8 per rat, at various times after injection of adjuvant in iron deficient and iron supplemented rats with adjuvant disease. *(solid) = $p < 0.05$ *(hollow) = $p < 0.01$ (Mann Whitney Test)

The third method by which we assessed inflammation in the joints was by scoring the following radiological features; bone density, new bone formation and soft tissue swelling, again from 0-4. This time we used the left ankle joint from each rat so that we had a comparison with the histological sections taken from the right joint. Results of scoring showed a similar picture to the histology with a reduction in radiological features of adjuvant disease in the iron deficient rats towards the end of the experiment (Figure 7).

Figure 8 (page 130) shows a typical radiograph of a control rat, 28 days post adjuvant showing a decrease in bone density, new bone formation and extensive soft tissue swelling. Figure 9 (page 130) however, shows a typical iron deficient rat joint at the same time point with no bone changes. So in summary we found that iron deficiency had no effects on local and systemic responses to adjuvant but did significantly reduce the joint inflammation as assessed by histology radiography and visual scoring.

As there is some evidence that reactive oxygen metabolites are involved in joint inflammation in rheumatoid disease, we went on to see if they played any role in this adjuvant model. We were proposing that iron deficiency might reduce the production of reactive oxygen metabolites in the joint tissue by preventing the formation of the toxic hydroxyl radical. It is very difficult to measure reactive oxygen metabolites directly because of their reactivity. However, it is possible to detect reactive oxygen metabolite activity using the chemical Luminol which reacts with oxidising species, such as the hydroxyl radical, producing an electronically excited ion which on returning to the ground state emits a photon. This process is known as chemiluminesence.

To investigate chemiluminesence as an indicator of reactive oxygen metabolite reactivity in the joint tissue, we induced adjuvant disease systemically as before, but this time we challenged the rats with a second injection of Freund's adjuvant into the foot pad. This gives an immediate

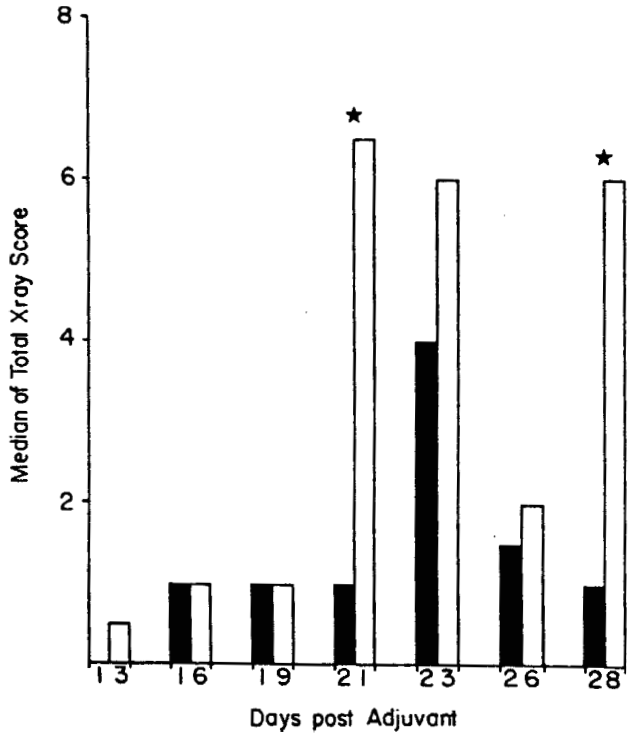

FIGURE 7. A comparison of the median total X-ray scores during the course of adjuvant disease in iron deficient and iron supplemented rats. Solid colums = iron deficient, hollow columns = iron supplemented. * $p < 0.05$ (Mann Whitney Test).

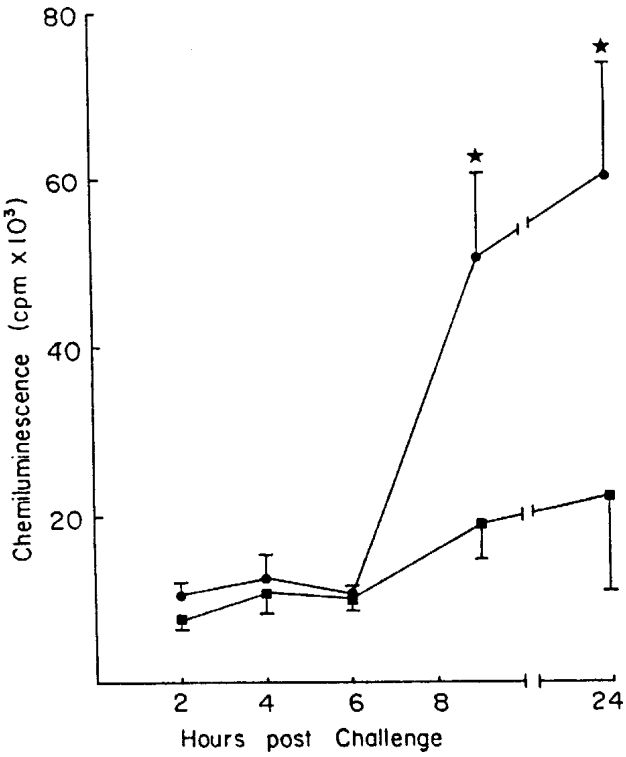

FIGURE 10. A comparison of the production of Luminol amplified chemiluminescence from the inflamed foot pad tissue of iron deficient and iron supplemented rats after challenge with Freund's complete adjuvant. Solid squares = iron deficient, solid circles = iron supplemented. * $p < 0.05$ (Student's Test).

inflammatory reaction. We removed the inflamed tissues at various times and immediately placed them into Luminol. Luminol amplified chemiluminesence was then measured using a Wallac 1216 scintillation counter set in the out of coincidence mode.

Initially reactive oxygen metabolite activity as measured by chemiluminesence was quite low in both sets of animals but then at 6 hours there was a sudden increase in reactive oxygen metabolite activity in the control animals and this was suppressed by the iron deficiency (Figure 10).

Inflammation in these animals was assessed by removing and weighing the inflamed tissues. Again a similar result was found, from 6 hours onwards the inflammation was reduced in the iron deficient animals (Figure 11, page 131). So there was a reduction in inflammation which coincided with a reduction in the sudden boost in reactive oxygen metabolite activity in the tissues.

In conclusion, iron deficiency appears to specifically protect the joint in rats with adjuvant disease. The anti-inflammatory effects of iron deficiency may be due to reduction in oxidative tissue damage. To return to the rheumatoid patients we suggest that after the initial inflammatory insult, iron deposition derived from microbleeding, sequestered within the synovium promotes further inflammation via oxidative mechanisms.

Edited comments from the question and answer session appear below

Dr Janet Vial

What is the relationship with iron and copper, because certainly copper compounds seem to give relief to arthritis?

Dr Fiona Andrews

In our study we found that copper levels were unaffected by the iron deficiency and were in the normal range for rats thus it would be difficult to speculate on the specific effects of copper on the arthritic process in this model from this work.

Dr Janet Vial

Copper is involved with superoxide dismutase which is one of the protective mechanisms against free radicals. Certain copper complexes can actually act in the same way as superoxide dismutase. There is a bit of experimental evidence that if one combines non steroidals with copper one can enhance their anti inflammatory activity and pain relieving ability quite significantly. This is still laboratory based work so maybe it is not quite so much an old wives tale as

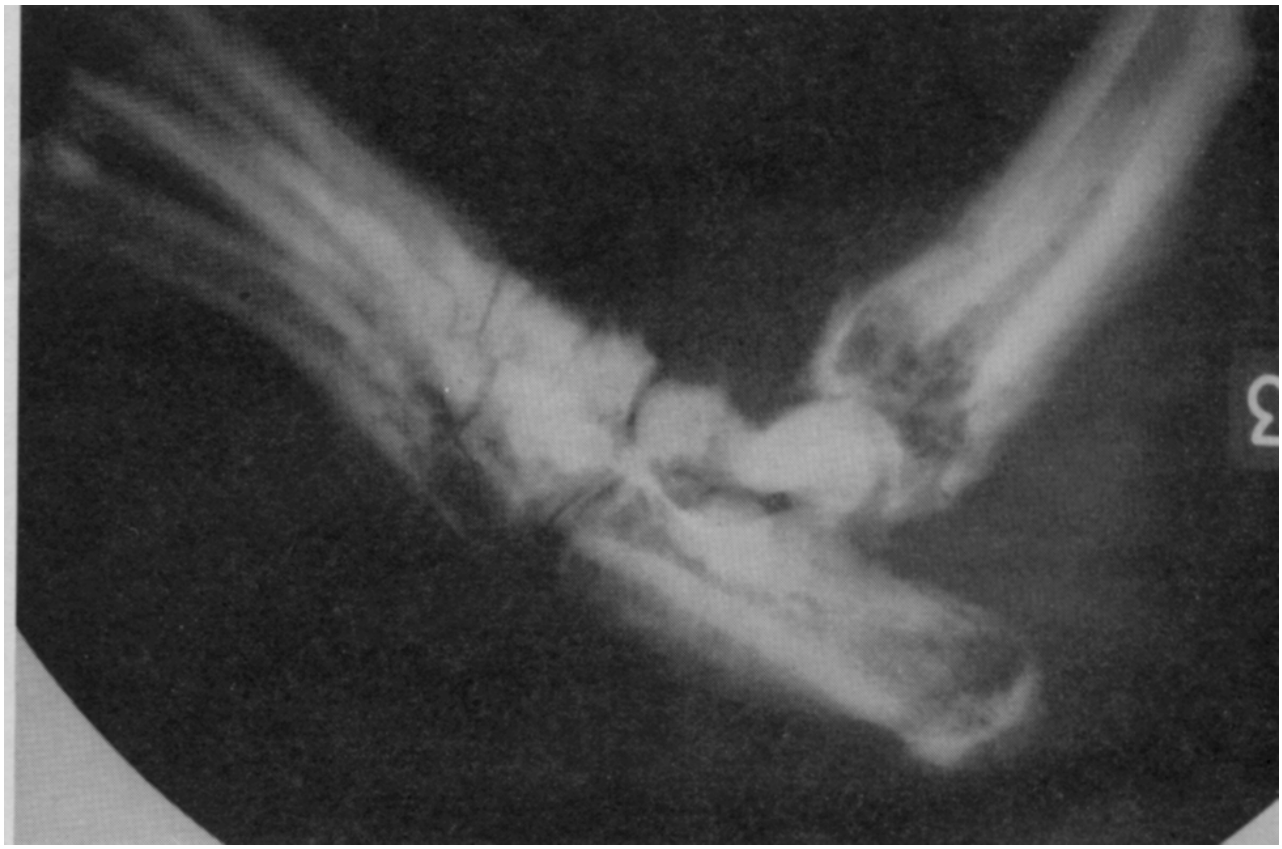

FIGURE 8. A typical radiograph of a control rat, 28 days after adjuvant injection showing a decrease in bone density, new bone formation and extensive soft tissue swelling.

FIGURE 9. A typical iron deficient rat joint 28 days after adjuvant injection with no bone changes.

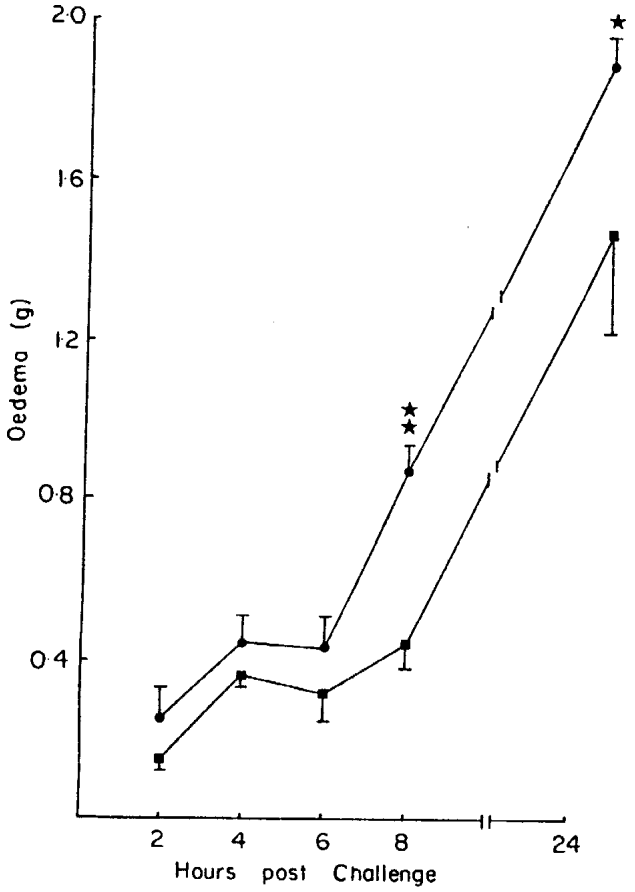

FIGURE 11. A comparison of the development of foot pad oedema in iron deficient and iron supplemented rats after challenge with Freund's complete adjuvant. Solid squares = iron deficient, solid circles = iron supplemented. * $p < 0.05$, ** $p < 0.01$ (Student's Test).

perhaps people have thought. Maybe in the future we will be seeing copper complex non steroidal agents.

Mr Christian Narkowicz

Is there any way of binding free the iron in the joints?

Dr Fiona Andrews

There has been some work to suggest that infusion of desferrioxamine into a model of inflamed synovial-like tissue, namely the allergic air pouch in rats, reduces the inflammatory reaction although I am not aware of studies where desferrioxamine has been infused into the human arthritic joint.

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5 Pearson CM. Development of arthritis, periarthritis and periostitis in rats given adjuvants. *Proc Soc Exp Biol Med* 1956; 91: 95-101.

This is an edited transcript of a recording made at a Free Radical Workshop during the joint SPUMS and Royal Hobart Hospital meeting on Hyperbaric and Diving Medicine in November 1988.

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FREE RADICALS IN HEALTH AND DISEASE

Janet Vial

An alternative title for my talk today would be oxygen, friend or foe. I am sure Joseph Priestly in 1775 never really appreciated the implications of his words when he wrote, "Though pure dephlogisticated air (which was his name for oxygen) might be useful as a medicine it might be not so proper for us in the usual healthy state of the body for as the candle burns so much faster in dephlogisticated than in common air so we might as may be said live out too fast and the animals powers be too soon exhausted in this pure kind of air". So from the very beginning Priestly perhaps appreciated the mixed blessing that oxygen is. Although we can point to many substances in our environment which are both good and bad, perhaps oxygen is unique in being essential to life but also being so potentially toxic to living cells.

I would like to go back in time. Back five billion years to the beginning of the earth. When the earth was

young it was a rather inhospitable place; very geologically active with volcanoes and fumaroles. There was no protection from ultra violet radiation and other radiation from extra terrestrial sources. The atmosphere consisted of small organic molecules like methane, ammonia, hydrogen, sulphur, and some water vapour. Although rather inhospitable to our eyes that combination of substances, plus the fact that there was no protection from extra terrestrial radiation set up the circumstances in which life could evolve. It has been shown in the laboratory that if one combines the substances which were in the early atmosphere with radiation one can produce amino acids which are the building blocks of proteins. With time, increasingly complex organic molecules were synthesized from the combination of simple molecules and still later molecules which had the ability to reproduce themselves developed. These were either DNA or substances like DNA and there may have been several trials before life as we know it evolved.

In the beginning the DNA and cells, when they came along, depended for their energy on the breakdown of the complex molecules around them. In time algae evolved which had the ability to synthesize carbohydrates using the energy from the sun and carbon dioxide. Photosynthesis had arrived. This was a very important stage in evolution because the by product of this photosynthesis was oxygen. For the first time significant amounts of oxygen appeared in the earth's atmosphere. This was toxic to the organisms that had never been exposed to oxygen before and I am sure many forms of life disappeared at that time. Those that survived either had to find niches that were away from oxygen or else they had to develop mechanisms to defend themselves against oxygen damage. Those organisms that learned to live with oxygen had many advantages, because metabolism using oxygen was very much more efficient than the previous anaerobic metabolism. This improved ability to generate energy was important pre-requisite for the development of multi celled organisms. Oxygen was important in another way. Ozone is derived from oxygen. Up until this time there was no protection for the surface of the earth from ultraviolet radiation. With increasing amounts of oxygen the ozone layer was able to develop and the surface of the earth was then protected to some degree from UV radiation. This allowed the evolution of life on land. Oxygen and development of organisms that both released and were able to use oxygen was a very important part of the evolution of life on earth.

I have mentioned cellular metabolism that uses oxygen. Most reduction of oxygen in cells occurs through the addition of four electrons to oxygen. That is important because it means that free radicals are not produced. Before I go further I will explain what free radicals are. That perhaps will explain why oxygen was toxic to those primitive cells that had no defence against it. So what are free radicals? Remember the structure of an atom or a molecule. The nucleus of an atom consists of protons, positively charged particles, and neutrons which have no charge. They form the nucleus and spinning around the nucleus in orbitals are electrons, the negatively charged particles. These electrons usually occur in pairs. These electrons, as well as spinning around the nucleus, also spin on their own axis which creates magnetic forces. However the two electrons that make up a pair spin in opposite directions, so their magnetic forces cancel each other out effectively. A free radical is a molecule that has only one electron in an outer orbital. As a result it is unbalanced and has the potential to act as a magnet. Because this is an unstable state the radical tends to grab other electrons to make its outer orbital stable. So free radicals tend to be very reactive and tend to react very readily with other molecules.

Oxygen is interesting in that it is a biradical. An oxygen molecule consists of two atoms of oxygen and the two atoms are each radicals in that they each have an unpaired electron in their outer orbital. One might ask why do not the two electrons pair up? Unfortunately they have parallel spins so they are not able to pair up because only electrons with opposite spins can pair up. That makes oxygen an unusual molecule as even if one adds another electron to oxygen there is still one unpaired electron. In fact one needs to add four electrons to the molecule to get a stable situation. So oxygen, and metabolism of oxygen, has a great propensity to produce free radicals. This is overcome in most cellular metabolism by an enzyme called cytochrome oxidase which adds four electrons in a single step therefore avoiding the production of free radicals. However some of the oxygen in the cell bypasses this system and electrons are added one at a time producing free radicals. So from every cell there is a leak of free radicals as part of cellular metabolism.

Other enzymes in the cell, which act on oxygen, also produce free radicals. This is an inevitable side effect of the production of important cell messengers and the metabolism of a number of drugs and chemicals also result in the production of free radicals. Exposure to ultra violet light and to ionising radiation also can produce free radicals. These are some of the sources of free radicals in the body (Table 1) that are known and there may be others that as yet we do not know about. The production, in the body, of free radicals is something that is happening all the time as part of its normal functioning and it is essential that the body has means of dealing with them to prevent potential damage.

Why are free radicals potentially harmful, why do we have to worry about them? The major oxygen free radical product of biological reactions is superoxide. It is an oxygen molecule with an extra electron and because it has an extra electron it is negatively charged.

In itself the superoxide does not seem to be particularly damaging. In the laboratory it is very difficult to show that superoxide does any harm to tissues so it is thought that it is a product of this superoxide that is responsible for free radical damage. The superoxide radical in the presence of

TABLE 1
Sources of free radicals in the body

Cellular metabolism
Enzymes (oxidases)
Metabolism of drugs and chemicals
Exposure to UV light and ionizing radiation

iron Fe+++ donates an electron the iron molecule producing Fe^{++} (Figure 1). Superoxide in the presence of hydrogen ions produces hydrogen peroxide plus molecular oxygen and then the combination of this hydrogen peroxide and Fe^{++} results in the production of hydroxyl radical (Figure 1) which is thought to be one of the villains of the piece. The hydroxyl radical has been shown to be a potent cause of damage in the body.

HABER-WEISS REACTION

$$O_2^{\cdot -} + Fe^{3+} \rightarrow O_2 + Fe^{2+}$$

$$2H^+ + 2O_2^{\cdot -} \rightarrow H_2O_2 + O_2$$

$$H_2O_2 + Fe^{2+} \rightarrow OH^{\cdot} + OH^- + Fe^{3+}$$

FIGURE 1. The chemical reactions by which the relatively harmless superoxide radical, the major oxygen free radical product of biological reactions, is thought to be converted to the tissue damaging hydroxyl radical ($OH^{\cdot}$).

How does this hydroxyl radical cause damage to the body? The next players in the saga are polyunsaturated fatty acids. Polyunsaturated fatty acids are fairly ubiquitous substances. They are very important constituents of cell membranes. Cell membranes are vital for function. If the cell membrane is destroyed then very shortly thereafter the cell dies. Polyunsaturated fatty acids are particularly prone to free radical attacks. They consist of a carbon backbone but they have a number of double bonds, or unsaturated bonds between the carbon atoms, and free radicals can attack at these points. When an hydroxyl radical attacks at a carbon double bond the result is the production of a lipid radical. In the presence of oxygen the next product is a lipid peroxide radical (Figure 2). This radical can break down, in certain circumstances, to form a number of different substances which in themselves are toxic to cells. They are responsible for the bad taste and bad smells of rancid fat and of food that contains fat when it goes off.

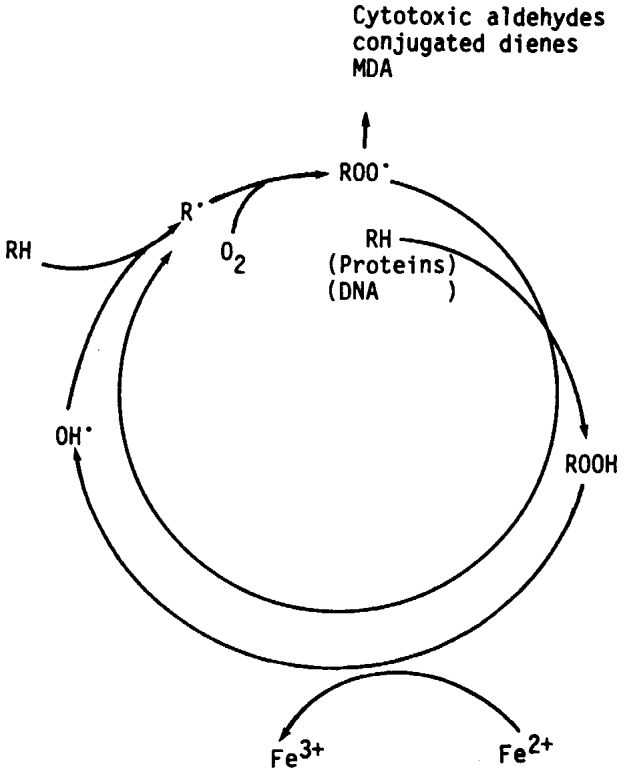

FIGURE 2. The reactions by which polyunsaturated fatty acids (RH) and the hydroxyl radicals ($OH^{\cdot}$) produce lipid radicals ($R^{\cdot}$), lipid peroxyl radicals ($ROO^{\cdot}$) and lipid peroxides (ROOH).

If that does not happen the lipid peroxide radical in the presence of more polyunsaturated fatty acids can produce lipid peroxide and at the same time produce a further radical from the polyunsaturated fatty acid (Figure 2). At this stage in the cycle other sorts of compounds can come in. Proteins or DNA can be introduced into the system and radicals can be generated from them. This can become a self sustaining reaction with eventual destruction of the organism if there is not some block in the system.

What happens to the lipid peroxide? It can hang around for a while in cell walls not doing much at all. But in the right circumstances and in the presence of small amounts of iron it can break down with the production of hydroxyl radical and the whole process can start all over again (Figure 2).

This damage particularly effects lipids which are very important in cell membranes, for cellular integrity, but as it also involves proteins and DNA, one can see very easily how radicals, if they get out of control, can wreak havoc at many levels in the cell and result in cell destruction.

Before getting carried away with the damaging effects of uncontrolled free radicals one should look to see if

there is another side of the coin. Interest in free radicals and related compounds as is something necessary to life is fairly new so there is not a lot of research in this area. It is becoming clear in a couple of areas that a certain low level of free radicals is needed for good health. For some enzymes full activity requires a low level of lipid peroxides. this applies to the cyclooxygenase enzyme which is responsible for producing prostaglandins, important mediators in inflammation, clotting and control of blood vessel diameter. The white cells are very important in defending the body against bacterial infection and a group of the white cells, the polymorphonuclear leukocytes, have the ability to release packets of free radicals in the right circumstances to kill bacteria. The body has thus used free radicals for its own defence against bacteria and people who do not have this ability are very prone to bacterial infections and often die prematurely as a result.

There are a couple more speculative areas where free radicals may be necessary for normal health. There is some evidence that free radicals are involved in the control of blood vessels and blood flow. An even more speculative area is in brain function. People with Down's Syndrome have an extra chromosome 21. Chromosome 21 contains the genetic code for superoxide dismutase which is a very important protective enzyme that prevents free radical generation. So people with Down's Syndrome have at least 50% more superoxide dismutase in their brain than normal people. It has also been found that some people with chronic psychiatric conditions have more superoxide dismutase in their brain than normal and it has been speculated that perhaps a certain level of free radicals is necessary in the brain for normal brain functioning. That is an area that is going to need more research but it is interesting to speculate.

How can one prevent tissue damage due to free radicals? First, by reducing production of free radicals through reducing exposure to oxygen, other chemicals and radiation that might produce free radicals. Reduction of the pool of labile transition metals will also reduce free radical production. I indicated before how iron could promote the production of hydroxyl radicals and other transition metals like copper and manganese can do the same thing. Protective enzymes and antioxidants are also important for preventing and reducing tissue damage.

Defence mechanisms vary in different parts of the body. Let us first consider the blood stream. Protein in the blood stream is important in defending against free radical damage. Some of the body's waste products which are carried in the blood stream actually have a function. Bilirubin, which is the breakdown product of red cells and uric acid, which is a breakdown product of DNA and nucleic acids are both important scavengers of free radicals in the blood stream. The antioxidants, Vitamin C and vitamin E are also important blood stream defences against free radicals (Table 2, page 135). In the cell membrane, Vitamin E seems to be the main factor protecting against damage, and is very important in breaking the lipid peroxidation cycle (Table 2).

Inside the cell, some of the proteins are important as are several protective enzyme systems. The glutathione system, which is a complex enzyme system, is involved in preventing free radical damage, as are the enzymes superoxide dismutase and catalase and the antioxidant vitamin C (Table 2, page 135). These multiple mechanisms tend to be overlapping. If there is a deficiency in one, free radical damage does not necessarily follow because often the others can compensate to some degree.

How do some of the enzyme systems in the cell work to prevent free radical damage? Superoxide is the first free radical product and superoxide dismutase converts it to hydrogen peroxide. Then catalase or glutathione peroxidase converts hydrogen peroxide to water preventing the production of hydroxyl radicals and damage (Figure 3). What about the lipid peroxide radicals and lipid peroxides? The oxidation of Vitamin E results in the lipid peroxide radical becoming a lipid peroxide and then glutathione peroxidase converts the lipid peroxide into innocuous alcohol that can not do any damage. Vitamin C transforms the oxidised Vitamin E back to reduced Vitamin E (Figure 4). This illustrates some of the interlinking of the different free radical defence mechanisms.

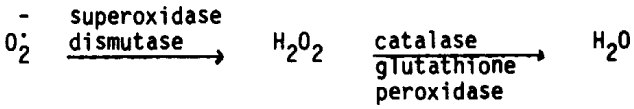

FIGURE 3. Dismutation of superoxide to hydrogen peroxide (H_2O_2) and conversion of hydrogen peroxide to water by either catalase or glutathione peroxide.

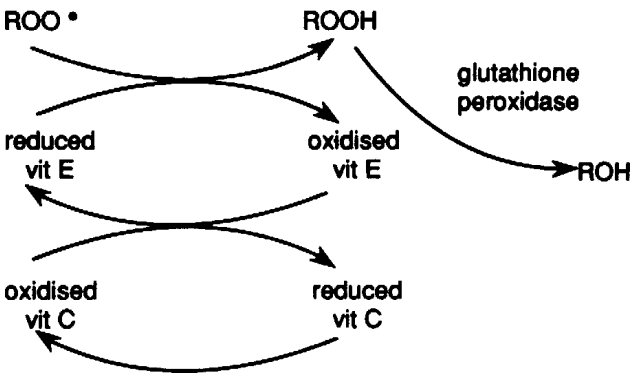

FIGURE 4. The lipid peroxyl radical ($ROO^{\cdot}$) is reduced to lipid peroxide (ROOH) by vitamin E and the lipid peroxide is converted to an alcohol by glutathione peroxidase. The oxidised vitamin E is reduced by vitamin C.

TABLE 2
Free radical defences in the blood stream, cell membrane and inside the cell.

Blood	Cell Membrane	Cell
protein	vitamin E	heme proteins
bilirubin		glutathione system
uric acid		superoxide dismutase (SOD)
vitamin C		catalase
vitamin E		vitamin C

Free radical damage occurs if there is an increase in production of free radicals or a reduction in free radical defence mechanisms. Some of the diseases where free radical mechanisms have been implicated are listed (Table 3). Aging is something of interest to most people, oxygen toxicity, atherosclerosis, reperfusion damage and cancer I will discuss in more detail shortly. There is some difficulty in finding out exactly if and to what degree free radicals are involved in human disease. The first difficulty is that they are often hard to measure. Many of them are very short lived so they are hard to capture, particularly in biological systems. So much of the evidence comes indirectly through studies which involve increasing free radical defence mechanisms or reducing free radical defence mechanisms. This makes the involvement of free radicals difficult to prove absolutely for many diseases, though the techniques are improving all the time and this is an area of avid research at the moment.

TABLE 3
Some diseases in which free radicals have been implicated

Aging	
Oxygen toxicity	lungs
	retrolental fibroplasia
Cataract formation	
Atherosclerosis	
Rheumatoid arthritis	
Parkinson's Disease	
Reperfusion damage in heart attacks and strokes	
Cancer	
Drug toxicity	

To deal with oxygen toxicity first. It has been known for a long time that patients exposed to very high concentrations of oxygen for a long period get damage to their lungs. It has been shown in premature baby nurseries that if babies are exposed to high concentrations of oxygen they get damage to their eyes sometimes producing blindness and also to a lesser extent damage to their lungs. Because exposure to high concentrations of oxygen has the potential to produce free radicals this has been suggested as a mechanism. Certainly there is quite a lot of indirect evidence from laboratory studies where free radical defence mechanisms have been reduced or enhanced and this has altered the extent of oxygen damage. Premature babies are thought to be particularly sensitive to oxygen damage because they have very low levels of vitamin E. Studies have been done to try to increase their levels of vitamin E to reduce damage.

We have been interested in this area of oxygen toxicity and have done some studies in collaboration with Dr Peter McCartney. The hyperbaric chamber is particularly useful for studying oxygen toxicity because one can expose people or animals to high concentrations of oxygen in a controlled environment. Our studies also have implications for hyperbaric treatment itself. Hyperbaric oxygen is increasingly being used to treat a number of medical conditions, but there is always a worry that perhaps some of the potential benefit could be counteracted by an increase in oxygen free radicals. So for maximising the value of this treatment it is important to know whether in fact free radicals are produced and whether there is some way of preventing the production of free radicals. So we have been interested in measuring free radicals. What we have been doing is measuring free radicals in blood. We have been taking blood and snap freezing it in liquid nitrogen. This has the advantage that it stops any further free radical processes. Also it is easier to examine free radicals using the techniques that we use, in a solid state rather than in a liquid state. We have been using electron spin resonance to measure free radicals.

The principal behind electron spin resonance (ESR) measurement of free radicals is that the unpaired electron in a free radical can act as a magnet and if put in a magnetic field it will line up in that magnetic field. If one then applies energy, in the form of microwave radiation, there may be a change in the energy level of the "magnet", i.e.. it will change its orientation in the magnetic field and this will be picked up as an absorption in the microwave radiation.

We have found that in the blood of volunteers in the hyperbaric chamber breathing 100% oxygen at 3 atmos-

pheres there is an increase in the ESR peak height compared to room air indicating an increase in free radical concentration. Not only does the peak increase but its position changes slightly, suggesting that the radical produced during hyperbaric oxygen treatment is perhaps different from the base line radical present before the subject went into the chamber.

We have now studied a number of police divers in the hyperbaric chamber. They breathe 100% oxygen at three atmospheres for three periods of 20 minutes with 5 minutes break breathing air. There is an increase in free radical peak height at the end of each oxygen period in the chamber. The encouraging thing is that the increase in height comes back to normal very quickly after re-exposure to room air. I think this demonstrates that the healthy human can tolerate an increase in free radical stress and cope with it quite readily.

There has been a lot of interest in free radicals and aging. Scientists have done some interesting calculations. They took a number of species of animals and measured their superoxide dismutase level, superoxide dismutase being an important free radical defence mechanism, and measured their metabolic rate. They divided the superoxide dismutase level by the metabolic rate and they found that this correlated roughly with the potential life span of the animals. This seemed to be good evidence that maybe free radicals were important in determining the life span of different species. Since then there have been a number of laboratory studies, depleting or increasing the free radical defence mechanism of experimental animals to see whether this makes any difference to their life span. I am sorry to report that it does not seem to make much difference to their potential life span. So perhaps swallowing vitamin E and vitamin C is not going to be the elixir of youth we thought. But one thing that did come out of these sort of studies is that many of the degenerative diseases that stop individuals from reaching their potential life span do seem to be free radical mediated. So it may be that enhancing free radical defence mechanisms will have some value in allowing more individuals to reach their potential life span without degenerative illness.

A major degenerative disease is atherosclerosis. The risk factors of cigarette smoking, hypertension, diabetes and high cholesterol, are well known. These risk factors are well established from the epidemiological evidence but how do these risk factors actually cause the atherosclerosis? On the surface of it, it might look simple, there is cholesterol in atherosclerotic plaques and perhaps the cholesterol just crosses into the blood vessel wall but it is not quite as simple as that. Cholesterol can not just pass by itself into the blood vessel wall, it does so inside macrophages. As macrophages normally do not take up cholesterol there has been a bit of a mystery as to how the cholesterol gets inside. There has been a lot of progress recently in this area. If one exposes cholesterol to oxygen or other oxidants and damages it, and makes a radical or a peroxide out of it then macrophages do take up the cholesterol. Macrophages take up damaged cholesterol but not normal cholesterol. This damage to cholesterol may be a very important point along the sequence of cholesterol to atherosclerosis, and so now there is increasing interest in looking at anti-oxidant treatment to prevent atherosclerosis. Certainly it has been shown in the laboratory that depleting the anti-oxidants, such as vitamin E, vitamin A and betacarotene, increases the oxidation of cholesterol. This is an area for interesting future research.

Another important group of diseases that limit the potential life span of many people is cancer. DNA can be damaged by the lipid peroxidation process I mentioned previously and damage to DNA certainly is a precursor for the development of carcinogenesis. What of the evidence of the involvement of free radicals in human cancer? There are a number of animal studies where animals have been depleted in anti-oxidants like vitamin A, C and E and it has been shown that these animals have an increased tendency to develop malignancies. Humans who eat a vegetarian diet which is high in betacarotene and vitamins A and C have less risk of both ischaemic heart disease and cancer. There have been a number of epidemiological studies where blood has been taken from normal populations and these various vitamins and anti-oxidants have been measured, the people have then followed for many years and then when some of them eventually died of cancer their blood levels have been compared with those who did not die of cancer. From these studies evidence is emerging that those who die from cancer or certain forms of cancer often do have lower levels of anti-oxidants than those who do not. It has been shown with lung cancer and breast cancer that those who get it have lower levels of vitamin E, for example, than those who do not get these malignancies. With gastrointestinal tumours it has been shown that those who get these malignancies have lower levels of vitamin A, C, E and betacarotene than those who do not. So some circumstantial evidence is starting to accumulate that these free radical processes may have something to do with cancer.

I would like to briefly discuss reperfusion damage in heart attacks. It is now clear that a heart attack usually results from a clot in one of the arteries supplying the heart, often at a point of atherosclerosis. There is increasing interest now in trying to limit the damage from heart attacks by getting these people to hospital very soon after their heart attack and giving them therapy to break down the clot and re-establish blood flow. Drugs such as streptokinase and tissue plasminogen activator are being increasingly used for this purpose. There is a potential problem in this treatment. When a tissue is not receiving blood it does not receive oxygen so it can not make free radicals. However it can not metabolize normally either and this results in the breakdown of the free radical defence mechanisms. So when the blood supply is restored and oxygen reintroduced there is a sudden burst of free radicals and the tissue has no way of defending itself against them. There is certainly some evidence that this period of reperfusion when blood flow starts again is a time when there can be damage. Some of the benefit of reintroducing the blood flow may be counteracted by the

disadvantage of this reperfusion damage. Because we are using this sort of treatment there is increasing interest in using free radical scavenging mechanisms to prevent damage. There are clinical trials, I know, going on in the United States looking at treatment with free radical scavengers. It remains to be seen how beneficial it will be in presenting reperfusion damage.

This brief outline shows that free radicals have the potential to be very important in many human diseases including some of the most common diseases that are likely to stop us from reaching our potential life span. I hope that I have provided some insight into how free radicals which were once the domain of academic chemists now have implications for medical research and hopefully in the future understanding of their role in disease will result in improvements in prevention and treatment.

This is an edited transcript of a recording made at a lectureduring the joint SPUMS and Royal Hobart Hospital meeting on Hyperbaric and Diving Medicine in November 1988.

Dr Janet Vial is Senior Lecturer in the Department of Medicine, University of Tasmania Clinical School, 43 Collins Street, Hobart, Tasmania 7000, Australia.

DIVING SAFETY MEMORANDA

Department of Energy
London SW1P 4QJ
May 1989

DIVING SAFETY MEMORANDUM NO. 4/1989 EXPOSURE LIMITS FOR IN-WATER DECOMPRESSION

Diving Safety Memorandum No. 5/1988 recommended that all surface decompression dives should be arranged so that the planned bottom times did not exceed the exposure limits defined in the table attached thereto.

At that time there was only limited data available on the experience of using in-water decompression techniques in the UK sector. Hence diving using this technique was not included in the safety memorandum.

From the 1988 dive data, it is evident that there has been an increased use of the in-water decompression technique, and that long bottom times using this technique have resulted in serious cases of decompression sickness. Though the amount of information available is limited, it is felt that the industry should be made aware of this trend.

It is therefore strongly recommended that the guidance on exposure limits given in Table 1 to DSM 5/1988 is also followed when using the in-water decompression technique.

R. GILES
Chief Inspector of Diving

DIVERS ALERT NETWORK (DAN) 14th DIVING ACCIDENT AND HYPERBARIC OXYGEN TREATMENT COURSE

October 21 - 31, 1989
Palau Pacific Resort, Palau, Micronesia.

Course Description

This eight day course in Diving Accident Management and Hyperbaric Oxygen therapy is designed for physicians, emergency medical personnel, including paramedics and nurses. Portions of the course may be of interest to dive masters, dive instructors, and other non-medical dive related personnel.

The aims of the course are to provide the facts relevant to understanding the management of diving accidents, especially those bearing on the basic physics and physiology, and the subsequent treatment methods available.

The course format will involve morning and some afternoon and evening didactic sessions of lectures and case presentations. These will be supplemented by small group interactions with the faculty for direct question and answer sessions, review of case histories and some special video instructional tapes. Six afternoons will allow spectacular two tank diving.

All proceeds from the Course go to support the Divers Alert Network (DAN).

Faculty

Drs. Peter Bennett, Carl Edmonds, Des Gorman and Yancey Mebane.

Course charge $US 495 payable to **Duke University Medical Center**. Register by sending cheque with name, address and telephone number to:

Office of Continuing Medical Education
Box 3108, Duke University Medical Center
Durham, North Carolina 27710, USA.

For **accommodation** and travel from USA contact:

"Duke/DAN Palau Course",
International Diving Expeditions,
11265 Knott Avenue,
Cypress, California 90630, USA
Telephone: (714) 897-3770

SOUTH PACIFIC UNDERWATER MEDICINE SOCIETY ANNUAL GENERAL MEETING 1989

THE MINUTES OF THE ANNUAL GENERAL MEETING HELD AT 09.30 ON THE 3RD JUNE 1989 AT LE LAGON RESORT, VILA, VANUATU

Present

All Members attending the Scientific Conference.

Apologies

Drs Acott, Barry, Cook, Gorman, Knight, McCartney, Rehfish, Sutherland, Veale, and Westlake.

1. **Minutes of the previous meeting**

These had been on display and were taken as read. Dr Brand moved that the minutes be accepted as a true record. Seconded Dr Haller.

Carried.

2. **Business arising from the minutes**

No business was raised.

3. **Reports**

3.1 Reports were made by the President and Secretary.

Dr Lourey moved that these reports be published in the Journal. Seconded Dr McKee.

Carried.

(See this page and page 121)

3.2 The Treasurer's report was read by the Secretary. Dr Barry will be asked to enquire whether it is now appropriate to depreciate the equipment the Society owns rather than writing it off. It was noted that no financial statement and no subscriptions had been received from the New Zealand Chapter for over twelve months.

3.3 The Treasurer moved that annual subcriptions be raised to $50.00 for members and $30.00 for associates. Seconded Dr Davies (Secretary).

Carried.

3.4 The Treasurer moved that "If any subscription was in arrears for a period greater than six months, the Treasurer, having duly notified that member, shall instruct the Secretary to remove that member's name from the current membership". Seconded Dr Davies (Secretary).

Carried.

4. **New Executive Committee**

The following members were elected unopposed.

President	Dr A Slark
Immediate Past President	Dr C Acott
Secretary:	Dr D Davies
Treasurer	Dr G Barry
Editor	Dr D Walker
Public Officer	Dr J Knight
Committee:	Dr G Lourey
	Dr D Gorman
	Dr P McCartney

5. **Life Member**

Dr Davies moved that "In light of his long record of service to the Society, having been a foundation member and long time Editor of the Journal, Dr Douglas Walker be elected to Life Membership of the Society." Seconded: Dr Brand.

Carried. (by acclamation).

6. **AGM 1990**

After preliminary investigation the Executive proposed that the Island of Palau be the site of the next AGM. This was agreed to by the Meeting.

7. **AGM 1991**

The Secretary has been in contact with the Indonesian Hyperbaric Society who have agreed to a joint meeting, probably in Jakarta. A sub committee of Drs Slark, How, Lloyd and Davies has been formed to work with them on the project.

The Meeting closed at 10.40 a.m.

PRESIDENT'S REPORT

I am pleased to report that the membership of the Society continues to expand and in fact extends well beyond the geographical limits that our name would imply. Currently we have 635 members from Australia, 158 from New Zealand and the rest from such divers places as Japan, Canada, Oman and Ireland.

The year has been an eventful one for the Society, in that arrangements have been finally made for the incorporation of the Society in the State of Victoria. You will recall that the need for this became apparent when we were threatened with legal action after we had found it necessary to dissociate the society publicly from the activities of an

entrepreneurial diving doctor. The incorporation will give the society proper legal status and the protection of its officers, members and funds which this provides.

Suggestions for the improvement of the Journal have been examined, and an editorial sub-committee has been formed and Dr John Williamson has been invited to assist with this with the possibility that the organisation be further strengthened in the future. It is planned that the Journal should take on the standard format favoured by the majority of scientific publications and that advertising should be more actively sought. You will already have seen that we felt that the cover was not the most appropriate place for a cartoon. Change in the size and structure of the cover have been precluded by large stocks of blue card. Again we have to thank Douglas Walker and John Knight for their continuing work in the production of the Journal.

The sudden collapse of the National Safety Council of Australia (Victorian Division) at the end of March had potentially disastrous consequences upon the safety organisation of many risk prone activities and particularly from our point of view the Divers Emergency Service, the evacuation of persons suffering from diving accidents, and those requiring hyperbaric treatment. The Executive Committee wrote to the Premiers and Ministers of Health of those states affected by the collapse pointing out the importance of preserving the service that had been provided by the National Safety Council of Australia (Victorian Division). The Committee also decided to donate $500 to the Royal Adelaide Hospital towards the telephone bill of the Divers Emergency Service.

A successful scientific meeting was held in Hobart in November 1988. However it was disappointing for me that the attendance seemed exclusively medical and did not have any of the local sports divers or instructors and that the publicity and content had not been directed at a wider audience. It is hoped that in the future at least one such regional meeting will be held annually, and that our educational function be extended.

Following the confusion that had resulted from the expectation of the Undersea and Hyperbaric Medical Society (UHMS) that we were intent upon a joint meeting when we had already abandoned the idea, I have been invited to UHMS Executive meetings. Unfortunately distance and expense has precluded my attendance, but I hope to go to the meeting in Hawaii, together with our Secretary.

I would like to conclude by thanking all the members of the Executive for their work in the past year and look forward to their co-operative enthusiasm in the future. In particular I would like to thank David Davies for the tremendous effort that he puts in as Secretary and to say how pleased that I am that he is prepared to continue.

Tony Slark

SECRETARY'S REPORT

It is my great pleasure to present my fourth Annual Report.

1. **The Membership**

This currently stands at about 800 of whom, 75 have joined this year but in the same time there have been about 100 lost, either by resignation, by being unfinancial or by moving address without letting us know, so that the Journals are returned. With the numbers of divers being trained in Australia and New Zealand and each one of these requiring a medical examination, the doctors who do these examinations ought to all be members of the Society. It is up to the general membership of the Society to encourage their colleagues to join and gain insight into the problems of divers.

In order to keep up with the membership and make a record of what is each member's specialty and whether or not he does diving medicals, I have made a card index of the entire Society using old application forms, current membership lists and also asked questions if I was corresponding with any of the members. These cards also have a spot for diving medical training and the date of joining the Society. I was considering incorporating the tax file and Medicare numbers but the cards are only 6 by 4. So if you do write to me for some reason, please let me know if you have any diving medicine training, if so, where and when, if you do diving medicals, if so where, and whether you are a diver or not, and if so what training organisation and to what level. All this information is on the new application forms, so do not feel you are being singled out for special attention.

2. **Incorporation**

This is now proceeding and is in the hands of our Solicitors. Dr John Knight has been appointed the Public Officer. The reasons for this were that John has acted unofficially in this capacity for some years and his address has been the contact address for the Society. As well, the public officer must be resident in Victoria and his function is to act as liaison officer with the Corporate Affairs Office.

The Executive has spent hours both singularly and as a group, going through the proposed constitution and dissecting it line by line, word by word. We had hoped to have it printed and distributed in time for this meeting, but time has run out.

3. **Education**

In addition to the Annual Scientific Meeting for members, regular courses are being conducted at Royal Adelaide Hospital under the guidance and drive of Dr Des Gorman. The availability of these courses puts paid to the argument that training for doctors in diving medicine is not available. There is no longer any excuse for the Mickey Mouse Medical.

In November a seminar was held in Hobart organised by Dr Peter McCartney. Most of the Executive attended, but

lack of advertising resulted in poor outside interest, so that the Society has been placed under some financial pressure.

4. **Diploma of Diving and Hyperbaric Medicine**

The Executive decided early in the year that for the Diploma to be a viable entity and become recognised as a reputable qualification, those doctors actively practising in this field should be given the opportunity to gain the award. So it was decided to invoke a grandfather clause and the appropriate people were invited to apply for the award. All 30 accepted. This brought the total number of holders of the Diploma to 50. Since that time Dr David Smart of Hobart and Dr Wong Ted Min of Singapore have applied to the Board of Censors and have been granted the award. Their written material submitted for the award was published in the Journal (1989; 19 (1):)

To further the aim of acceptance of the Diploma, as an indication of higher qualification, the Executive wrote to all the Medical Boards in Australia and New Zealand setting out the requirements for the Diploma and requesting that it be recognised. We are still waiting for replies from several states.

5. **Standards Association**

For some reason consensus could not be obtained on DR88026 for Entry level divers so the Committee had to be reconvened. SPUMS was invited to have a representative on that Committee and John Knight volunteered and was appointed. Any submissions or suggestions about the proposed standard should be communicated to Dr Knight.

6. **WA Government Task Force**

You may recall that SPUMS made submissions to this last year and the preliminary recommendations have been published in the SPUMS Journal. Since that time final submissions have been made and the Minister is trying to make up his mind. Currently the accent is on self regulation by the industry and accreditation of all diving instructors to at least NCAS Level 2. Those training organisations not yet accredited are being assisted by the Ministry to update their teaching. Cross over courses are being conducted for experienced divers who either have no qualification or they wish to gain an acceptable qualification. A great many of these show major deficiencies in their knowledge of medical problems, of equipment and of technique.

If, over the next couple of years, the industry is seen to be incapable of self regulation then legislation will be brought down.

It should be noted that the Queensland Government is making similar moves as a result of the many accidents that seem to be occurring along the Great Barrier Reef.

7. **National Safety Council of Australia (Victorian Division)**

The financial collapse of the National Safety Council of Australia (Victorian Division) (NSCA) has wide ramifications throughout the diving community. Firstly, several talks at this meeting were to be given by NSCA members who have all had to cancel. Secondly, the NSCA supplied the funding for the Diver Emergency Service (DES) Network which is based at the Royal Adelaide Hospital. As the hospital is already in financial difficulties, it was agreed that a donation of $500.00 be made to the hospital to cover at least part of these expenses. I believe the Diving Organisations should be required to cover these expenses as it is their divers who benefit from this facility.

The Hyperbaric units in Queensland, South Australia and Victoria were all owned and operated by the NSCA and their potential closure would wreak havoc with not only the sports divers, but all government sponsored diving by the Police, Port and Harbours, Fisheries and so on. Letters have been sent to the Premiers and Health Ministers of each of these states, outlining the ramifications. So far the only response has been to acknowledge receipt of the letters.

In the three states affected, the NSCA conducted all transfer under pressure evacuations. It was pointed out to the politicians that closure of the facilities in the states would leave Sydney, Fremantle and Hobart as the only places where hyperbaric treatment could take place.

8. **Acknowledgements**

In closing, I would like to thank the Executive Members for their help throughout the year. The President has proved to be a great backstop and is an excellent reference about diplomacy.

Dr John Knight is an untiring worker for the Society. He puts the Journal together almost single handedly and now also represents the Society on the Standards Association and as Public Officer.

The Treasurer, Dr Grahame Barry, continues to monitor the finances closely and has great success in encouraging slow payers to part with their ill retained profits.

Dr Des Gorman is always a source of advice, a fund of knowledge, and enthusiastic teacher.

Membership of the Society has been fairly static the last couple of years. There are too many members sitting back, taking a passive role waiting for the few workers to get things done. The Society desperately needs new members, it needs to have a more prominent role in the diving community. The Medical Boards, the Australian Medical Association and the politicians must all be made aware that divers have their own medical problems and a general undergraduate medical course does not give doctors the knowledge to deal with these.

As far as the Secretary's position goes, the job is only as good as the effort that is put into it. If the Society gets

much bigger, I believe we must consider employing a full time secretary. The day to day running of the organisation should be put into the hands of a Secretariat with an Executive Director to handle the organisation of meetings, seminars and publish a newsletter in addition to the quarterly Journal. Such a role is a bit much for one person trying to run a full time practice on the side.

David Davies

TREASURER'S REPORT 1988/1989

The most noticable feature of the year's finances is a drop of overall assets of $ 5829 from $ 38,052 in 1988 to $ 32,223 in 1989, together with a fall in income of $ 4027 of which the greatest amount can be attributed to a drop in subscriptions of $ 3401. This represents a loss of about 100 members.

Nevertheless, the expenditure was also reduced and, had it not been for the emergency donation to the ailing Diver Emergency Service (DES) Network, brought on by the collapse of the National Safety Council of Australia (Victorian Division), we would have been marginally in the black.

On the financial statement it will be noticed that the closing balances are gradually diminishing. These balances are important as there is a two month gap between the end of our financial year (April 30) and the beginning of the subscription year (July 1). During this period a Journal is issued, and money must be kept in hand to cover production and mailing costs. It should be pointed out that the majority of items listed in the expenditure column under "Secretarial", "Postage" and "Equipment" pertain to the Journal which costs in the vicinity of $4000 per issue. This makes it our largest expenditure item. There is little doubt that the money is well spent, as the SPUMS Journal now has a reputation as the leading medical sports diving publication in the world.

Production costs continue to rise, the large figure of $10,487 for 1988 included the cost of 12 months supply of the blue cover material. Postage rates too are not expected to fall.

As a consequence of these factors I strongly recommend that the membership fees be raised for the coming year to $ 50.00 for full members and $ 30.00 for associates and I trust that the AGM will give this matter its full support. As well I suggest that any member or associate who is more than six months overdue with his subscription be struck from the list of members after being duly notified by the Treasurer.

I hope the Vanuatu Meeting has been a great success and look forward to joining you again next year.

Grahame Barry

AUDIT REPORT SOUTH PACIFIC UNDERWATER MEDICINE SOCIETY

NEWPORT BEACH
N.S.W. 2106
15 May, 1989

I have conducted various tests and checks as I believe are necessary considering the size and nature of the Society and having so examined the books and records of The South Pacific Underwater Medicine Society for the year ended 30 April, 1989 and report that the accompanying Statement of Receipts and Payments (page 124) has been properly drawn up from the records of the Society and gives a true and fair view of the financial activities for the year then ended.

David S. Porter, FCA.
Chartered Accountant

(Registered under the Public Accountants Act, 1946, as amended).

DIVER EMERGENCY SERVICE UPDATE

Readers will remember that in the January-March issue of the Journal we reproduced a letter written by the Secretary to the Premiers of Queensland, South Australia and Victoria. We reproduce below letters from the South Australian and Queensland Governments expressing their determination to continue to fund hyperbaric facilities and provide a service for hyperbaric retrieval.

Unfortunately the Secretary has yet to hear from the Government of Victoria. Dr Knight, who wrote a similar letter to the Premier of Victoria, has just received the letter from the Ministry of Police and Emergency Services reproduced below. However an article in The Age of 31.8.89., also reproduced below, throws considerable doubt on the future of hyperbaric facilities in the State of Victoria.

3rd Level Citi Centre
11 Hindmarsh Square
Adelaide, SA. 5000
22 May 1989

Dear Dr. Davies,

Thank you for your recent letter concerning the provision of hyperbaric and diving medical services in South Australia.

I am certainly aware of the importance of these services, not only for divers but also for the treatment of a

SOUTH PACIFIC UNDERWATER MEDICINE SOCIETY
STATEMENT OF RECEIPTS AND PAYMENTS
FOR YEAR ENDING 30th APRIL 1989

		1989	**1988**	**1987**
OPENING BALANCE				
Standard Chartered Finance Ltd.		1,000	1,000	1,000
National Mutual Royal Bank		5,177	7,361	7,659
National Australia Bank		789	403	80
Cash on hand		22	26	60
	Total Opening Balances	6,988	8,790	8,799
INCOME				
Subscriptions		24,074	27,475	22,945
Interest		1,161	1,787	1,680
	Total Income	25,235	29,262	24,625
	Total Receipts	$ 32,223	$ 38,052	$ 33,424
EXPENDITURE				
Secretarial		4,143	3,735	2,696
Stationery		576	198	240
Journal		7,310	10,487	6,481
Postage		4,047	4,198	2,465
Travel		7,775	10,335	4,415
Equipment, see note		372	850	7,451
Miscellaneous		122	358	576
Bank charges		232	286	268
Returned cheques		25	25	42
Audit		200	200	-
Legal expenses		180	392	-
Donation to DES		500	-	-
	Total expenditure	25,482	31,064	24,634
CLOSING BALANCES				
Standard Chartered Finance Ltd.		1,000	1,000	1,000
National Mutual Royal Bank		4,228	5,177	7,361
National Australia Bank		1,503	789	403
Cash on hand		10	22	26
	Total Closing Balances	6,741	6,988	8,790
	Total Payments	$ 32,223	$ 38,052	$ 33,424

NOTE Equipment is written off as purchased.

broad spectrum of medical conditions.

Although the additional costs to the South Australian Government are not inconsiderable, the Royal Adelaide Hospital is reviewing the services, in association with the Director of the Hyperbaric Unit, Dr Des Gorman, and I am confident that permanent arrangements will be made to enable the Unit to continue to operate beyond the 30th June 1989.

Don Hopgood
Deputy Premier & Minister of Health

State Health Building
147-163 Charlotte Street
Brisbane, Queensland 4000
14 June 1989

Dear Dr. Davies

Thank you for your letter concerning possible interruption to the hyperbaric and diving medical services following the collapse of the National Safety Council of Australia (Victorian Division).

The Queensland Government has been concerned with the loss of services as a result of the collapse and has provided funding as a short term measure to ensure the continuation of services in the interim.

The principal concern centred on the continued availability of the portable recompression chamber service to support the rapidly expanding recreational diving activities in North Queensland.

The Honourable M.J. Ahern, MLA, Premier and Treasurer and Minister for State Development and the Arts, earlier this month announced that the State Government was negotiating the purchase of key National Safety Council of Australia assets to maintain safety standards for Queensland's tourist diving industry.

On-going discussions are being held so that equipment will be integrated into the existing emergency network. The operation of the portable hyperbaric unit would be linked to the fixed recompression chamber currently at the Australian Institute of Marine Science (AIMS). It has been proposed that the fixed recompression chamber be released at the Townsville Hospital.

The Queensland Government will also continue its strenuous efforts to secure private enterprise involvement so that existing treatment facilities are continued.

Retrievals and hyperbaric treatment facilities will continue to operate in this State.

Ivan J. Gibbs, MLA
Minister for Health

PREMIER'S DEPARTMENT
100 George Street
Brisbane, 4000
29th June, 1989.

Dear Dr. Davies,

The Honourable the Premier has passed a copy of your letter of 4th April, to me and has asked me to deal with it.

I regret not being able to reply to your letter earlier but you will no doubt appreciate the efforts which the Queensland Government has made since the financial collapse of the former National Safety Council of Australia (NSCA), Victorian Division's operation to ensure that the portable hyperbaric facility and retrieval operations are maintained at Townsville in North Queensland.

Recently, the Government decided to acquire the portable Drager "Duo-Com" recompression chamber which, in turn, will become part of a field treatment, retrieval/transportation operation integrated with a fixed hyperbaric chamber located in Townsville's General Hospital. Specialist medical services will be available to assist in all phases of this integrated operation.

It is expected that a private enterprise organisation comprising a rationalised operation involving former NSCA pilots, air crew, maintenance engineers, life support technicians (LST) etc. will provide an ongoing service in response to community needs.

It is understood that opportunities for further training of those Government agencies likely to utilise or need this type of service will become available in future.

J.J. Mulheron
Acting Director-General

Government of Victoria
Ministry for Police and Emergency Services
25.August.89.

Dear Dr Knight,

HYPERBARIC FACILITIES IN VICTORIA

The Minister has asked me to than you for your letter of 8 April 1989.

Appropriate measure have already been put in place by the State Government and its agencies, in respect of matters within the State's purview, to deal with shortfalls in service and protection to the community caused by the closure of the NSCA.

I am informed that the Health Department's Ambulance Directorate has leased a portable recompression unit, which is capable of being transported by helicopter or truck, and is based at the La Trobe Valley Airfield. The unit is staffed on a 24 hour on call basis by former NSCA personnel, under qualified medical supervision. The western coastline of the State is subject to an arrangement with the South Australian Ambulance Service, under which patients are transferred by pressurised fixed-wing aircraft for direct admission to the Alfred Hospital's hyperbaric chamber.

In general terms, I understand that the Ambulance Directorate is of the view that the standard of the hyperbaric care in Victoria has been maintained since the collapse of the NSCA.

Please accept my apologies for the delay in responding to your letter. It has been mistakenly assumed that the correspondence had been referred to the Health Department for direct reply to you. It would be preferable if any further enquiries in relation to the present matters were directed to that Department.

David Young,
Acting Assistant Director,
Fire and Emergency Services Division.

This article appeared in The Age, of Melbourne,on 31.8.89. It is reproduced in full. It gives a rather different impression to the letter above.

POLICE LACK FUNDS TO BUY NSCSA'S MOBILE DECOMPRESSION CHAMBER

Paul Conroy.

The Victoria Policed are unlikely to buy a mobile decompression chamber to improve depleted emergency services for divers with the bends.

Members of the police search and rescue squad have been notified that funds are not available to buy the unit, which is owned by the Victorian branch of the National Safety Council of Australia and would cost at least $ 50,000.

An officer with the squad had already been trained to operate the unit.

Victoria's professional divers and thousands of sporting enthusiasts already face the prospect of losing the state's only hospital based decompression chamber at the Alfred Hospital. It is also owned by the NSCA.

The two units are likely to be sold, along with other NSCA assets, as a result of the financial collapse of the group.

A hospital spokesman said 108 patients had been treated at the Alfred Hospital since January 1988. The unit also treats patients with gas gangrene and toxic-gas accident victims.

The head of the Alfred's intensive-care unit, Dr David Tuxen, said services in Victoria could be plunged into crisis unless sponsors were found to buy another unit. The chamber is on 24-hour service and involves the employment of 40 nurses and seven other staff.

Dr Tuxen said there was a privately operated decompression chamber at Port Melbourne, but it did not provide a 24-hour service.

LETTERS TO THE EDITOR

RADIAL KERATOTOMY

Lions Eye Institute, Perth.
3rd April 1989

Dear Sir,

It has come to my attention, from a general practitioner who had attended a SPUMS meeting, that comments were made about radial keratotomy which perhaps warrant clarification. I write this letter both as a cornea specialist who has an interest in protecting the cornea and treating corneal conditions, and at the same time as a surgeon who performs radial keratotomy, not wishing to have this procedure wrongfully defamed.

The two queries related to the safety of scuba diving following radial keratotomy and in conjunction with this the statement that there has been a reported case of eyeball rupture whilst scuba diving following radial keratotomy.

I would consider myself to have a good knowledge of the recent literature about the cornea and in particular refractive surgical procedures. I am unaware of either of these two problems.

To confirm this I did a literature search for all traumatic injuries and also contacted one of my mentors, Dr George Waring in Atlanta, who is the chief investigator for the PERK study and probably the most knowledgable person with regard to radial keratotomy. He stated that he sees no reason to advise people that have had radial keratotomy done against scuba diving and sees no rationale for this. He states that the pressure inside the mask is equalised. and the eyeball is not under increased pressure. He added that many divers have it performed so that they can actually dive without the hinderance of spectacle or contact lens correc-

tion. Even in the event of accidental removal of the face mask, the pressure would not be sufficient to cause rupture of the globe. There is significant evidence with animal studies and from clinical experience with blunt trauma to human eyes, the pressure would not be sufficient to cause wound rupture. Also he is unaware of any incident of wound rupture in deep sea divers. If there is such a case reported in the non-ophthalmic literature I would be grateful for the reference.

With regard to the armed forces stance on radial keratotomy, the Army has released its statement which disallows it in their personnel but this report was certainly very biased even to the uninitiated. As yet I have not seen a similar document from the Navy or Air Force. With regard to the armed forces of the United States, all refuse flight status to people with radial keratotomy, but allow them to participate in all other levels of military activity.

This letter is not a paranoic defence of radial keratotomy, but purely a clarification of some points about a procedure which unfortunately has many myths and untruths associated with it. If any of your readers have further queries I would be happy to answer them.

Geoffrey J.Crawford.
FRACO, FRCS

NINTH INTERNATIONAL CONGRESS ON HYPERBARIC MEDICINE

Prince Henry Hospital,
P.O.Box 233,
Matraville,
NSW 2036
April 1989

Dear Sir,

The proceedings of the Ninth International Congress on Hyperbaric Medicine, held in Sydney in 1988, are now available.

They can be purchased from

Medical Convention Services,
P.O.Box 335,
Heidelberg,
Victoria 3084,
Australia.

The price including postage is $ Australian 35.00 or $ US 29.00

Ian P.Unsworth,
Director of Diving and Hyperbaric Medicine,
(President of the Ninth International Congress on Hyperbaric Medicine)

JOURNAL OF WILDERNESS MEDICINE

Emergency Department,
Vanderbilt University School of Medicine,
Nashville,
Tennessee 37232,
U.S.A.
April 3 1989

Dear Sir,

I would be most grateful if you could announce to your members in some fashion the upcoming Journal of Wilderness Medicine, due for quarterly publication in 1990. It will publish original research on all aspects of wilderness medicine: including high altitude and climbing; cold and heat related phenomena; natural environmental disasters; immersion and near drowning; diving and barotrauma; hazardous plants, reptiles insects and marine animals; animal attacks; search and rescue; ethical and legal issues; wilderness trauma management.

The Journal should have considerable appeal to members of SPUMS (of which I am a member). John Williamson will serve as a member of the Editorial Board. We are in need of high quality manuscripts for publication, particularly in the early going. Making your members aware of our presence would therefore be of great assistance.

For information about subscriptions or advertising write to

Journals Promotion Department,
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London EC4P 4EE, United Kingdom.

Papers for editorial consideration should be sent to me in Nashville.

Paul S.Auerbach,

GRAVESTONE PHOTOGRAPH WANTED

Department of ENT, Queen Elizabeth Military Hospital
Stadium Road, Woolwich
LONDON SE18 4QH, United Kingdom
23rd May 1989

During the SPUMS meeting on the Maldives in 1985 someone showed a slide of the gravestone of an unfortunate RAMC officer who succumbed to Stingray poisoning on Thursday Island in 1915 . Unfortunately I cannot remember the speaker's name but I will happily pay the film and postage costs of any SPUMS member who can help me find theofficer's name or provide a photograph of the gravestone.

Nick Cooper, Major RAMC.

BOOK REVIEWS

CASE HISTORIES OF DIVING AND HYPERBARIC ACCIDENTS.
Edited by Charles L.Waite.
225 pages. Soft cover. 1988.
Undersea and Hyperbaric Medical Society,
9650 Rockville Pike, Bethesda, Maryland 20814, U.S.A.

This is a compilation of a series of case reports by 19 experts (15 from the U.S.A., 3 from the U.K. and one from Australia) in different fields of diving and hyperbaric medicine. It was funded by the Dreyfus Foundation and the National Oceanic and Atmospheric Administration (NOAA) in the U.S.A.

The chapters encompass decompression sickness and pulmonary barotrauma (half the book, 13 chapters), dysbaric osteonecrosis, drowning and near drowning, oxygen toxicity, hypoxia, carbon dioxide poisoning, carbon monoxide poisoning and nitrogen narcosis.

The text was an attempt to compile the experience of the authors in their approach to cases, which were supplied by the authors and others, to demonstrate the various clinical presentations.

The fact that diving medicine encompasses many different specialties, was highlighted by this text. Most chapters start with an introduction to the particular subject, however this introduction is often so brief that it is of little value. The real value is in the authors' approach to the cases.

Most of the cases are adequately informative and illustrative. Unfortunately some are incomplete in clinical data and, even more important, do not broach any differential diagnoses. A few have either typographical errors or incorrect diagnoses, but even these can be used for student training.

The most annoying aspect of the book is the total absence of an index so that if, for example, one wanted to look up such aspects as asthma, cardiac arrhythmias, etc., it is almost impossible.

It is therefore not a reference text. It is a book which one should be given to read from cover to cover with a black pen to compile your own index, and a red pen to delete the occasionally over-simplistic and anecdotal cases. The black pen could also be used to add in a number of possible diagnoses which have not been discussed or refuted by the authors.

Despite the above criticisms, the book is an essential for the diving physician and paramedic, but should be read in conjunction with other diving medical texts, as the authors so correctly state in the preface.

As I am one of the contributors I should record my involvement with this text.

Carl Edmonds.

DANGEROUS MARINE CREATURES
Dr Carl Edmonds
ISBN 0 7301 0214 9
First Edition 1989
Reed Books Pty. Ltd., 2 Aquatic Drive, Frenchs Forest,
New South Wales 2086, Australia.
Distributed by Gordon and Gotch Ltd.
Price $ Aust 24.95.

About 15 years ago Dr Carl Edmonds published "Dangerous Marine Animals of the Indo-Pacific Region", an illustrated guide to all the local dangers and the treatments required, which was an immediate success with intelligent divers. It has been unobtainable for some years.

Now Dr Edmonds has produced "Dangerous Marine Creatures" a lavishly illustrated, hard cover, book for all those who swim, dive and paddle in the sea and for those who fish for and eat sea creatures. Most would expect that trauma would occupy the largest part of the book, but the emphasis is on stinging creatures (72 pages), followed by those creatures which cause physical injury (37 pages), those which poison the eater (27 pages)and those that cause infections and dermatitis (11 pages). A fifth section provides five appendices, dealing with cardiopulmonary resuscitation, the use of tourniquets, ligatures (a rather suprising inclusion) and pressure bandages, the use of local anaesthetic agents, the treatment of allergy and anaphylaxis, the use of antivenoms and a summary of venoms and toxins to be found in marine creatures. The whole is rounded off with a list of references and recommended reading. There is an adequate index including such things as Polar Bears (whose livers are likely to give the eater Vitamin A poisoning) and amoebic encephalitis, neither of which spring to mind as being likely to be included in a book with a lion fish, a shark and a scorpion fish on the cover.

The book opens with a disclaimer where Carl Edmonds' wry humour shifts the blame for everything except the jokes to other shoulders. The humour surfaces throughout the book. On page 85 while discussing the removal of *Chironex* tentacles it is suggested that "Businessmen can use

plastic credit cards" to pare the tentacles off the skin.

The general layout is a brief summary of whatever group of animals is being discussed emphasing those known to be dangerous. This is followed by a summary of the clinical features written for the layman. The first aid treatment follows and then comes the medical treatment, on the realistic assumption that most doctors know nothing of marine medicine. On page 12 is found "In the event of an injury from a marine animals, this book should accompany the patient, and be made available to the medical practitioner who is responsible for management." And finally each description ends with advice on how to avoid the problem.

The reader's interest is held at all times, especially by the extracts from Dee Scarr's Scuba Diary. She is a dive guide at Bonaire in the Caribbean who touches every sea creature that she can. She even stroked bristle worms without being damaged. Presumably this was because she never stressed the worms enough for them to raise their bristles.

Readers of the SPUMS Journal will recognise the illustration accompanying the description of Scombroid poisoning. For this reviewer the presence of this picture offsets the disappointment of finding only "Photos of fish causing tetradotoxin poisoning" as the caption to illustrations of three different fish (one of which appears to have scales) on page 147.

This book should be in the library of everyone who goes diving in the tropics, of everyone who teaches diving, and of everyone who may have to treat injured divers or those who have acquired seafood poisoning. It should be in the luggage of everyone going on a diving holiday or to a tropical or subtropical island. Those categories must include very member of SPUMS !

John Knight.

ARTICLES OF INTEREST FROM OTHER JOURNALS

LOSING YOUR LUNCH UNDERWATER

Two Techniques Toward Safe Sickness

If you have to vomit underwater, it can be extremely hazardous. If you remove the regulator to throw up, you will probably inhale water due to involuntary gasping. If you try to vomit through the regulator, you may clog it and render it inoperable.

You are best off if you carry an alternate air source. You can throw up through one regulator and breathe through another. The value to your buddy of the second stage into which you vomited will be questionable, but at least you will be safe.

If you have only one regulator, remove it from your mouth, and locate it tightly against one corner of your mouth. Depress the purge button completely to obtain maximum free flow and hold it that way. This should allow you to vomit into the water without clogging the regulator yet still get air when you gasp.

I have watched many people on the surface throw up and then go diving, claiming they are fine once they empty their stomachs. If you are a "one tosser" and this works for you, that is fine. But too many people are prone to throw up more than once. If the second or third time occurs underwater, it could be serious. Following the procedures outlined above can help render the process safe. Unfortunately you cannot practice these skills effectively until you require them, but it is better to try one of these than to take any other actions.

Of course, the best way to deal with nausea is to prevent it. Too many people go diving without proper nourishment. When you arise in the morning, your stomach is acidic. If all you put into it is coffee or orange juice, you are compounding the problem and asking for nausea. Try eating cereals, pancakes or french toast and see if your queasiness is less than usual.

If you still become nauseous, try nonprescription seasickness medications until you find one that is effective, yet has no side effects. Strong ginger tea works for some people. Seabands that use "acupressure" (See *Undercurrent* March, 1989) are effective for many people, while producing no side effects whatsoever. Try Pepto Bismol to settle your stomach. When the boat is underway, ist in the center of it in fresh air with your back and head against a wall and watch the horizon. This simple procedure helps many who are prone to *mal de mer.*

The author, Dennis Graver, is the Director of Education for NAUI.

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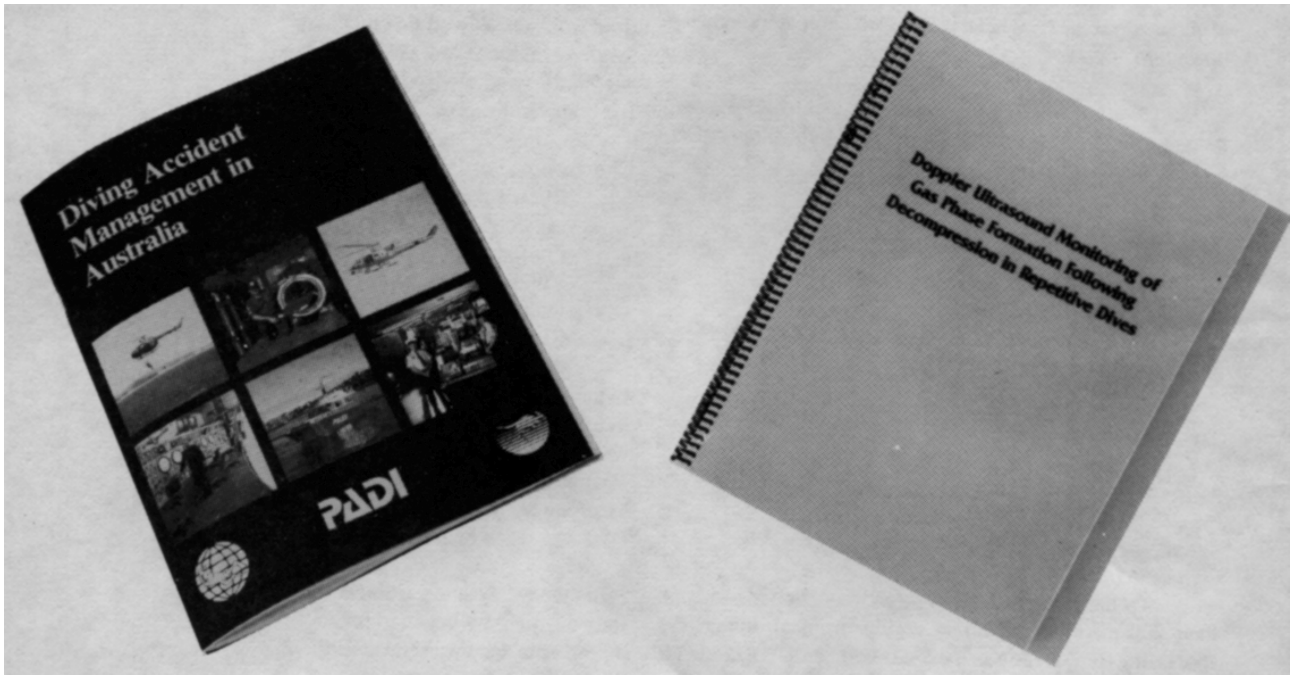

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