TREATMENT AND RESULTS OF THIRTY HYPERBARIC CASES AT THE RECOMPRESSION FACILITY HMAS STIRLING

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The therapeutic recompression facility at HMAS STIRLING in Western Australia was opened in November 1984. Since that time it has treated nearly forty diving and hyperbaric cases from the Western Australian area. This article is a review of the first thirty cases treated there.

BACKGROUND OF PATIENTS

In this survey, we have looked at the factors of age, sex, diving experience, training and military status. (See Tables 1 to 5)

TABLE 1

AGE OF PATIENTS

AGE	DIVING CASES	CARBON MONOXIDE (CO) POISONING
10-20	1 (3.25%)	1 (50%)
20-30	12 (43%)	0
30-40	12 (43%)	1 (50%)
40-50	2 (7.5%)	0
> 50	1 (3.25%)	0

TABLE 2

SEX

SEX	DIVING CASES	CO POISONING
Male	23 (82%)	1 (50%)
Female	5 (18%)	1 (50%)

TABLE 3

DIVING EXPERIENCE

EXPERIENCE	NUMBER	%
During Training	4	141
Less Than 1 Year	5	18%
1 - 10 Years	10	36%
Greater Than 10 Years	8	28.5%
Unknown	1	3.5%

TABLE 4

DIVE TRAINING

TRAINING	NUMBER	%
Self-taught	8	29%
Dive Training	18	64%
- Commercial	6	21%
- Dive Clubs	2	7%
- Unknown	10	36%
Professional	2	7%

TABLE 5

STATUS OF DIVERS

STATUS	NUMBER	%
Military	1	3.5%
Civilian	27	96.5%

PRE-DISPOSITION TO DECOMPRESSION SICKNESS

In this review, we look at the factors of omitted or insufficient decompression stops and of repetitive dives (see Table 6).

Using the Royal Navy Physiological Laboratory Tables as a reference, 88 per cent of the DCS patients either omitted completely or performed insufficient decompression stops. 64 per cent were also repetitive diving. Only 12 per cent actually obeyed the tables.

TABLE 6

PREDISPOSING FACTORS IN DCS CASES

FACTORS	NUMBER	%
Insufficient or No Decompression Stops	22	88%
Repetitive Diving	16	64%
Within Diving Tables	3	12%

PRESENTING SYMPTOMS

These symptoms were wide and varied, as expected. The most common symptom seen in the decompression sickness (DCS) cases was joint pain (28 cases). This was notably shoulder and neck pain (15 cases), followed by knee pain (5 cases), and elbow pain (4 cases). Other important symptoms include tingling in the extremities (6 cases), paraesthesia (9 cases) and general symptoms of malaise, which included light-headedness, headache, lethargy and nausea (21 cases).

In cerebral arterial gas embolism (CAGE), the most common symptoms were disorientation and decrease in consciousness, whilst in carbon monoxide (CO) poisoning, the most common symptoms were drowsiness and memory disturbances.

DELAY BETWEEN SYMPTOMS AND TREATMENT

In many cases long distances must be travelled before reaching the chamber. In this survey, we looked at the delay between symptoms and recompression treatment (see Table 7).

TABLE 7

DELAY BETWEEN SYMPTOMS AND TREATMENT

DELAY	CAGE	DCS	CO POISONING
Less Than Six Hours	2	-	
Six To 24 Hours	-	3	1
One To Seven Days	1	15	1
One To Two Weeks	-	2	
Two to Four Weeks	-	4	
Greater Than One Month	-	1	

DIAGNOSIS

There were 2 cases of CO poisoning, 3 cases of CAGE and 25 cases of DCS. The DCS cases were 6 neurological (3 cerebral, 2 spinal and 1 vestibulo-cochlear), 17 musculo-skeletal (10 with peripheral nervous system involvement and 7 without), 1 respiratory, 1 skin and 1 gastro-intestinal.

TREATMENT

The initial treatment given to each of the patients was reviewed. This is a summary of the chamber treatments only, and not of the first aid or adjuvant therapies (see Tables 8 and 9).

Of the 30 patients treated, there were 19 who relapsed (63 per cent). These included 18 cases of DCS and 1 case of CAGE. These cases were treated with RN Table 61 (75 per cent), RN Table 62 (8 per cent) and 5 Metre Soaks (17 per cent). (See treatment table diagram in the next column).

At discharge, complete resolution was achieved in 24 cases (80 per cent) while six had some residual deficit. This was mainly mild joint pain or paraesthesia in the extremities. These six cases included one delayed treatment of a CAGE case and five cases of DCS with delays of between 4 days and eight weeks.

COMMENTS

The majority of the patients in this series were males between the ages of 20 and 40 years, with a reasonable amount of experience. They had mainly been dive-trained but a significant number had been self-taught, and this was especially true amongst the pearl and abalone divers. They were almost entirely civilian divers.

TABLE 8

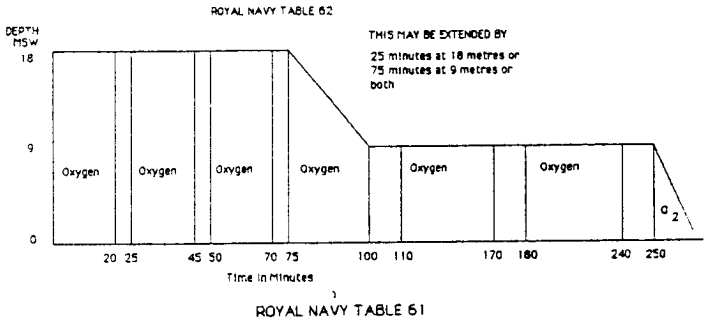
TREATMENT USED

TABLE	CAGE	DCS	CO POISONING
RN 62	-	13	1
RN 62 E	1	11	-
RN 54	1	-	-
OTHERS	TABLE 6 (MALDIVES)	TABLE 6 (THAILAND)	15 M O_2 SOAK

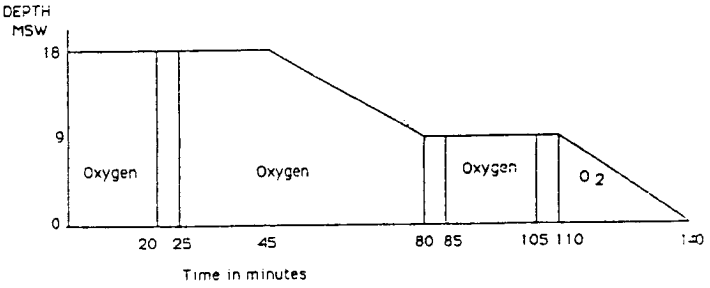
TABLE 9

DELAY, RELAPSE AND RESOLUTION RATES

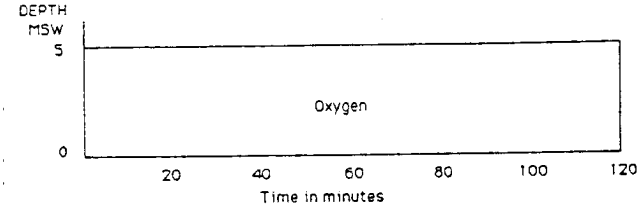
DISEASE	NO.	DELAY	RELAPSE	RESOLUTION
CAGE	2	< 6 Hours	0	Yes (2)
	1	1-7 Days	1	No (1)
DCS	3	< 24 Hours	2	Yes (3)
	15	1-7 Days	10	Yes (12)
				No (3)
	2	1-2 Weeks	2	Yes (2)
	4	2-4 Weeks	3	Yes (3)
				No (1)
	1	> 4 Weeks	1	No (1)



ROYAL NAVY TABLE 61



FIVE METRE SOAK



Adherence to the diving tables was very poor and as there is a risk of decompression sickness even whilst staying within the tables (12 per cent of those this series said that they had dived within the tables), the risks of going outside the tables are obvious. Repetitive diving only helps to exacerbate these risks.

CAGE was treated generally within six hours. The one exception to that was initially treated in a chamber in the Maldives twenty-four hours after the onset of symptoms. DCS, however, presented usually at 2 to 3 days with substantial numbers up to 4 weeks and one at 2 months.

The majority of the cases treated were musculo-skeletal decompression sickness (57 per cent), though it is noted that three cases of CAGE (10 per cent) occurred during this period.

Treatment was usually with a RN Table 62 or an extended RN Table 62. Although initial improvement was usually good, 63 per cent relapsed. This was probably due to the delay in commencement of treatment. Of these relapses, 67 per cent resolved completely with further treatments.

This review illustrates the cross-section of cases that could be expected to present at a treatment facility. The question of delay before the patient presents is an important one, regardless of whether it is due to travelling delays or patient reticence at presenting. As it directly affects the chance of relapse and eventual recovery, efforts should be made to educate divers to keep treatment delays to a minimum.

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BEER, BUBBLES AND THE BENDS

THE BIOPHYSICS OF BUBBLE FORMATION IN DECOMPRESSION SICKNESS

HP De Decker

NOTES

It would be useful if the reader could pour himself (herself) a tall glass of slightly chilled beer (for illustrative purposes!).

All pressures are given as atmospheres absolute (ATA). Although not strictly SI, this unit has been used, following its popularity in diving literature, because of the ease of conversion from depth to ATA. Each 10m increment in depth equals an increase in pressure of 1 ATA. For the purists 1 ATA = 1 $kg.cm^2$.

I am indebted to Paul Hanekom of the Research Diving Unit, Department of Oceanography, University of Capt Town for the use of his library and for stimulating my interest in diving physiology.

I am fascinated by beer, and not only because of its inebriating effect. To me it illustrates some of the most fundamental aspects of that most dreaded of divers' diseases, the bends. Decompression sickness (DCS) or the bends, is an illness that follows a decrease in environmental pressure which is sufficient to cause the formation of bubbles from inert gases dissolved in the body tissues. It occurs in pilots and tunnel workers suddenly exposed to a large decrease in pressure. It is mainly seen, however, in divers who return from depths where the increased hydrostatic pressures cause high partial pressures of nitrogen in their tissues. This extra nitrogen is then released as bubbles if the ascent is too rapid. A beer, of course, is far from being a complete model of this complex syndrome with its cascading haematological effects, but it serves a useful purpose in illustrating the most fundamental aspect of DCS, bubble formation. And that is what we will explore. Stare into the depth of your beer to where the bubbles appear as if by magic from a single spot in an unending rising string of pearls. How do they form? What hidden forces shape their burst into existence? How does this relate to the bends?

DECOMPRESSION SICKNESS

It has been known since the middle of the last century that DCS is an illness related to bubbles in the blood and tissues.[1] Before 1968, however, literature on bubble formation was virtually non-existent, mainly due to the difficulty of actually observing bubbles in vivo, and to the use of subjective endpoints such as pain, paralysis and other clinical manifestations.[2] Until quite recently, no direct observations of bubble formation had been made in vivo and the link between the bends and bubbles could only be inferred from post mortem investigations. This meagre evidence, however, was used to construct decompression tables by which divers currently calculate a safe ascent rate from any depth. Although the tables are usually effective for the prevention of DCS, this is obviously an unsatisfactory basis for its treatment. Effective treatment of the disease necessarily requires detailed knowledge of bubble formation as this is its initiating event. But before explaining how the bubbles form, we need to know what they are. For answers, we look at the bubbles in your beer, and then see if we can apply our knowledge to living tissues.

THE BUBBLES

Physics

Let us first determine the forces which act on a bubble in a liquid or in tissues. For a bubble to exist, the total gaseous pressure inside the bubble must be equal or greater than the crushing pressures exerted on it.[3] The crushing pressures are:

1. the ambient pressure (Pamb), which by Dalton's Law equals the sum of the constituent gas pressures, ie.
   $$Pamb = P_{N_2} + P_{O_2} + P_{CO_2} + P_{H_2O} \text{ etc} \quad (1)$$
2. the tissue pressure (Ptissue), or the pressure the tissue exerts in resistance to deformation, and
3. the pressure due to surface tension (Py) of the bubble surface. This is given by Laplace's law as the relation between surface tension (y) and the radius of the bubble (r):
   $$P = 2y/r \quad (2)$$

It is obvious from this relationship that Py is negligible in large bubbles, but that very small bubbles are