should this be required, as well as giving the photographers a bubble free shallow water dive!

In conclusion, whilst plastic bags are universally available you may have a little trouble obtaining very large ones. I get mine from Transpak Industries in Glenfield, Auckland (ISD phone 64-9-4444823 and ask for Frank Richardson) and I should like to take this opportunity of thanking Allan Bloomfield, their Managing Director who kindly supplied bags for both my trips to the Kermadec Islands.

Figure 3. Warming the winter diver by filling the wet suit with heated water.

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THE ZIERING ALPHABET

AN APOLOGY

Owing to a printing error the name of Tony Catley, who drew the illustrations of the Ziering alphabet, was omitted.

HYPERBARIC OXYGEN THERAPY RATIONALE FOR TREATMENT OF DIVING ACCIDENTS

Andrew A Pilmanis

SUMMARY

Use of a hyperbaric chamber with 100 per cent 0_2 by mask is the currently accepted treatment for diving related air embolism and decompression sickness. However, the basic rationale and goals of hyperbaric oxygen therapy are not universally understood by members of the diving community. This lack of understanding often leads to attempts at inadequate and misdirected measures resulting in, at the least, delays to proper treatment and, at the worst, to harmful measures. This paper is an attempt to clarify the currently accepted basic rationale and goals of recompression oxygen therapy for diving accidents.

In 1964, Goodman and Workman[1] introduced the hyperbaric oxygen treatment tables for decompression sickness (DCS) and air embolism. These tables are currently almost universally used for treatment of these ailments. The success of these tables is generally recognized. The treatment method involves the use of 100 per cent 0_2 by mask delivered intermittently inside a hyperbaric chamber at various pressures for various times depending on the severity of the condition.[2,3]

The University of Southern California Catalina Marine Science Center hyperbaric Chamber facility was involved with 554 diving accident incidents between 1974 and 1985 (Table 1). There were 179 decompression sickness patients and 101 air embolism patients treated in the chamber according to the above mentioned treatment procedures.

It was observed that, in general, a number of misconceptions existed in the diving community about the rationale or objectives of these treatment procedures. It is the purpose of this paper to clearly define this rationale as perceived by the author. These are not original concepts, and are not universally presented in this manner. However, the concepts are, in general, accepted by the majority of the hyperbaric medicine field. There is obviously overlap among the five goals of recompression therapy listed. In addition, the order of the listing does not necessarily reflect priority or level of importance.

GOALS OF RECOMPRESSION THERAPY

1. Bubble Size Reduction

Since all divers are taught the basic pressure/ volume/ density relationships defined by Boyle's Law, this goal is the most obvious. Although this aspect has definite beneficial results (especially in the case of air embolism), as is apparent in Figure 1, the Boyle's Law effect is not nearly as dramatic when viewed from the standpoint of the decrease in the diameter of a sphere rather than simply by volume reduction, ie. a bubble in the body. For example, at 50 metres of sea water (msw), 165 feet of sea water (fsw), which is the initial treatment depth for air embolism, the volume in a bubble has been reduced to 1/6. However, the diameter of that bubble has only decreased to 55 per cent. Certainly that 45 per cent reduction is beneficial in permitting blood flow to previously occluded tissues. However, by itself this factor will not cure the problem.

CATALINA MARINE SCIENCE CENTRE HYPERBARIC CHAMBER

	1974	1975	1976	1977	1978	1979	1980	1981	1982	1983	1984	TOTAL
Total Hyperbaric Chamber Treatments	3	21	35	31	45	38	38	47	93	70	68	489
Decompression Sickness	0	6	13	15	7	16	13	26	32	28	23	179
Air Embolism	2	7	5	10	15	13	11	7	6	14	11	101
Clinical HBO	0	1	0	2	4	0	0	0	0	1	0	8
Retreatments	1	7	17	4	19	9	14	14	55	27	34	201
Chamber Treatment Not Required	0	3	5	4	6	9	14	4	10	7	3	65
Total Number of Treatments	3	24	40	35	51	47	52	51	103	77	71	554

TABLE 1. Catalina chamber 10-year diving accident experience.

DEPTH IN FEET	PRESSURE IN ATA	RELATIVE VOLUME	RELATIVE DIAMETER
0	1	100.0%	100.0%
33	2	50.0%	79.3%
66	3	33.3%	69.3%
99	4	25.0%	63.0%
132	5	20.0%	58.0%
165	6	16.6%	55.0%
297	10	10.0%	46.2%

FIGURE 1. Bubble volume and diameter changes with pressure changes.[3]

Often divers attempt to "treat themselves" by going back underwater with a fresh tank of air. Ignoring the environmental stress aspects associated with such practice, it can be seen from Figure 1 that the beneficial result will be minimal. There are two detrimental aspects to this practice that make it categorically unacceptable:

a) The recompression on air is simply a "repetitive dive" adding even more nitrogen to an already supersaturated system. Sooner or later the diver must surface, resulting in re-expansion of the existing bubbles, and, perhaps, in the formation of new ones.

b) There is delay in transportation to a proper treatment facility.

2. Hyperbaric Oxygenation of Hypoxic Tissues

Reduction of blood flow associated with bubbles in the blood vessels results in tissue hypoxia. The majority of the clinical manifestations of the two diseases are the result of this tissue hypoxia. Normal alveolar PO_2 is approximately 100 mm Hg. At 18 msw (60 fsw) in a chamber breathing 100 per cent oxygen, the alveolar PO_2 is approximately 2000 mm Hg. This would appear to be of obvious benefit to the hypoxic tissues. However, there are two factors that reduce the perceived major benefit:

a) High oxygen levels can have little effect on hypoxic tissues if the oxygen-rich blood is not delivered to those tissues, ie. bubbles must not only be reduced in size but be totally removed for the full oxygen effect to occur,

b) Oxygen molecules are relatively large and, therefore, do not diffuse readily. In hypoxic swollen tissues with large diffusion distances there may be limited benefit from extremely high inhaled O_2 levels. However, in combination with the other aspects, ie. bubble reduction, this aspect can be very beneficial.

3. Bubble Resolution

The ultimate objective of recompression therapy is to eliminate the source of the disease, ie. the bubbles. Figure 2 illustrates this mechanism. Bubble resolution is based on nitrogen diffusion out of the bubble, into the surrounding tissues, into the blood, into the alveoli, and ultimately exhaled from the lungs. The rate of diffusion is directed by the nitrogen partial pressure gradient. If left untreated, bubbles may remain intact in the body for weeks due to the small gradient between the bubble and the surrounding nitrogen-rich tissues. If compressed in a chamber or underwater, breathing air (80 per cent nitrogen),

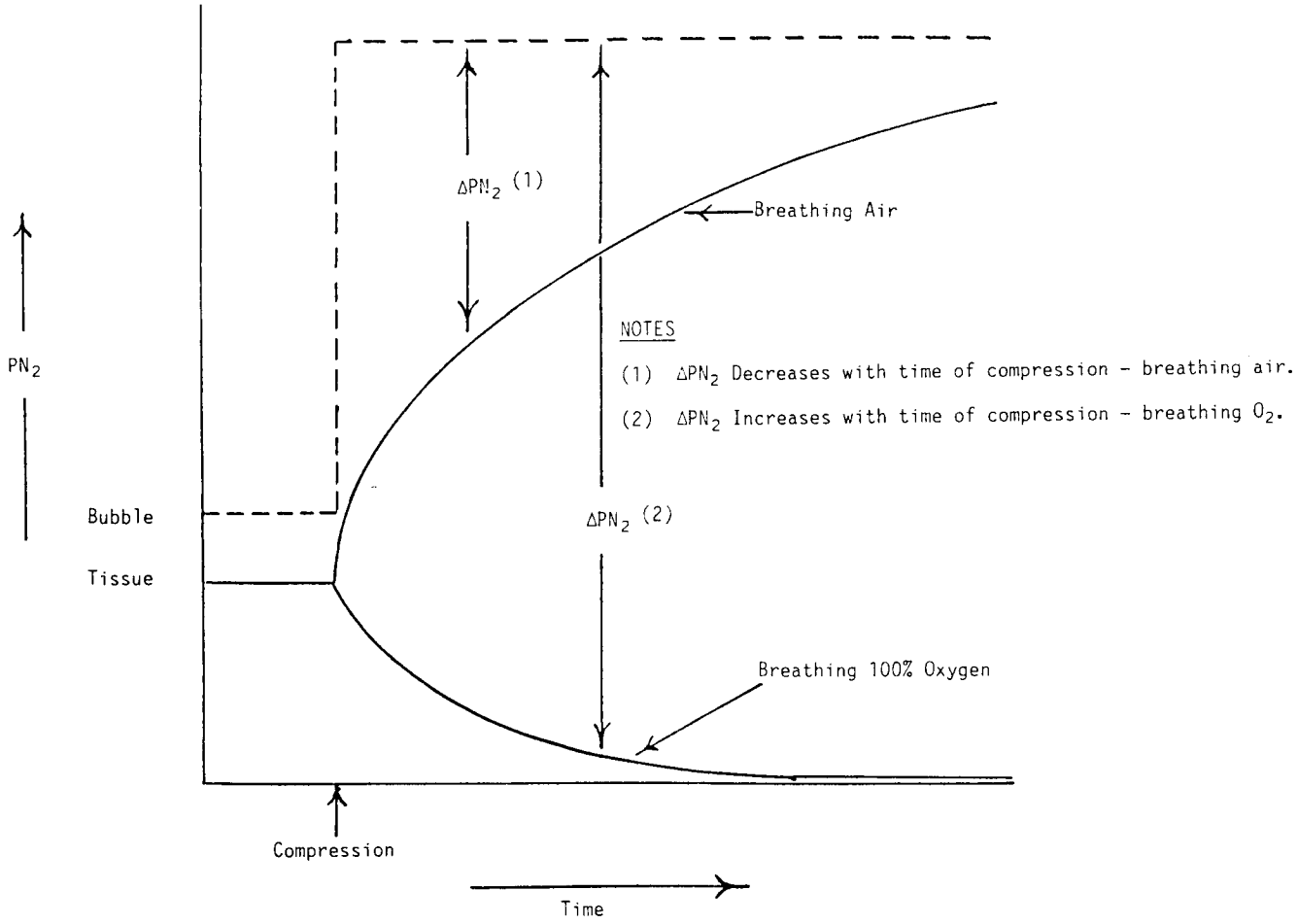

FIGURE 2. Effect of compression on the PN_2 gradient from a bubble using air and 100% O_2.

initially there is an increased nitrogen gradient promoting bubble resolution. However, with time, that gradient decreases until it stabilizes at the same small difference found at sea level prior to compression. But now the whole body has undergone a nitrogen uptake process resulting at a much higher base level of nitrogen in the tissues. Sooner or later, the patient must surface and "off load" that nitrogen. When this is done the bubble, which has not necessarily resolved, can re-expand. There have been high treatment failure rates with the old air treatment tables. Now contrast the air-breathing situation with breathing 100 per cent oxygen in the chamber. Instead of a nitrogen uptake curve as in the case of breathing air, there is a nitrogen elimination curve when breathing 100 per cent oxygen. The partial pressure gradient of nitrogen between the bubble and the surrounding tissues increases to a maximum with time when breathing oxygen. This, in turn, drives the nitrogen molecules out of the bubble, to the lungs, and gradually reduces the size of the bubble until it collapses and disappears, ie. curing the disease. This process of course, takes time ie. the 5-6 hour treatment tables.

Although to a lesser degree, this is the same reason why sea level 100 per cent oxygen breathing by the patient is the primary first aid measure during transport to a chamber facility. 100 per cent oxygen units should be available at all dive sites.

Without treatment there is a slight difference between tissues and bubble partial pressures of nitrogen. There is a slight gradient out of the bubble. Eventually the bubble will resolve. I do not know whether anybody can say accurately how long it takes a bubble to resolve in the body without treatment. I believe that it takes weeks not days. We treated a fellow three or four weeks after his dive and upon pressurization to 18 msw (60 feet) he had immediate resolution. How can one explain that other than the bubbles were still there? That is an assumption but some trends point in that direction. I think bubbles last for weeks in the body. Now think about that for a minute. Decompression tables, repetitive diving tables, all the tables one uses have only one function, to prevent bubble formation. The tables assume that the diver does not have bubbles before the dive. The table is to prevent bubble formation. If one already has bubbles decompression tables are not necessarily valid. We have had about seven cases where after a great deal of looking through the history of their dives, going back a month, even two months, we finally found the insult. They had insults in dives up to three months before. They had been bent for three months. Those bubbles never resolved. They had been there the whole time and had been slowly gaining nitrogen from subsequent dives. If the bubble is in a tissue that generates that is fine. But if it is in the spinal cord or the bone marrow or brain or the liver, that is a different matter. Post mortems on goats with spinal DCS and

on experienced divers who had recovered from spinal DCS showed a tremendous number of lesions in the spinal cord.

4. Rehydration

There are a number of sources of dehydration associated with diving:

a) Immersion diuresis*

b) Breathing dry air

c) Exercise

d) Cold

e) Decompression sickness or air embolism

* In Latin urinare means "to dive" and, in the past, divers have been referred to as "urinators".

It is beneficial to give fluids to patients with air embolism or decompression sickness either by mouth or intravenously. Diuretics such as alcohol or caffeine-containing fluids should be avoided.

THE VICIOUS CYCLES OF DECOMPRESSION SICKNESS

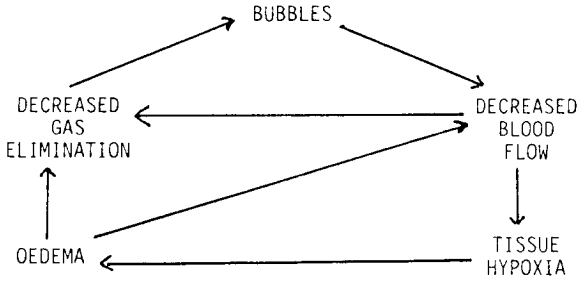

FIGURE 3. Oedema problems in delayed diving accidents.

5. Reduction of Neurological Oedema (Figure 3)

Tissue hypoxia from any source leads to swelling or oedema. Of particular concern in decompression and air embolism are the neurological tissues, ie. the brain and the spinal cord. Bubbles in these regions obstruct blood flow and result in oedema. This oedema is difficult to reverse and treat. The longer the delay to treatment, the worse this situation becomes. Currently, two methods are used to reduce neurological oedema related to diving problems:

a) Hyperbaric oxygen has been shown to reduce oedema through vasoconstriction.

b) Steroids, specifically dexamethasone (Decadron), are given in high dosage to reduce oedema.

CONCLUSIONS

1. Do not take a diver with decompression sickness or air embolism back underwater for treatment.
2. Administer 100% oxygen by mask and give fluids to the patient during transport.
3. Transport as rapidly as possible to a properly equipped and staffed double-lock multiplace hyperbaric chamber facility.
4. Treat the patient in such a chamber with pressure and 100 per cent oxygen by mask according to standard oxygen treatment tables.

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SCOPODERM AND DIVER PERFORMANCE

Mike Davis

I was very lucky in my first few years of diving as an undergraduate, in the early 1960's, to be involved in most of the field projects undertaken by Alan Baddeley, who is now Director of the Medical Research Council (MRC) Applied Psychology Unit in Cambridge. Alan, in those days, was just beginning to look at some of the effects of diving on diver performance, in particular, nitrogen narcosis. So I learnt a bit, albeit rather superficially, about the problems of applied psychology experiments in the field, problems which are pretty considerable. The overall concept of 'diver performance', that it was not just nitrogen narcosis we were worried about, but that there were a whole variety of components that contributed, became increasingly understood by a number of groups around the world at that time. One of the problems about diver performance, probably the biggest, is how to assess it under field conditions. For many years there were really two main schools of thought on how to approach this, either to use an array of simple tasks which define different areas of performance, for instance, manipulative and various cognitive and reasoning tasks, or to go for the analysis of complex tasks like assembling an oil well head. A common feature of all these studies has been how much greater is the effect seen in open water compared to simulated chamber dives.

When I arrived in Christchurch in the late 1970's to find myself running a recompression chamber with some other enthusiasts, I looked at one or two small areas of research that we might tackle. One was to go back to what I had been involved with a decade earlier,