DECOMPRESSION SICKNESS IN DIVING

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SYNOPSIS

A clinical series of 115 cases of decompression sickness is presented. A very brief review of literature and the current attitudes towards decompression sickness is given, together with a discussion of the various clinical manifestations. This disease as it affects civilian and sports divers, is much more diverse and serious than is described in the traditional naval series. The reasons for this are propounded and recommendations for greater diving safety are made. The efficiency of current therapy is assessed and some suggestions are made regarding the use of oxygen recompression therapy under water.

INTRODUCTION

Following the development of the air pump by Von Guericke in 1650, Robert Boyle was able to expose animals to decompression. In 1670 he reported these experiments and included the first description of decompression sickness a bubble moving to and fro in the watery humor of the eye of the viper. Hoppe-Seyler repeated the experiments of Boyle, and in 1857 he described the obstruction of pulmonary vessels by bubbles and the inability of the heart to function adequately under those conditions. He also recommended recompression to remedy this. Le Roy de Mericourt in 1869, and Gal in 1872 described an occupational disease in sponge divers, which was also attributed to the breathing of compressed air and was equated with Caisson disease. Paul Bert in 1878 demonstrated in a most conclusive manner that the decompression sickness is primarily the result of an inert gas (nitrogen in the case of compressed air divers and caisson workers) which had been dissolved in the blood and tissues of the body, being released during or following the return to normal pressures. Gersh and Catchpole (1951) in reviewing the literature and their own work on the neurological manifestations of decompression sickness, demonstrated that gas bubbles formed in circulating blood after a short latent period from the time decompression commences. Most bubbles are filtered by the lungs; some pass through the lungs and reach the central nervous system and other organs and occlude arterioles of the same calibre. The clinical manifestations depend on the site of the vascular obstruction and collateral supply, and are largely a matter of chance. The early occurrence of venous bubbles, often during the diver's ascent and their subsequent emergence as a diminished quantity in the arterial system when decompression sickness develops, has been adequately verified by the ultrasonic doppler techniques in both animals and man.

Haldane, Boycott and Damant in 1907 demonstrated a technique of decompression whereby the diver ascends in a series of stages. This allows the gradual exhalation of the accumulated inert gas, thus reducing or preventing the number of bubbles able to form within the diver's body. Recompression therapy was introduced for the treatment of decompression sickness, and this was modified by Goodman and Workman *et al* (1965) with the introduction of 100% oxygen to hasten the elimination of inert gas from the body, while minimally compressing the diver in a recompression chamber to reduce the size of the gas bubbles causing the clinical symptoms. More recently the use of oxygen mixtures to bridge the compression gap between the air recompression therapy tables and the oxygen recompression therapy tables, allows greater flexibility in the treatment of this disorder (Edmonds *et al*, 1976). The very recent introduction of under-water oxygen recompression therapy has resulted in more rapid therapy being instituted in remote localities.

A clinical classification was presented (Golding *et al*, 1960) as an attempt to differentiate non-serious and serious cases, so that identification prognosis and therapy could be more appropriately standardised. This clinical classification of Type I and Type II decompression sickness is not wholly satisfactory. The classification was neither defined nor applied in the same spirit as it was initiated. Type I is defined as pain-only decompression sickness or joint bends. Type II includes those presenting with symptoms other than pain, or with abnormal physical signs. The central neurological, spinal, cardiovascular, respiratory and gastrointestinal manifestations are potentially serious. Naval (Bennett and Elliott, 1969; Rivera, 1963; Slark, 1962) and recent Caisson series (Bennett and Elliot, 1969; Griffiths, 1969) have had a disproportionate dominance of joint bends, compared to the civilian series (Erde and

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Edmonds 1975). In the latter the neurological and cardiorespiratory symptoms are much more frequent. To support or specify this variation in severity between the quoted Naval series and the rarely documented civilian cases, this relatively large series of civilian cases, are now examined. The only civilian series of comparable size was the Hawaiian group, described by Erde and Edmonds.

MATERIAL AND METHODS

A series of cases of decompression sickness were referred for treatment to two major recompression therapy centres in the Indo-Pacific region, the Singapore Armed Forces based at Singapore and the Royal Australian Navy School of Underwater Medicine at Sydney, Australia. The 115 consecutive cases included 40 from Singapore and 75 from Sydney. Records were made of the diving history, the clinical features, the response to treatment and other interrelated factors.

Inclusion of cases of decompression sickness in the series required either indisputable signs of this disorder or the development of clinical symptoms during or after ascent, which were relieved or cured by recompression therapy. Cases of pulmonary barotrauma were excluded from the survey i.e. those cases with haemoptysis, pleuritic pain, pneumothorax, mediastinal or cervical surgical emphysema etc.

In assessing the result of treatment, the following grading system was used. Complete cure—4; Almost complete cure—3; Definitely improved—2; No definite change—1; Clinically deteriorated or died—0. The major treatment employed included—the Air Tables, 5A, 5B, 5C, 5D, 5E in the Australian Navy Diving Manual, these are equatable with tables 1, 2, 3 and 4 of the U.S. Navy Diving Manual; The Oxygen Treatment Tables, 6A, 6B, of the Australian Navy Diving Manual, which are equatable with Tables 5 and 6 of the U.S. Navy Manual; the high oxygen pressure tables, both in a compression chamber and underwater (Edmonds *et al*, 1976) and finally the use of oxygen at atmospheric pressure.

Symptomatic treatment was administered routinely, according to the severity of the case. Thus many received intravenous infusions, urinary catheterisation, steroids, anti-epileptic and tranquilliser drugs for neurological cases, as well as electrodiagnostic and clinical monitoring procedures.

RESULTS

Most of the cases were amateur or sports divers, fishermen divers, pearl divers, abalone divers or other locally employed divers. All were using compressed air as the medium, either in the form of self contained underwater breathing apparatus (SCUBA) or by the use of a line taking the air from a mechanical compressor on the surface or from cylinders also at sea level i.e. a surface supply breathing apparatus (SSBA). Table I gives a description of the population involved and the type of dive profile.

As a generalisation, the dives were far in excess of those allowed by recompression tables. Only 24% attempted some form of decompression. As depicted in Table II, in 89% of dives there were sufficient data available to assess the decompression performed. The majority of both the Singapore and Australian groups exceeded the allowable duration underwater and did not perform adequate decompression staging. In 11% of cases there was insufficient information available to make a judgement in either direction. In those cases in which the dive was

TABLE I

	Mean	Standard Deviation	
Age	32.4	9.5	years
Depth	30	10.5	metres
Duration	120.6	112.1	minutes
Onset of Symptoms (from start of ascent)	33.1	48.0 *	minutes
Delay in Treatment (from start of symptoms)	50.9	40.4	hours

* not relevant due to extreme skew deviation

TABLE II

	Singapore	Australia	Total
Diver exceeded recommended tables	32 (80%)	55 (73%)	87 (76%)
Insufficient information available	1 (2.5%)	12 (16%)	13 (11%)
Dived in accordance with tables	7 (17.5%)	8 (11%)	15 (13%)
Total	40	75	

stated to be performed in accordance with the recommended tables, there is still some room for doubt—as both depths and duration were often merely estimations.

Table III gives the overall incidence of symptoms attributable to decompression sickness, and includes all those symptoms or signs which are recorded in the case reports. It does not in any way attempt to infer severity.

Table IV shows the predominant manifestations, this is described as either a Type I or Type II decompression sickness. Type I is usually designated as minor manifestation of decompression sickness, and Type II as a serious manifestation.

Type I Decompression Sickness

This is the less serious form of decompression sickness as regards morbidity and mortality, mainly affects the musculoskeletal system, and this is commonly termed "Bends". Of the total, 47% fell into this category, although 15% more cases had evidence of joint pains as well as more serious symptoms, thus placing them into the Type II decompression sickness group. As in most other series of decompression sickness affecting divers, the upper limbs were most affected, with the following order of predominance—shoulders, elbows, knees and hips. As in a previous series, when multiple joints

TABLE III

INCIDENCE OF SYMPTOMS N = 115

Musculoskeletal 71 (61.7%)		
Shoulder	56	48.7%
Elbows	42	36.5%
Arm	9	7.8%
Wrist	9	7.8%
Hand	1	.9%
Knee	21	18.3%
Hip	10	8.7%
Leg	9	7.8%
Thigh	7	6.1%
Ankle	3	2.6%
Neurological		
Cerebral	19	16.5%
Paresis and Paralysis	44	38.3%
Subjective sensory loss	59	51.3%
Loss of sensation	54	47.0%
Loss of proprioception	21	18.2%
Loss of bladder function	41	35.6%
Loss of bowel function	21	18.2%
Pain in spinal column	15	13.0%
Unconsciousness	23	20.0%
Inner Ear		
Vertigo	10	8.7%
Deafness	3	2.6%
Tinnitus	2	1.7%
Respiratory		
Chest pain	18	15.6%
Cough	12	10.4%
Dyspnoea	20	17.4%
Gastrointestinal		
Abdominal pain	18	15.7%
Nausea	25	21.7%
Generalised		
Malaise	31	27.0%
Dizziness	20	17.4%
Anorexia	6	5.2%
Fever	4	3.5%
Integumental		
Oedema	10	8.7%
Itching	9	7.8%
Rashes	6	5.2%

TABLE IV

PREDOMINANT DECOMPRESSION SICKNESS MANIFESTATIONS

Type I —	Decompression Sickness	54 cases	47%
Type II —	Decompression Sickness	61 cases	53%
	Cerebral	11 cases	10%
	Spinal	22 cases	19%
	Both spinal and cerebral	22 cases	19%
	Inner ear	3 cases	3%
	Cardiorespiratory	3 cases	3%

TABLE V

FREQUENCY OF RESPONSE TO THERAPY

Response (grade)	Singapore		Australia		Total
4	19	49%	52	72%	63%
3	12	31%	11	15%	20%
2	4	10%	5	7%	8%
1	4	10%	2	3%	6%
0	0		2	3%	2%

were involved, they tend to occur in neighbouring joints.

Type II Decompression Sickness

In the Singapore cases, spinal lesions dominated the clinical presentation. The Australian cases had a greater number of cerebral and cardiorespiratory manifestations, probably reflecting the closer proximity of the diving to the therapeutic facility. It was noted that cerebral and cardio-respiratory manifestations tend to occur soon after the completion of the dive, and in some cases resolve spontaneously. Sometimes spinal lesions supervene after a delay. Spinal cases are much less correctable by procrastination, and therefore are more likely to be transported long distances, taking a longer time. Aborted or inadequate water recompression therapy was often attempted, and usually served to aggravate clinical symptoms and result in unnecessary delay of treatment.

In the cases of Type II decompression sickness there are often other manifestations, which can be seen from Table III. Spinal cord involvement was the most likely cause of severe persistent disability from decompression sickness.

Time of Onset of Symptoms

Although the mean time between ascent and the first presentation of symptoms is 33.1 minutes, a standard deviation is not relevant as the distribution is strongly skewed. Twelve developed symptoms during the repetitive dive, with ascent or whilst staging. Thirty-one cases developed symptoms upon surfacing or very soon after. A further twenty-three within the first ten minutes—making a total of sixty-six decompression sickness cases developing within this time. The longest duration between ascent and the initial development of symptoms was 19 hours, and this particular case did respond well to recompression therapy. The second longest case developed symptoms 5 hours after ascent.

The comparison of the responses to treatment from different therapeutic regimes is a rough one, and definite conclusions cannot be drawn from this comparison, as the cases were not randomly selected for each regime. On the contrary, the severity of the case and the local conditions were paramount determining factors in the decision as to which therapeutic regime to apply. Thus oxygen would be administered "on the surface" at 1 ATA in a mild case of Type I decompression sickness of a long duration and distant from recompression facilities. Originally in the Singapore group there was a tendency to treat according to the Air Tables, although in later years the Workman's Oxygen Tables were applied. The Australian group were more often treated with the Australian high oxygen pressure Tables i.e. the maximum safe oxygen pressure administered either in a compression chamber, underwater or at 1 ATA.

In assessing the response to therapy the previous

TABLE VI

RESPONSE TO VARIOUS TREATMENTS

	Mean Response	Number
Air Tables	2.29	24
Workmen's O_2 Tables	2.31	59
Australian O_2 Tables	3.58	52
O_2 administered at 1 ATA	3.2	10

mentioned grading system is used i.e. Complete cure—4; Almost complete cure—3; Definite improvement—2; no definite change—1; clinically deteriorated or died—0. It is important to realise that the response is compared to the initial severity of the case.

The total number of regimes used exceeds the number of cases, because some cases responded insufficiently to one type and so required another. In the majority of cases oxygen was also used intermittently following the recompression regime, to avoid or diminish the recurrence of minor symptoms. In many of the paraplegic cases i.e. those with spinal decompression sickness, hyperbaric oxygenation was also used subsequently to the initial recompression therapy. This regime is used both in the Singapore and Australian areas with apparently good clinical results.

When observing the time required for full recovery, it was observed that 60% were fully recovered within 24 hours of initiating treatment. Another 17% recovered within one month, 14% more within the year. Approximately 7% were permanently affected, and 2% died from the decompression sickness.

DISCUSSION

The more serious nature of civilian cases, as compared to the Naval series was again evident. The dramatic superiority of Workman's Oxygen Tables over the conventional Air Tables was not as evident as in the Naval series. Perhaps this was because of the more established and serious nature of the civilian cases. This in turn may be due to the specific problems encountered amongst a non-disciplined diving population. These problems include:—

Divers who are physically unsuited for the type of dive they are performing e.g. due to obesity etc.

The unawareness of correct diving procedures e.g. decompression staging, dive planning etc.

Using inadequate equipment, often without depth gauges or underwater watches for decompression.

No facilities for decompression.

Insufficient gas supplies etc.

The performance of rapid ascents, there being frequently a history of an emergency or unplanned ascent.

Unnecessary delays in early treatment.

The local administrative authority being unprepared for diving accidents, aggravating the delays.

Inadequate local recompression treatment facilities.

The advantages of Workman's Oxygen Tables to the operators were very evident, requiring less time and resulting in less inconvenience to the therapists. Edmonds oxygen underwater treatment regime was of use in remote localities, when the divers were prepared for the eventuality of decompression sickness. In this regime the patient is submerged with a supply of oxygen from the surface, comprising a large (220 cubic ft.) oxygen cylinder with the hose extending to a maximum depth of 9 metres underwater, and having a demand valve attached. The demand valve fits through a full face mask, thus reducing the dangers of unconsciousness, vomiting underwater and drowning. A companion diver is required, but because of the depth there is no problem regarding decompression sickness in the assistants. The diver sits on a stage or a weighted shot rope hanging over the side of the boat. If possible a sheltered lagoon or harbour is preferred to reduce the incidence of sea sickness in both the diver and the attendant. After 30-120 minutes the diver is brought to the surface at a rate of 12 minutes per metre, by hauling the shot rope upwards. The diver continues breathing oxygen throughout and is left on intermittent oxygen (one hour on, one hour off,) when he reaches the surface to reduce the incidence of recurrence in most cases. This regime will usually result in a considerable relief or complete cure, but even at the worst the diver is given some hours of nitrogen elimination, a reduction of the severity of the clinical complications, and time in which transport can be arranged to the nearest recompression facility.

When transport is obtained it is imperative that it is achieved with as little physical disturbances as possible, and carried out at a pressure equivalent to sea level. Both the physical disturbance and the exposure to lower than atmospheric pressure will increase the volume of gas bubbles associated with decompression sickness. Many civilian and commercial aircraft can be pressurised to 1 ATA and are thus suitable for the trip for diving casualities. During transportation humidified oxygen may be administered to the patient. All other treatment procedures are based on general medical principles. The blood volume deficit should be remedied, cerebral and spinal oedema reduced, haemotological aberrations remedied, and specific symptoms (convulsions, delirium, urine retention, gastrointestinal symptoms etc.) must be treated on their merits.

In the experience of both authors, delayed cases are often treated with hyperbaric oxygen, as opposed to the attempts to remove the symptoms by increased pressure. This is especially so in cases of spinal decompression sickness of some days duration. Despite this, an attempt should always be made to remove or reduce symptoms initially by the application of pressure, and to remove bubbles by the inhalation of the highest oxygen mixture possible, with reference to oxygen toxicity. These are more fully described in the conventional medical texts on diving (Bennett and Elliott, 1969; Edmonds *et al*, 1976).

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