be considered decompression-related until proved otherwise in order to prevent the consequences of delay.[16] The main concern is that what is apparent clinically may be the tip of the "neurological iceberg" pathologically. No symptoms should be ignored. Of 470 cases, treated by the North Sea Medical Centre over the last 24 years, 115 patients turned out not to have had dysbaric illness. Decompression sickness can masquerade in many different guises.

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This paper is based on a presentation at a Biomedical Seminars' course on the Management of Compressed Air Decompression Illness held in Amsterdam, August 10th and 11th, 1990.

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DECOMPRESSION ILLNESSES

18 months experience at the Alfred Hospital Hyperbaric Service

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Abstract

One hundred divers presented to the Alfred Hospital Hyperbaric Unit with decompression sickness (DCS, 95 divers) or cerebral arterial gas embolism (CAGE, 5 divers) were reviewed with particular attention to potential predisposing causes, response to treatment and determinants of outcome.

Twenty-six divers presented with DSC following dive profiles outside current table recommendations. The remaining 78 divers developed DCS despite diving within tables. Other commonly identified potential risk factors were multiple dives and/or multiple ascents (55 divers), rapid ascent (17 divers), previous DCS (12 divers), alcohol (6 divers) and altitude (5 divers). No risk factor could be identified in 17 divers. Presenting symptoms were often

mild, however significant neurological deficits were identified in 80 divers. All diver were commenced on 18 metre tables and had an average of 3.5 ± 2.2 treatments. Thirty divers had incomplete clinical resolution despite 3.9 ± 2.5 treatments and one with severe CAGE died. Late presentation (> 120 hours) and past DCS were common in patients with incomplete resolution ($P < 0.05$).

It was concluded that seemingly mild DCS is associated with significant incidence of neurological deficit and incomplete resolution. More conservative dive times, avoidance of the identified risk factors and early recourse to treatment are recommended.

Introduction

Since the establishment of the Hyperbaric facility at the Alfred Hospital, Melbourne, the service has witnessed a steadily increasing number of divers presenting with DCS: 1987-88 45 divers, 1988-89 79 divers, 1989-90 80 divers. The number of divers presenting with DCS in Victoria is at least twice that of any other state.[1] This may partly be attributed to the growing number of participants in the sport (approximately 4,000-10,000 per year), growing diver awareness of this condition, as a result of the educational efforts of sport diving organizations, and publicity about the development of the new hyperbaric facility. This paper reviews in detail the clinical patterns of, and possible predisposing factors to DCS, its response to hyperbaric oxygen (HBO) treatment and clinical outcomes in the first 100 divers treated.

Methods

The first 100 divers presenting with DCS or CAGE were studied with particular attention to possible predisposing factors, clinical symptoms, findings on examination and duration of treatment.

On presentation a detailed history of symptoms, dive profiles and predetermined risk factors was taken. The risk factors considered were multiple ascent/multiple dives, rapid ascent, diving outside current sport dive table recommendations, alcohol, altitude exposure following diving, and a history of DCS.

Clinical examination included general examination for potential aetiological factors. Careful neurological examination and simple tests of neurocognitive function were also done. Neurological examination included detailed assessment of sensation, power, reflexes, and balance using a sharpened Romberg test (the diver was asked to stand in a heel toe position with arms crossed and eyes closed; the number of falls in one minute were recorded). Neurocognitive function was assessed by a careful history of memory, concentration and task performance. Serial seven's (repeated subtraction of 7 from 100 until the lowest positive number is reached; the number and nature of errors and the speed of performance was noted) and short term memory of 5 subjects (number remembered correctly was noted) were performed routinely. Chest X-ray, lung function tests, full blood examination and blood biochemistry were performed before all first treatments.

All patients commenced treatment with a Royal Navy Recompression Table 62 (RN Table 62). If DCS or CAGE was severe and/or unresponsive after 40 minutes at 18 m then Table 62 was extended at 18 m or 9 m or both. Ensuing treatment profiles were based on 18 m Royal Navy Table 61 (RN Table 61) or a modified Table 61 (termed Table 60.5A). Table 60.5A consisted of descent to 18 m on air over 5 minutes, then two 25 minute periods on 100% O_2 at 18 m were each followed by a 5 minute air break. Ascent to 9 m was on 100% O_2 and took 15 minutes, and was followed by a 5 minute air break at 9 m. Finally the ascent to surface was also on 100% O_2 and lasted 15 minutes (total time 1 hour 40 minutes, attendant on air for the first 1 hour 20 minutes then on 100% O_2 during 5 minutes air break at 9 m and during the final ascent).

Chi-squared analysis or Fisher's exact test was performed on all data; p values of < 0.05 were considered statistically significant.

Patient data

TABLE 1

DEMOGRAPHIC DATA
Mean Values Shown + SD

	DCS	CAGE
Number of patients	95	5
Age (years)	30.9	25.2
SD	± 7.3	±7.8
Male : Female	73 : 22	3 : 2
Recreational divers	81	4
Professional divers	14	1
dive instructors	4	1
oil rig divers	0	1
other	10	0
Maximum depth (metres)	21.5	24.6
SD	±8.3	±6.2

There were no statisticially significant differences between those with DCS and CAGE

Demographic data is outlined in Table 1. Five divers were diagnosed as suffering from CAGE. With the exception of one diver, who sustained a massive fatal CAGE, the divers with CAGE, did not differ significantly from divers with DCS with respect to aetiology, clinical manifestations or outcome. These four divers with mild CAGE have therefore been grouped with divers suffering from DCS for subsequent analysis. The most common presenting complaint in divers with DCS was peri-articular pain which persisted despite the administration of analgesics in some divers (Table 2). It was often this lack of response to analgesics or the passage of time which prompted presentation rather than the onset of pain. Other common complaints were of profound lethargy (68 divers) which was accompanied by disturbed cognition in 34 divers and was manifest as poor concentration and difficulty with performing relatively simple mental tasks (such as comprehension of written material). Mild headache was also common (60 divers), whereas paraesthesiae or muscle weakness were less common (42 and 20 divers respectively).

Clinical findings

A wide range of neurological deficits was found (Table 2). While 28 divers presented with neurological disturbances as their sole symptom, 80 divers had clinical evidence of nervous system injury, making this more common than the most common presenting symptom. Musculoskeletal involvement was less common, with persistent joint tenderness evident in 7 of the 72 divers in the absence of effusion or obvious joint deformity. In spite of symptoms consistent with DCS, 15 divers had no detectable abnormality on examination.

Table 3 lists the potential predisposing factors which were considered. The most frequent factors identified in the development of DCS in this group were multiple ascents (arbitrarily defined in this review as 2 or more dives per day or 2 or more ascents in a single dive), and diving outside current sport dive table recommendations (PADI Tables).[2] Rapid ascent (17% of divers) were attributed to lack of diver experience, out of air emergency ascents or equipment malfunction. Alcohol was drunk between 6 to 12 hours prior to diving in 6 divers.

Five divers developed symptoms of DCS at altitude following diving. Interestingly, only one of these involved hypobaric conditions of flight 24 hours after diving. One diver developed recurring symptoms upon returning home to the foot-hills around Melbourne (400-600 m) each night after treatment, 2 divers developed recurrence of DCS while crossing the mountain ranges returning to Melbourne by train after primary treatment in Adelaide (630 m) and 1 diver developed joint pain during ascent in an elevator to the 30th floor of a building. Twelve divers had a previous history of DCS.

TABLE 2

SYMPTOMS AND CLINICAL FINDINGS ON PRESENTATION

Symptoms	
Pain	72
Lethargy	68
Headache	60
Altered sensation	42
Impaired mentation	34
Weakness	20
Clinical findings	
Neurocognitive	80
sensory deficit	36
disturbed sensorium	27
weakness	21
reflex abnormality	18
impaired co-ordination	11
Periarticular tenderness	7
Other	
skin rash	1
None	15

Following diagnosis patients received an average of 3.5 ± 2.2 treatments. Sixty-three divers experienced complete symptomatic and clinical resolution (following 3.2 ± 1.7 treatments) while 37 divers had residual clinical or

TABLE 3

POSSIBLE RISK FACTORS FOR DCS IN 100 DIVERS

Outside dive tables		26
Other indentifiable risk factors		93
multiple ascents/dives	55	
rapid ascent	17	
previous DCS	12	
alcohol	6	
to altitude after diving	5	
No obvious cause		17

The figures are equivalent to percentages

symptomatic abnormalities of these 3 did not respond to treatment (3.9 ± 2.5 treatments). Incomplete response was characterized by residual neurological symptoms ranging from minimal paraesthesiae (14/37, 38%) through to minor cognitive impairment (17/37, 46%).

Of the 3 non-responders, one diver presented with massive CAGE, unconscious and with generalized seizures. Despite a 3 day saturation dive at 18 m his neurological and overall state continued to deteriorate. CT brain scan demonstrated widespread cerebral infarction. The diver died within 48 hours of completion of treatment. The remaining two divers had a previous history of DCS and one presented very late (38 days after the causative dive).

Time to presentation was considered as a possible marker of response to treatment (Table 4). Divers who presented more than 5 days after the responsible dive, were arbitrarily labelled as late presenters. This group of 21 divers had a slightly higher incidence of incomplete resolution but this was not statistically significant.

The incidence of most risk factors for the development of DCS were not significantly different in divers with and without residual problems (Table 5). There was a trend for diving outside the tables to be more common in the incomplete responder group ($p = 0.08$) but the only risk factor which significantly predisposed to incomplete clinical resolution was previous DCS (22% vs 6%, $p < 0.03$).

The majority of divers presenting with DCS were recreational (Table 6). Professional divers exceeded table recommendations more frequently than sports divers (33% vs 22%, not statistically significant). The former group demonstrated a greater incidence of previous DCS.

Neurological involvement, which was present in most patients, was not a predictor of response to treatment; 80% of responders presented with clinical evidence of neurological injury (50 divers), compared with 81% (30 divers) in the residual clinical deficit group.

Discussion

This report has shown that a large and increasing number of divers presented with DCS and CAGE in the state of Victoria, that DCS arose in 74% of divers despite diving within diving table recommendations, that symptomatically mild DCS was associated with an 80% incidence of neurocognitive abnormalities and that incomplete resolution occurred despite prolonged treatment in 37%.

Although the rapid escalation in the popularity of diving has produced a much large population at risk of DCS, most divers are presenting with symptomatically milder forms of the illness when compared with traditional descriptions. This undoubtedly reflects increasing diver education and more conservative diving practices, especially amongst recreational divers. These factors have resulted in less severe forms of the illness and an increased understanding of neurological deficit and incomplete resolution remains concerning. This highlights the importance of identifying and avoiding risk factors both for the development of DCS and for its incomplete resolution.

The lack of a single indentifiable factor responsible for the development of DCS is compounded by variability between divers. It is now recognised that repetition of "safe diver profiles" may still produce DCS.[3,4] In light of such variability, some authors question the reliability of diving tables where strict adherence to protocol cannot be considered to provide complete immunity against DCS.[4,5] There was no obvious predisposition to the development of DCS in 17% of divers, while 74% of dive profiles were within current sport dive table recommendations.[5] This is of particular relevance with the proliferation of dive computers and the practice of multi-level diving which is providing the sports diver with a new flexibility in "safe dive planning". However, the infallibility of dive computers remains a commercial goal rather than a scientific reality as attested to by reports of malfunction and cases of DCS.[6-9] Similarly the application of unproven physiological models and the inability to assess diver tissue nitrogen leads some authors to challenge the application of such instruments in sport diving.[10-12]

Dive profiles, while important, are not the only determinants of nitrogen kinetics during hyperbaric exposure which remains a complex interaction between diver and environmental related factors. It is the nature of this interaction and the identification of DCS determinants that remains controversial.[13,14]

That 55% of our patients reported performing multiple dives and ascents demonstrated the importance of the increased tissue saturation after multiple hyperbaric exposures. Decompression requirements are altered in the presence of residual tissue nitrogen saturation from previous descents.[15]

The use of alcohol prior to diving was recorded in 6% of representing divers. While its potential to impair diver judgment is well known, its effects at a cellular level and influence upon biochemical events at the gas/tissue interface are yet to be determined. Alcohol may impact on nitrogen kinetics via vasodilatation and potential stabilization of venous gas emboli which may alter the excretion of the gas load and predispose to the development of DCS.[14,16,17]

Upon completion of the dive, the stored tissue nitrogen is gradually eliminated, however early exposure to altitude creates a pressure gradient for the rapid dissolution of tissue nitrogen and the evolution of gas bubbles from nitrogen nuclei[18] as attested to by 5 divers in this series. Once a critical volume of gas is exceeded symptoms occur in

TABLE 4

OUTCOME OF DIVERS WITH EARLY AND LATE PRESENTATION

	All Divers	Early Presenters (< 120 hours)	Late Presenters (> 120 hours)
Number of divers	100	79	21
Presentation interval in hours and SD	100 ± 195	37 ± 29.5	340 ± 329
Resolution	63	52 (66%)	11 (52%)
Incomplete resolution	34	25 (32%)	9 (43%)
No response	3	2 (3%)	1 (5%)

There were no statistically significant differences between the early and late presenting groups

TABLE 5

RESPONSE TO TREATMENT IN RELATION TO POSSIBLE RISK FACTORS

Aetiology	Incomplete or no response n (%)	Resolution n (%)	P*
Total number of divers	37	63	
Outside dive tables	13 (35)	13 (21)	0.08
Other identifiable risk factors			
multiple ascent/dives	22 (59)	33 (52)	
rapid ascent	5 (14)	12 (19)	
previous DCS	8 (22)	4 (6)	0.03
alcohol	2 (5)	4 (6)	
altitude	0 (0)	5 (8)	
No obvious cause	5 (14)	12 (19)	

* Only $p < 0.1$ shown

TABLE 6

COMPARISON OF DCS RISK FACTORS PROFESSIONAL VS RECREATIONAL DIVERS

	Professional n (%)	Recreational n (%)	P*
Total	15	85	
Outside tables	5 (33)	19 (22)	
Outside identifiable risk factors			
multiple ascents or dives	8 8 (53)	47 (55)	
rapid ascents	2 (13)	15 (18)	
previous DCS	6 (40)	6 (7)	0.002
alcohol	0 (0)	6 (7)	
altitude	1 (7)	4 (5)	
No obvious cause	3 (20)	14 (16)	

* Only $p < 0.1$ shown

relation to the tissue involved.[15]

A complicating factor, which may contribute to the increased number of divers presenting in Victoria, is the potential role of hypothermia. Although hypothermia was not originally addressed, Victorian waters are colder than the northern states and therefore promote a different nitrogen elimination profile. Early experiments conducted on temperature acclimatized eels (at 11°C) led Belaud and Barthelemy [16] to conclude that the DCS threshold was actually raised by hypothermia. This was in part supported by Dunford and Hayward[19] where divers who commenced the dive cold, demonstrated fewer venous gas emboli than those who remained warm throughout the dive, or who had later became hypothermic. These findings allude to the temperature dependence of nitrogen uptake and elimination which is modified by the circulatory responses of hypo- and hyperthermia. During the dive, at normothermia, the tissues gradually become saturated with nitrogen. This process may be accelerated by exercise which increases muscle and subcutaneous blood flow and the rate of tissue saturation.[15] As the dive progresses peripheral cooling and vasoconstriction occur, the reduced circulation leads to reduced gas flux and "trapping" of nitrogen which potentiate the risk of DCS.[15,16] Although worse with hypothermia, this process occurs with subcutaneous cooling in the absence of central hypothermia. Further, should the diver engage in vigorous exercise at this point (such as occurs on dive courses, salvage and recovery, and rescue) the alteration in physical forces upon intra and extra cellular fluid tension with movement, generates negative hydrostatic pressures sufficient to produce cavitation and bubbles.[20,21]

Nitrogen kinetics may similarly be affected by obesity, in view of the high lipid solubility of the gas, and compounded by the frequent association with lack of cardiorespiratory fitness.[16,22] This is supported by Dembert et al.[14] who demonstrated a 5 to 6 times greater risk for the development of DCS in Naval divers in the highest quartile of full body weight. The gas forming capacity of lipid is due both to tissue mass and nitrogen solubility[20], and bubble formation is a function of tissue saturation and cavitation tendency.[21]

There was a very high incidence of neuropathology (80%) in our series of divers. The reported incidence of clinically identifiable neurological involvement with DCS has ranged from 24-89% even in the absence of symptoms.[23-27] The lack of symptoms in the presence of neurological involvement was probably due to the accompanying neuropsychological impairment due to DCS[25, 27] which impairs insight. This insight often returned during treatment.

While the mechanism of neurological DCS remains unproven, one of the many controversial theories has been provided by Hallenbeck et al.[28] who performed venographic studies of the epidural venous system following hyperbaric exposure in dogs. Doppler studies showed venous bubbles in the lungs which were accompanied by a rise in central venous pressure which was transmitted to the epidural veins producing stasis and ultimately obstruction. Neuronal damage ensued as venous sludging lead to hypoxia and infarction and was compounded by the activation of inflammatory chemical mediators at the blood/bubble interface.

In later study,[29] extravascular interruption of spinal perfusion was demonstrated as a potentially significant contributor to hypoxic neuronal injury. Decompression allows gas expansion so that tissue pressure can exceed perfusion pressure. This "mass effect" is of particular relevance to the spinal and cortical vascular watershed areas.[30,31]

Significant neurological injury is consistent with a large nitrogen load as suggested by Melamed and Ohry[23] where 75% of DCS with neurological involvement followed diving to depths greater than 30 m. The mean depth in our population was 22 m and was associated with clinical neurological involvement in 80%. Kunkle and Beckman,[31] suggest that bubbles, with their associated potential neurological sequelae, may develop de novo in the arterial circulation, following supersaturation of blood during rapid ascent. Such "atraumatic emboli" may develop at an ascent rate as low as one foot/second (60 feet a minute is the recommended rate of ascent for the USN decompression tables). The lower temperatures of Victorian water, with a reduced body temperature, may reduce the threshold for bubble formation.

The neurological injury of DCS may also be contributed to be subclinical pulmonary barotrauma where the bubble load may be increased by the passage of air emboli from broncho-alveolar venous fistulae to the central nervous system.[23,24]

Previous neurological assessment of patients with DCS has demonstrated a high incidence of cognitive dysfunction, and that isolated spinal involvement is in fact uncommon.[23,25,26,32,33] This is consistent with Rozsahegyis' earlier contention that DCS is a diffuse neuronal insult secondary to unique neuronal vulnerability of the nervous system related to:

1 Spinal cord movement which potentially leads to inter-and intra-cellular cavitation with resultant bubble formation.

2 The high metabolic rate of neurones which are therefore sensitive to the metabolic disruption and inflammatory responses initiated by bubble formation.

3 The lipid rich myelin sheath surrounding neurones which provides a potential nitrogen reservoir allowing diffusion of gas into the axon.[20]

The subsequent neuronal damage may therefore culminate in a multifocal encephalomyelopathy.[34]

Once symptoms evolve, the interval between their development and the institution of recompression, has been identified by some investigators as the major factor in determining clinical outcome.[23,25] This study showed a trend toward less favourable outcome in those divers who presented late but this did not reach statistical significance. 75% of divers with a previous history of DCS demonstrated clinical residuals despite treatment, presumably due to pre-existing residual tissue injury.[22]

The diverse, non-specific, and often mild nature of the symptoms of DCS,[13,36] were often underestimated and attributed to other causes. For example, it was not infrequent for divers to attribute fleeting myalgias to lack of fitness, or headache to minor sinus barotrauma. This was often compounded by the unfortunate myth that only "incompetent divers" get bent and the possibility of DCS was then dismissed. This culminated in late presentation following failure of the symptoms to remit or to respond to analgesia. We believe that any symptoms following diving must be considered as potential manifestations of DCS and should be reviewed as soon as possible by a doctor experienced in diving and hyperbaric medicine. The neurological cost of delay is potentially high, with residual neurocognitive deficits evident in 20-80% of reported series,[24,26,27,32] and in 48% of "late presenters" in this study.

Although the efficacy of delayed hyperbaric treatment has not been well established this should not prevent the initiation of treatment even if patients present more than 10 days beyond the onset of symptoms.[36,37]

Conclusions

The results of a retrospective review of 100 divers with DCS and CAGE have demonstrated that:

1 Diving outside the tables, a well recognized risk factor, was not the most common precipitant. Diving within current sport diving table recommendations was not protective for the development of DCS. Common risk factors identified were multiple dives, multiple ascents, rapid ascent, previous DCS, alcohol and altitude. Cool climate diving with or without clinical hypothermia may also contribute to the high incidence of DCS in Victoria.

2 Although presentation was most commonly a result of muscle and joint pains and lethargy, neurological manifestation the most common manifestations of DCS were, often in the absence of symptoms.

3 Late presentation and past history of DCS was associated with a higher incidence of residual deficit.

Although individual risks for DCS may be low, the large and growing sport diver community is resulting in an increasing number of divers presenting with DCS with risk of permanent deficit and impaired higher mental function and future work capacity.

Recommendations

We believe the following recommendations should be adopted by the recreational diving community we serve.

CONSERVATIVE DIVING PRACTICES

1 Diving well within the dive tables rather than to their limits.

2 A maximum of 2 dives per day with a long surface interval between them.

3 Only one ascent per dive.

4 Shorter dive times in cold water.

5 Meticulous avoidance of other identified potential risk factors, especially hypothermia, early exposure to altitude, rapid ascent and diving following the use of alcohol

EARLY PRESENTATION

For **any** symptoms (even if seemingly mild) following diving,.early presentation to a recompression facility or a hyperbaric physician.

We believe that adopting the above practices could potentially reduce the risk of DCS to on third of its current levels.

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