# **ORIGINAL PAPERS**

## DECOMPRESSION ILLNESS AFTER AIR DIVES TREATED IN SINGAPORE 1991-1998

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#### **Key Words**

Air embolism, decompression illness, hyperbaric oxygen, treatment, treatment sequelae.

### Abstract

The Naval Medicine & Hyperbaric Centre (NMHC) is the only recompression chamber facility in Singapore. We receive all local cases of decompression illness (DCI) as well as a substantial number from the surrounding South-East Asian countries.

From 1991 to 1998, 169 patients were referred to NMHC for suspected DCI, either decompression illness (DCS) or cerebral arterial gas embolism (CAGE). Of these, 108 cases of DCS and 5 cases of CAGE were subsequently included in this study. The patients were treated according to our facility's clinical protocols using recompression schedules based on Royal Navy Treatment Tables. Selected demographic, historical, clinical and prognostic data of the eventual study cohort were captured in a computer database and analysed retrospectively.

The majority comprised male divers (86.1%) and most were recreational divers (75.9%). Almost one-fifth of the patients (18.5%) admitted to a previous history of DCI. Alarmingly, two-thirds received no attempts at standard diving first aid at the dive location following onset of symptoms, and only 44.4% began recompression therapy within 24 hours of their dive injury. One quarter of all patients continued to dive despite the onset of symptoms. 71.3% of all patients presented with neurological complaints, which most commonly involved numbness and/or paraesthesia of the extremities. Joint pain was frequently localised to the shoulders, and the incidence of upper limb arthralgia was more than twice that of lower limb pain in this series. No patient deteriorated or failed to respond to recompression and 81.5% achieved complete symptom resolution following completion of the prescribed treatment sessions. Patients who were classified as Type I DCS tended to receive fewer treatments than patients with Type II DCS, although there was no difference in short-term outcome between the two groups. For the patients with CAGE, treatment outcome was good when recompression was initiated early.

Recompression therapy using short oxygen tables leads to an acceptable outcome in the majority of patients with DCS, even when treatment is delayed. Our data support reports elsewhere that joint pain in DCS associated with bounce diving is more likely to be localised in the upper compared to the lower limbs. In our series, patients with pain-only complaints tended to require fewer treatments than those with Type II DCS, although we found no differences in the short-term outcome between the two groups.

### Introduction

Decompression illness (DCI) is the archetypal diver's disease, encompassing a spectrum of clinical signs and symptoms which arise when changes in the ambient pressure result in the unnatural introduction of gases into body tissues. Estimates of DCI incidence have ranged from as high as 1 per 6,000 dives for the general diving population, to as low as 1 in over 50,000 for "undeserved" cases among divers who have no apparent increased risk for DCI.<sup>1-4</sup> Fatalities are even more uncommon, and it may generally be said that diving is a relatively low-risk activity for the medically fit individual who observes safe diving practices.

Recreational diving has been growing steadily in popularity in South-East Asia in recent years. The rate of growth of the sport diving industry in the region has been estimated at between 17-20% annually over the past 5 years and this trend may well be expected to continue over the next few years. The Naval Medicine and Hyperbaric Centre (NMHC) is the only diving medical and hyperbaric facility in Singapore, and is recognised by the Divers Alert Network as a centre for the treatment of diving emergencies such as DCI. Although its raison d'être is centred around the support of military diving operations, it also manages a growing number of civilian referrals for diving-related injuries, as there are, at present, relatively few accredited recompression facilities in South East Asia.

This brief report summarises the findings of a recent review of 108 cases of decompression sickness (DCS) and 5 cases of cerebral arterial gas embolism (CAGE) that were treated at our facility between 1991 and 1998.

#### Methods

### DATA COLLECTION AND ANALYSIS

Detailed clinical and treatment histories of all cases of decompression illness that are referred to our facility are documented in standardised records. A chart review of 169 patients that had been evaluated by our centre for suspected DCI (either DCS or CAGE) from 1991-1998 was performed by the authors and the relevant information was extracted into a computer database. Only divers who had been using compressed air as the breathing gas were considered. 56 subjects were excluded for one or more of the following reasons: inadequate clinical evidence for diagnosis of DCI or an alternative diagnosis made, refusal or default of recompression therapy, or secondary referral after treatment had been partially completed in another hyperbaric facility. 113 patients were eventually included in the final analysis. Cases of DCS have been analysed separately from the patients with CAGE.

The grading of treatment outcome was based on both objective and subjective parameters, and classified into the following categories.

### Complete recovery.

Total resolution of symptoms and signs Partial recovery with minor residual symptoms

Incomplete recovery with the persistence of symptoms and/ or signs that were not distressing nor incapacitating. Patients in this category typically had vague and intermittent niggling complaints which did not affect their activities nor cause them significant discomfort.

Partial recovery with major residual symptoms.

Incomplete recovery with the persistence of deficits that were significantly distressing or incapacitating to the patient.

No recovery.

Initial outcome was defined as the patient's clinical condition as assessed within 24 hours after the first recompression session, whereas short-term final outcome refers to the patient's clinical condition as assessed 24-48 hours after completion of all prescribed treatments. In this review, we have used the traditional Type I and II DCS nomenclature as we have found it to be useful and expedient in our clinical practice, although we recognise its short-comings compared with an evolving classification that is based on descriptive symptomatology.<sup>5,6</sup> The definition of Type I DCS was restricted to musculo-skeletal pain and dermatological complaints only, whereas Type II DCS was a far broader category comprising those patients with neurological and cardio-respiratory symptoms and signs.

Statistical analysis was performed, using the SPSS computer package for the Windows environment. The main instruments used were the Pearson Chi-square test (2 tailed) for comparing proportions and the Student's T-test (2 tailed) for means. Comparisons were considered to be statistically significant for p < 0.05.

#### CLINICAL MANAGEMENT

All cases were treated in one of two multiplace chambers that are equipped with built-in breathing systems (BIBS) for oxygen delivery. Standard Royal Navy (RN) oxygen tables were used, mainly Tables 61 and 62. Patients were typically started on Table 62 and oxygen extensions added according to the observed clinical response after the first oxygen period. For very mild cases of musculoskeletal or dermatological DCS (Type I DCS), Table 61 was at times used as the initial table, although the treatment would be extended to follow the Table 62 protocol should there be unsatisfactory resolution of symptoms at the initial treatment depth. No ancillary or adjuvant therapy specific to DCI was used, other than intravenous hydration in those patients who were clinically dehydrated. All CAGE patients were on intravenous fluids during recompression.

All patients were reviewed daily, and subsequent management was guided by the patient's condition. Patients who continued to complain of significant symptoms following the initial recompression treatment (major residual symptoms/signs) usually underwent a repeat session of the first table, whereas those who demonstrated marked improvement were retreated on RN Table 61. These treatment sessions were repeated daily until no further improvement was observed on 2 consecutive treatments, or until complete resolution of the presenting complaints was achieved.

#### **Results DCS patients**

### DEMOGRAPHICS

The number of patients that were treated annually by our facility for DCS was fairly constant between 1991-1995 at about 8 a year. That number has increased steadily over the past 3 years and is now about 26 cases a year (Table 1). Eighty two (75.9%) were recreational divers. There were 14 (13.0%) commercial and 12 (11.1%) military divers. There were 93 males (86.1%) whose ages ranged from 20-58 years (mean 31.2 years). The 15 females (13.9%) had a mean age of 30.2 years with a range of 21-48 years (Table 2).

### TABLE 1

### DCS PATIENTS TREATED

Year	<b>DCS Patients treated</b>
1991	9
1992	6
1993	8
1994	7
1995	10
1996	15
1997	26
1998	27
Total	108

#### TABLE 2

## **AGE-GENDER DISTRIBUTION**

Age	Male	Female	Total
20-24	23	1	24
25-29	18	7	25
30-34	21	4	25
35-39	19	1	20
40-44	7	2	9
45-49	3	0	3
>49	2	0	2
Total	93	15	108

### PREVIOUS HISTORY OF DCS

Twenty of our patients, almost a fifth (18.5%), had previously suffered at least one episode of DCS for which they had sought medical attention, although none admitted to any residual symptoms from this past encounter. Five (4.6%) divers had a history of bronchial asthma, and one (0.9%) with chronic hypertension was on long-term medication. None of our patients volunteered a history of cardiac valvular or septal defects, and physical examination did not reveal any cardiac abnormality in any of the divers.

### DIVE PROFILE

The average depth of the dive immediately preceding the onset of symptoms was 27.2 m, while the mean maximum depth reached for all patients was 31.2 m. Other researchers have noted that a sizeable proportion of recreational divers develop DCI after just one day of diving<sup>3,4</sup> and we found that 22.2% of our patients were afflicted following just one dive. The information provided by many patients regarding their dive profiles was often incomplete or imprecise, but it appeared that many, if not most, recreational divers were performing repetitive and/or multilevel diving.

Among our patients, only five (4.6%) divers claimed to have descended no deeper than 10 m on all dives, although about 1 in 10 (12/108) divers reported sustaining their "hit" immediately after a dive of 10 m or less. However, of the latter group, most had completed at least one other dive on the same day. Unfortunately incomplete data concerning the other dive profiles and surface intervals often prevented us from making meaningful comments on whether repetitive limits had been exceeded.

#### ON-SITE MANAGEMENT AND EVACUATION

Alarmingly, 27 (25.0%) of our patients continued to dive following the onset of symptoms. The mean number

of additional dives undertaken by these patients was 5. When recreational divers alone were considered, 21 (25.6%) persisted in diving despite their symptoms.

Barely a third, 36 divers (33.3%), reported receiving any diving first aid (100% oxygen or rehydration) at the dive site. Only 48 (44.4%) of the afflicted divers began recompression therapy within 24 hours of symptom onset, but this may be due to the fact that our facility is far from many of the popular dive sites in the region.

### SYMPTOMS

Reliable information regarding time from surfacing to onset of symptoms was obtained from only 48 patients (44.4%). In this group the mean time was 3 hours and 39 minutes. Thirty nine divers (81.3%) had symptoms presenting within 3 hours, 42 (87.5%) within 6 hours and 44 (91.7%) within 12 hours of surfacing.

The presenting symptomology is shown in Table 3. Neurological symptoms and/or signs (Type II DCS) were the most frequent complaint with 77 divers (71.3%) reporting them. The majority (72 patients) presented with numbness and/or paraesthesia. 10 patients had upper limb weakness, while lower limb weakness was also present in 10 divers. Visual disturbances (3 patients) and bowel and bladder dysfunction (7 patients) were relatively uncommon.

Musculoskeletal pain and aches were also prevalent (64.8 %), although only 31 divers (28.7%) complained of pain or aches as the only symptom (Type I

#### **TABLE 3**

### SYMPTOMS OF DCI IN 108 PATIENTS

Symptom	Number	(% of total)
Joint Pain/Ache	70	(64.8)
Shoulder	33	(30.6)
Elbow/arm	39	(36.1)
Hip	10	(9.3)
Knee/leg	21	(19.4)
Back	8	(7.4)
Neurological	77	(71.3)
Numbness/paraesthesia	72	(66.7)
Upper limb weakness	10	(9.3)
Lower limb weakness	10	(9.3)
Bowel/bladder difficulties	7	(6.5)
Visual complaints	3	(2.8)
Fatigue/lethargy	36	(33.3)
Headache	10	(9.3)
"Chokes" (Respiratory)	5	(4.6)

DCS). Pain was most commonly localised to the upper limbs and particularly the shoulders (30.6%), a pattern that has been reported by other investigators. The frequency of upper limb joint pain was approximately twice that of joint pain in the lower limbs (55 vs 27).

### OUTCOME

Following the initial recompression, only 36 patients achieved complete symptom resolution and 15 responded poorly with either no relief or minimal relief (Table 4). However, after completion of all prescribed sessions, almost all patients demonstrated substantial recovery, with 88 patients (81.5%) achieving complete recovery and only one patient having major residual deficits (Table 5). No patient deteriorated during or following treatment. There was no major complication suffered by any patient that directly resulted from recompression therapy for DCS. There was no recorded oxygen-induced convulsions or pulmonary barotrauma.

We found no statistical relationship between outcomes and time from injury to treatment, or between Type I and II DCS. Patients with neurological complaints did not appear to fare any worse than patients with musculoskeletal or constitutional symptoms only. We found, however, that patients who had been classified as Type I DCS at presentation tended to require fewer treatments that those who were diagnosed as Type II (1.90 vs 2.68, p<0.05).

### **CAGE** Patients

Five cases of CAGE were treated at our facility during this period. All were male, ages ranging from 23 to 40. Three were recreational divers. All presented with a history of rapid, uncontrolled ascent accompanied by an acute onset of significant neurological deficit, such as loss of consciousness or hemiplegia, during ascent or upon surfacing. No patient had any clinical or X-ray indication of pulmonary barotrauma such as pneumothorax, pneumomediastinum or subcutaneous emphysema, nor did any have clinical evidence of a cardiac septal defect.

Two patients were comatose upon arrival and mechanically ventilated. One of these was transferred to us, after a delay of about 24 hours, from a foreign hospital. His condition continued to deteriorate following

### TABLE 4

#### **RESPONSE TO INITIAL RECOMPRESSION TREATMENT**

Recovery	n		Treatm	ent L	Delay	DCS Type		Previous DCS History					
		<	24 hrs	>	24 hrs		Ι		II	Po	sitive	Neg	gative
Complete	36	16	(33.3)	20	(33.3)	9	(29.0)	27	(35.1)	6	(30.0)	30	(34.1)
Partial/ Minor	57	25	(52.1)	32	(53.3)	20	(64.5)	37	(48.1)	13	(65.0)	44	(50.0)
Partial/ Major	10	4	(8.3)	6	(10.0)	1	(3.2)	9	(11.7)	0		10	(11.4)
None	5	3	(6.3)	2	(3.3)	1	(3.2)	4	(5.2)	1	(5.0)	4	(4.5)
Totals	108	48		60		31		77		20		88	
		n/s				n/s				n/s			

Note. Figures in parentheses refer to percentages within each sub-category; n/s = not significant)

### **TABLE 5**

### SHORT-TERM FINAL RECOVERY FOLLOWING COMPLETION OF ALL PRESCRIBED TREATMENTS

Recovery	n	Treatn	nent Delay	DCS Type	P	Previous DCS History		
		< 24 hrs	> 24 hrs	Ι	II P	ositive	Negative	
Complete	88	41 (85.4)	47 (78.3)	26 (83.9) 62	(80.5) 17	7 (85.0)	71 (80.7)	
Partial/ Minor	19	6 (12.5)	13 (21.7)	5 (16.1) 14	()	2 (10.0)	17 (19.3)	
Partial/ Major	1	1 (2.1)	0	0 1	(1.3)	1 (5.0)	0	
Totals	108	37	54	31 77	20	)	88	
		n/s		n/s	n/s	s		

Note. Figures in parentheses refer to percentages within each sub-category; n/s= not significant

the first treatment (RN Table 63) and he eventually died. Fortunately for the second patient, we were able to commence recompression on Table 62 within 8 hours. He regained consciousness and a measure of lucidity midway through the second treatment. A total of four sessions of Table 62 was eventually administered, and he responded remarkably well with virtually no residual functional deficits after the final treatment. He has since returned to work and has had no further complaints over almost a year of follow-up.

Two other patients presented initially with transient loss of consciousness subsequent to a rapid ascent, which was followed by neurological symptoms. Both were treated within 6 hours, and each achieved complete recovery following two recompression sessions.

Our last patient complained of transient loss of consciousness accompanied by numbness and weakness of his lower limbs following a precipitate ascent. He was initially evaluated at another non-hyperbaric medical facility and was only referred to us after almost 48 hours. By this time his complaints had mainly resolved except for the numbness. Two treatments were administered, but only marginal improvement was noted.

No specific pharmacological adjunct was used for any of these patients.

### Discussion

The rising number of patients with decompression illness that have been referred to our facility over the past few years is most easily, and also most likely, explained by the surging popularity of recreational diving both in the region and globally. No doubt, the increasing popularity of diving destinations in South East Asia has also added to these numbers.

In so far as the epidemiology of DCI and other diving injuries are concerned, recreational divers represent the population which is most at risk. It is not difficult to see why. The general level of training is uneven, regulation of dive operators is problematic, and frequently recreational divers themselves seem willing to "take the odd chance" to maximise personal enjoyment rather than individual safety.

The proportion of patients in our study who admitted to a previous history of DCS is rather high. DAN noted a figure of only 6.6 % (confirmed cases) in a recent report.<sup>4</sup> It has been suggested by some that divers who have had a past history of DCS are at an increased risk of future DCS, but it is unclear whether this is due to an intrinsic genetic or physiological factor, or whether it is the unsafe diving technique practised by the diver in question that places him at increased risk. The high figure reported in this study may perhaps be explained by a process of self-selection and an element of recall bias. It may reasonably be expected that divers who have previously suffered from DCS would be more familiar with the signs and symptoms of the disease and more aware of its consequences. Hopefully they would be more likely to seek treatment. We were unable to discern any relationship between a history of previous DCS and treatment outcome, following basic stratification for other parameters.

As alluded to above, some of our patients had been performing fairly shallow and "safe" dives but had nonetheless been afflicted with DCS. Closer questioning and clinical evaluation of these patients often revealed no other definite risk factors. There has been some interest in the phenomenon of "shallow water bends", particularly among the lay diving community. This refers to the onset of DCI following apparently innocuous dive profiles at shallow depths and of short duration. It is unclear if this phenomenon actually exists, although it has been proposed that some reported cases may have been due to arterial gas embolism (AGE), e.g. in the presence of a previously unsuspected congenital cardiac septal defect.<sup>7</sup> Other reports may have omitted information about preceding dives that would have contributed significantly to the inert gas load.

Our finding that a quarter of the patients persisted in diving despite their symptoms is a rather disturbing one. It is uncertain whether these patients did so because they were unable to appreciate that they could have developed DCS, or whether they simply chose to ignore their symptoms. Nevertheless, it is worrying that the dive supervisors and operators were not more vigilant to the possibility of DCS and failed to advise their charges accordingly.

The presenting symptomology is consistent with reports published elsewhere.<sup>3,4,8,9</sup> It has been suggested that DCS resulting from bounce diving is more commonly associated with upper limb pain, in contrast to the greater proportion of lower limb complaints that are encountered in compressed air workers and saturation divers. This claim is compatible with our results. A recent retrospective study has also supported this observation, and concluded that counter-current exchange of inert gas may be implicated in the distribution of limb pain in DCS.<sup>10</sup>

The treatment results in our series of 108 patients with DCS compare favourably with those reported elsewhere (Table 6), although studies in which the majority of patients received early recompression (12 hours or less following symptom onset) tend to report better outcomes. Recent data from DAN's diving accident database have strongly suggested that for up to 12 hours following onset of DCI, earlier times to treatment correlate with improved prognosis.<sup>4</sup> However, we found no statistically significant impact that delay to treatment (within 24 hours or more than 24 hours) makes on either the initial or final response to recompression. Most of our patients typically require more than 12 hours to arrive at our facility, and it is likely that the critical threshold or "golden hour" for optimal results with recompression is within 12 hours of the injury.

Nevertheless, the generally satisfactory outcomes support the argument that recompression should be attempted even when it is delayed and there are numerous reports in the literature documenting favourable outcomes in such situations.<sup>11-13</sup> We recently managed a young woman with neurological DCI who only sought treatment at our facility almost 5 days following the onset of her symptoms. Her complaints, which included patchy numbness and paraesthesia over her arms and feet, as well as weakness of hand grip, were completely resolved following a single recompression session using RN Table 62. A case-control or even a controlled trial would be useful in shedding further light on the optimal temporal envelope for treatment.

The number of CAGE patients treated is too small to be subjected to any meaningful statistical analysis, but our experience seems to suggest that the outcome is generally good to excellent provided that treatment is initiated early. This small series also supports previous observations that the association of CAGE with pneumothorax and significant pulmonary barotrauma is uncommon.<sup>14,15</sup>

### TABLE

## SELECTED REPORTS OF TREATMENT OUTCOMES FOR DECOMPRESSION SICKNESS

Author	Year	Cases	Results	Remarks		
Erde and Edmonds <sup>11</sup>	1975	100	5 patients treated with air tables. 20/95 treated with oxygen tables left with incomplete recovery.	Recreational divers		
How et al. <sup>8</sup>	1976	115	<ul><li>63% complete recovery.</li><li>6% no significant clinical improvement.</li></ul>	Both air and oxygen tables used. Mean delay to treatment 50.9 hours		
Bayne <sup>17</sup>	1978	50	Complete recovery in all cases. 49 with full recovery after a single treatment.	Equal numbers of Type I and II DCS		
Kizer <sup>18</sup>	1980	157	17% with significant residual symptoms	10% were AGE cases. Average delay to treatment > 7 hr.		
Gray <sup>19</sup>	1984	812	<ul><li>751 cases treated with oxygen tables.</li><li>83 % full recovery after 1 treatment.</li><li>7 deaths.</li></ul>	244/248 Type I DCS and 54/57 Type II DCS full recovery after 1 treatment.		
Gorman et al. <sup>20</sup>	1987	88	15 cases with residual symptoms/ signs detected on follow-up	USN oxygen tables. Follow up with neurological clinical evaluation, EEG and CT scan.		
Brew et al. <sup>21</sup>	1990	125	68 patients with residual symptoms/ signs following completion of prescribed treatment.	AGE cases included. Mean delay to treatment was 57 hours for DCS and 12.7 hours for AGE.		
Gardner et al. <sup>9</sup>	1996	100	30 patients with partial recovery.	USN and RNZN (oxygen-helium) tables. Mean delay to treatment 8 hours.		
Arness MK <sup>3</sup>	1997	94	Complete recovery in 91% of cases.	USAF-modified USN oxygen tables. 82 % of cases treated within 24 hours of onset of symptoms		

### Conclusion

Recompression therapy using short oxygen tables leads to an acceptable outcome in the majority of patients with DCI, even when treatment is delayed. However, since improved outcome has been associated with shorter times to recompression (within 12 hours), and this seems particularly true of CAGE, one avenue of enhancing secondary prevention is to focus on properly educating the diving community to better recognise DCI in its myriad presentations and so encourage earlier evacuation. It is also vitally important that dive operators and supervisors be suitably equipped and trained to provide the appropriate first responder care to diving casualties and, in particular, in the administration of 100% oxygen. With recent advances in transportable chamber technology and as experience with them in the field increases, one option would be to explore the feasibility of making such chambers more readily available.16

Despite the wealth of clinical experience with recompression protocols, many unanswered questions remain regarding patient selection and the relative merits of different tables and protocols. Our understanding of prognostic factors and adjuvant pharmacotherapy is also inadequate. These and other issues have to be addressed through a concerted effort by the diving medical community in order to further improve the delivery of care to our patients.

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