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Risk factors and treatment outcome in scuba divers with spinal cord decompression sickness

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Abstract

Purpose: This study was designed to determine the recompression strategy and the potential risk factors associated with the development of severe diving-related spinal cord decompression sickness (DCS).

Material and methods: Sixty-three injured recreational divers (52 men and 11 women; 46 ± 12 years) presenting with symptoms of spinal involvement were retrospectively included. Diving information, symptom latency after dive completion, and time interval between symptom onset and hyperbaric treatment were studied. The severity of spinal cord DCS was rated numerically for both the acute event and 1-month later. Initial recompression treatment at 2.8 atmosphere absolute (ATA) with 100% oxygen breathing or deeper recompression at 4 atmosphere absolute with nitrogen-oxygen or helium-oxygen breathing mixture was also noted.

Results: Twenty-one divers (33%) had incomplete resolution after 1 month. The clinical severity at presentation was the only independent predictor of poor outcome (odd ratio, 2.68; $P < .033$). Time to treatment did not influence the recovery with a similar median delay (3 hours) between the divers with or without long-term sequelae. Choice of recompression procedure was not also a determinant factor for treatment outcome.

Conclusion: The initial clinical course before treatment is a major prognostic factor of spinal cord DCS. Delay to recompression less than 3 hours and use of deep treatment tables did not improve outcome in DCS divers.

1. Introduction

Decompression sickness (DCS) encountered by divers results from the formation of intravascular and extravascular inert gas bubbles previously dissolved within tissues after inadequate decompression from hyperbaric exposure. Neurological injuries are predominant in DCS, and spinal cord is the most commonly affected site. The clinical features of spinal involvement are numerous, and neurological symptoms may vary considerably from minimal subjective sensory abnormalities to complete paraplegia with bladder dysfunction that could result in permanent disability. The pathophysiological mechanisms of spinal cord lesions include several hypotheses, that is, arterial, venous, and autochthonous bubble theories, but extravascular bubble growth in spinal white matter and venous cord infarction resulting from bubble embolization of epidural vertebral venous system causing obstruction of venous drainage and subsequent coagulation activation and platelet aggregation

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are probably the main explanation for the pathogenesis of this myelopathy [1].

By reducing bubble volume and by hastening inert gas elimination, recompression therapy with hyperbaric oxygen (HBO) remains the mainstays of modern treatment of DCS. On the basis of historical experience, recompression to 2.8 atmosphere absolute (ATA) using 100% oxygen (eg, US Navy table 5 or 6) is the most commonly procedure with a high rate of efficiency and a low incidence of side effects [2-4]. However, published human outcome data comparing these so-called short oxygen tables with other treatment tables using deeper recompression to 4 to 6 ATA with nitrogen-oxygen or helium-oxygen mixtures are limited and conflicting [5-7], raising some questions regarding the optimal recompression strategy.

Numerous studies have been conducted in the past with the aim on identifying possible determinants of outcome in neurological DCS [8-11], particularly the clinical presentation before treatment [12,13], the symptom latency after surfacing [14-17], and the delay between the onset of symptoms and recompression treatment [11,16,18-24]. However, identification of preponderant factors that could prevent divers from developing severe neurological DCS is still elusive.

From the previously mentioned considerations, the purpose of this study was to investigate the influence of potential risk factors associated with a poor outcome in spinal cord DCS. We also sought the real prognostic value of a simple clinically based scoring system previously developed by Boussuges and coworkers [25].

2. Methods

2.1. Study design

From June 2002 to October 2007, we retrospectively reviewed the clinical and diving data on 63 consecutive air divers referred to our hyperbaric center with symptoms indicative of spinal cord DCS.

Clinical diagnosis of spinal cord injury was made when the criteria of bilateral sensory or/and motor deficit were recognized after the diver surfaced. If the need arises, other characteristic signs consistent with involvement of spinal cord in DCS such as acute back pain or bladder dysfunction were recorded. In 6 (9%) cases, we also reported a combination of spinal and cerebral or vestibular manifestations.

Initial severity of spinal myelopathy was evaluated according to the gravity score of Boussuges and coworkers [25], a system in which the prognostic value was prospectively validated. The score is calculated from 5 weighted clinical variables as follows: repetitive dive, clinical course before HBO treatment, objective sensory deficit, motor impairment, and urinary disturbance (Table 1). It has been demonstrated that cases with scores greater than 7 predict more severe sequelae than those of cases with scores of 7 or less. On the basis of the last neurological examination after all hyperbaric treatments, clinical outcome was also determined by the recovery status 1 month postinjury, that is, full recovery or presence of residual neurological symptoms defined as persistent objective sensory, motor, or urinary disorders.

Table 1 Parameters used in the Boussuges scoring system and their numerical weightings

Manifestation	0	1	2	3	4	5	6
Repetitive dive							
No	X						
Yes			X				
Clinical course before HBO							
Stability				X			
Deterioration						X	
Objective sensory deficit							
No	X						
Yes					X		
Motor impairment							
None	X						
Monoparesis, paraparesis, or tetraparesis					X		
Hemiplegia				X			
Paraplegia							X
Urinary disturbance							
No	X						
Yes						X	

First-aid normobaric oxygen was systematically administered during transportation. After admission and complete clinical evaluation, all patients underwent a recompression treatment with HBO and standardized intravenous therapy with administration of methylprednisolone (120 mg), pentoxyfilline (200 mg), buflomedil (vasodilator; 400 mg), and aspirin (250-500 mg) according to our treatment procedures. Initial treatment table regimen was 100% oxygen breathing at 2.8 ATA for either 150 minutes (equivalent to US Navy table 5) or 330 minutes (equivalent to US Navy table 6), or a schedule involving breathing 50% oxygen with 50% helium or 50% nitrogen at 4 ATA for the first 150 to 180 minutes followed by 100% oxygen breathing at 2.8 to 2.5 ATA, with different stops until surfacing for 300 to 330 minutes (Comex 30 table or GERS B (Groupe d' Etude et de Recherche Sous-marine) French table, Fig. 1, respectively [26]). US Navy Table 5 was generally used for mildly injured divers with minor neurological symptoms, whereas decision about the prescription of either oxygen table for 330 minutes or 4 ATA table was rather recommended for severely cases according to the practice of the physician on duty. Additional HBO sessions (2.5 ATA for 70 minutes) twice daily were given until the patients fully recovered or until no further improvement could be observed after 3 further sessions. The study design was approved by the local ethical committee.

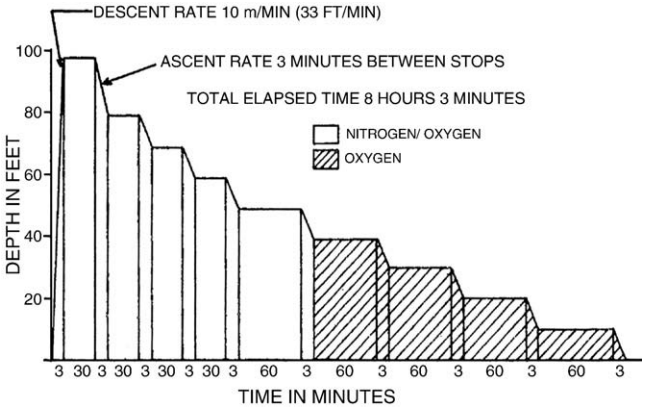

Fig. 1 GERS B treatment table. Reprinted from Berghage et al [26].

2.2. Statistical analysis

Data are expressed as mean ± SD or median with range for nonparametric variables. Clinical outcome was used as the dependent variable, that is, full or incomplete recovery. Diving information, clinical characteristics, and treatment procedures were treated as categorical variables. Univariate analysis was performed with χ^2 test or Fisher exact test to identify significant variables ($P < .05$) predicting incomplete recovery. Variables with a P value less than .20 were used as covariates in multivariate analysis with backward elimination logistic regression to control for potential confounders and to determine independent predictors of severe spinal cord DCS. In this model, highly intercorrelated independent variables ($r > 0.7$) were avoided. Odds ratios (ORs), adjusted ORs, and 95% confidence intervals (95% CIs) were calculated. Comparison for 2 variables between the injured divers with and without sequelae was also performed using the Mann-Whitney U test. Calculations were done using the Sigmastat 3.0 software program (SYSTAT Inc, Richmond, Ca).

3. Results

There were 52 (82%) men and 11 (18%) women with a mean (±SD) age of 46 (±12) years who were retained for analysis. Forty-eight (76%) were experienced amateur or diving instructors. Diving profiles were as follows: mean (±SD) maximum depth of 40 (±13) meters of seawater and mean (±SD) dive time of 30 (±12) minutes. Five patients (8%) performed a provocative decompression profile (ie, fast ascent or omitted decompression stops according to their dive computers), and repetitive dive was only recorded in 11 (17%) cases. The median time from surfacing to onset of initial symptoms was 5 minutes (range, 0-600 minutes), and the median delay to hyperbaric treatment was 3 hours (range, 2-24 hours). Of the 63 injured divers, 21 (33%) had incomplete resolution of neurological symptoms after 1 month, including 8 (12%) patients presenting severe disability (bladder dysfunction, ataxia, or motor impairment; Table 2). The mean (±SD) number of additional HBO was 5.8 (±7.1), with 8 (12%) patients receiving 20 treatments or more.

Distribution of categorical variables and results of statistical analysis are detailed in Table 3.

The univariate analysis revealed that significant variables associated with a poor outcome were age (>45 years; $P = .04$), a short delay of onset of symptoms after the dive ($P = .015$), the presence of acute back pain preceding neurological manifestations upon reaching the surface ($P = .023$), an initial high severity score ($P < .001$), and a number of additional HBO sessions greater than 5 ($P < .001$). Neither time to treatment or choice of treatment table was significantly related with clinical outcome.

Because follow-up HBO therapy and severity score were highly intercorrelated ($r = .78$), additional HBO was not included as a covariate in the second part of statistical analysis. When the multivariate analysis was applied, the only independent predictor of poor outcome was the severity score of Boussuges (OR, 2.68; 95% CI,1.08-6.73; $P = .033$).

Separate analysis showed that divers with a high initial severity score were taken to treatment in the same time than those with a score less than 7, and the delay between onset of symptoms and initial treatment table was not different in divers with full recovery (median, 3 hours) when compared with divers with incomplete recovery (median, 3 hours). Fisher exact test performed between clinical outcome and choice of recompression (ie, 2.8 vs 4 ATA) in each subgroup of patients with a low (≤7) or high (>7) severity score did not

Table 2 Clinical outcome at one month according to the initial presentation

Symptoms at presentation	At 1 mo			
	Full recovery	Minor signs	Moderate symptoms	Severe disability
Subjective sensory loss, n = 28	28 (100)	0	0	0
Objective sensory loss only, n = 11	6 (54.5)	3 (27.2)	2 (18.1)	0
Motor impairment and sensory loss, n = 24	8 (33.3)	2 (8.3)	6 (25)	8 (33.3)
Cerebral involvement, n = 2	2 (100)	0	0	0
Cochleovestibular manifestations, n = 4	4 (100)	0	0	0

Minor signs indicate subjective symptoms with no limitation to activities to daily living. Moderate symptoms indicate objective symptoms with mild impact to activities to daily living, whereas severe disability corresponds to major objective symptoms with substantial impact to activities to daily living. Numbers in parentheses are % (total).

Table 3 Analysis of clinical outcome in divers with spinal cord DCS according to diving information, clinical characteristics, and treatment procedures

Variables	Univariate analysis				Multivariate analysis		
	Total (n = 63)	Full recovery	Sequelae	*P*	OR (95% CI)	*P*	OR_{adj} (95% CI)
Age (y)				.04	3.68 (1.19-11.29)	.10	
≤45	31	25	6				
>45	32	17	15				
Sex				.30	0.39 (0.08- 2)		
Male	52	33	19				
Female	11	9	2				
Diving experience (no. of dives)				.35	2.4 (0.61-9.67)		
≤150	15	12	3				
>150	48	30	18				
Dive time (min)				.40	0.55 (0.18-1.65)		
≤30	36	17	13				
>30	27	19	8				
Depth (meters of seawater)				.30	3.25 (1.09-9.66)		
≤40	36	28	8				
>40	27	14	13				
Control ascent				.29	–		
Yes	58	37	21				
No	5	5	0				
Delay onset of symptom (min)				.015		.10	
≤5	36	19	17		1		
5-60	19	15	4		0.3 (0.08-1.08)		
>60	8	8	0		–		
Delay to treatment (h)				.15		.60	
≤3	35	24	11		1		
3-6	15	12	3		0.55 (0.13-2.35)		
>6	13	6	7		2.55 (0.69-9.39)		
Back pain				.023	4.04 (1.3-12.3)	.40	
Yes	25	12	13				
No	38	30	8				
Severity score of Boussuges				<.001	123.5 (19.01-802.04)	.033	>2.68 (1.08-6.73)
≤7	41	39	2				
>7	22	3	19				
Treatment table regimen (ATA)				.11	2.5 (0.83-7.53)	.50	
2.8	40	30	10				
4	23	12	11				
Additional HBO				<.001	148 (16.16-1365.77)	–	
≤5	38	37	1				
>5	25	5	20				

display also any direct association (P = .43 and P = .9, respectively; Table 4). In addition, among the 19 divers with a score greater than 7 and incomplete resolution of neurological symptoms, we found no significant differences in the median initial severity score between the 2 treatment procedures, excluding a possible selection bias in the choice of initial treatment table (P = .39, data not shown).

4. Discussion

The prevalence of injured divers with incomplete recovery after spinal cord DCS is reported between 22% [17] and 61% [21] in the recent literature, supporting our results despite the differences in diving population, treatment procedures, and delay in time to hyperbaric recompression.

Table 4 Distribution of clinical outcome as a function of treatment table according to the initial severity score of Boussuges

	Treatment table		Full recovery	Sequelae	*P*
High severity	2.8 ATA	USN table 5	0	4	.9
		USN table 6	1	5	
	4 ATA	Comex 30 table	2	6	
		GERS B table	0	4	
Low severity	2.8 ATA	USN table 5	29	1	.43
		USN table 6	1	0	
	4 ATA	Comex 30 table	7	1	
		GERS B table	2	0	

High severity defined as a score greater than 7 and low severity as a score less or equal to 7. Fischer exact test was used to determine the lack of association between outcomes and table regimen (2.8 ATA vs 4 ATA). USN indicates US Navy.

The main finding of this study is that initial clinical presentation depicted by the severity score of Boussuges in neurological DCS provides major prognostic information about the clinical outcome and the severity of this myelopathy, with a positive predictive value of 86% and a negative predictive value of 95%. This result is in good agreement with an earlier series of 217 injured divers treated in the UK, which showed that application of this score had useful predictive qualities, with a very significantly higher proportion of severity score exceeded 7 in cases with severe sequelae ($P < .0001$) [27]. Recently, it has been also demonstrated that spinal lesions detected with magnetic resonance imaging within 10 days of injury were more present in divers with a high severity score and in divers experiencing a poor outcome ($P < .001$) [28]. In general, other scoring systems that relate initial clinical manifestations to long-term outcome associated with analysis of previous study assessing the risk factors of severe DCS also support the impression that the presence of objective disorders and deteriorating condition on admission are important predictors of poor outcome [12,13,19,21].

When univariate analysis was applied to our current data, we found that other variables selected as potential risk factors, that is, age, symptoms latency, and occurrence of acute back pain, were also associated with residual deficit. After adjustment by logistic regression, however, the relationship between these variables and the outcome did not seem to be relevant. Although the role of age as possible determinant for the severity of DCS is uncertain [10,22], a short delay of onset of symptoms after surfacing is presently considered as a risk factor of worse outcome [14-16]. It has been also observed that acute back pain resulting from spinal cord involvement during DCS development was strongly related with magnetic resonance imaging abnormalities and persistence of neurological sequelae [28]. These discrepancies with our results reveal that numerous cofactors are intercorrelated with clinical outcome, suggesting that univariate tests of uncontrolled epidemiological data should be used cautiously.

In this report, neither with a univariate nor with a multivariate analysis time elapsed from onset of symptoms to hyperbaric recompression seemed to influence the final outcome. It is important to note that the comparison of median delay to treatment between the 2 groups of injured divers with or without sequelae did not show any differences (ie, 3 hours) excluding a potential methodological bias. Furthermore, we have shown that application of a short delay less than 3 hours was not significantly associated with a better outcome. At present, medical evidence supporting the relative importance of time to HBO treatment in DCS is controversial, notably if we consider that the usual delay for recompression found in most of clinical studies is quite long, with median time between 6 and 24 hours [10,18,19,21]. Few studies have reported shorter delays for treatment, but the time interval in which a potential benefit from hyperbaric treatment could be obtained is uncertain. Some authors commented that DCS might be more responsive to recompression treatment in the first minutes rather than after hours had elapsed [24,29], but data documenting this assumption are mainly based on theoretical considerations and preliminary results in animal studies [30]. A recent report reveals that the window of opportunity for recompression may be quoted as 6 hours, with less beneficial effect on outcome if applied thereafter [22]. A clinical audit conducted on 390 divers that had HBO in less than 6 hours in Scotland between 1991 and 2003 concluded that the relationship between time to treatment and poor condition on discharge for severely affected cases was weak, with an insignificant increased risk of sequelae [20]. In a series of 96 divers treated for neurological DCS, Boussuges et al [25] also stated that a median delay of 3.5 hours between emerging from the water and treatment did not differ among divers with or without sequelae. Hence, the previously mentioned findings suggest that optimal time interval may be less than 6 hours. However, our current observations, in accordance with Boussuges and coworkers, confirm that shorter delays are not a guarantee of full recovery. Because prompt delay imposed by transportation is not associated with clinically significant benefit, early HBO treatments seem injustified, and the absolute use of helicopter for emergency evacuation should be appropriately discussed before a transfer to a competent hyperbaric facility nearby the diving site.

In this study, as shown in Table 3, there seems to be no significant improvement in treatment outcome for divers with spinal cord DCS treated with enhanced tables at 4 ATA versus tables at 2.8 ATA with 100% oxygen breathing. This lack of association between clinical outcome and hyperbaric treatment procedure points out that the choice of initial treatment table does not seem preponderant whatever the clinical presentation before recompression, implying the possibility to use effectively tables at 2.8 ATA for 330 minutes (eg, US Navy table 6) for the treatment of severely patients. This result agreed with the general recommendation followed by most hyperbaric facilities in the world that the standard practice of US Navy table 6 is adequate for

most of neurological DCS [2]. Our data clearly indicate also that US Navy table 5 or equivalent is sufficient for the treatment of divers with minor neurological symptoms with a recovery rate of 96%. This finding strengthens our opinion that this regimen may be used not only for musculoskeletal pain or skin bends, as recommended in conventional practice, but also for neurological decompression illness with a low initial severity score at presentation (Table 4). Although short oxygen table has been experimentally recognized as being the best compromise between optimal pressure for reducing bubble size and maximum safe pressure at which oxygen can be breathed [31], published data to support the use of other treatments in injured divers and comparison with other procedures are lacking. In a previous study reviewing 20-year experience of DCS treatments, Leitch and Green [7] showed that oxygen tables with initial air recompression to 6 ATA (US Navy 6A) provided no benefit in DCS cases, which failed to respond satisfactorily at 2.8 ATA. Bond et al [6] emphasized also that divers treated for DCS or cerebral arterial gas embolism had a better recovery when recompressed with regular tables using US Navy tables 5 or 6 compared with enhanced tables with extended recompression time or initial pressure of 6 ATA. However, the authors stated that the increase in successful treatment outcomes for divers treated with regular tables was certainly due to selection bias and the fact that the injured divers were not randomized into the treatment categories. Finally, we could find only a single previous report in which treatment outcomes of helium-oxygen recompression table at 4 ATA (Comex 30) and oxygen tables at 2.8 ATA (US Navy table 6 with or without time extension) were compared [5]. In that study, no significant difference could be demonstrated between the 2 protocols in final clinical scores at discharge, but there was a trend toward a better improvement in the helium-oxygen–treated group of patients between the clinical status at presentation and at completion of initial recompression table. Furthermore, no deterioration was observed after Comex 30 tables contrary to oxygen tables. Besides the advantage of helium-oxygen mixture to maintain higher treatment pressures for reducing bubble size, the authors put forward the specific physical properties of helium as an alternative to nitrogen for allowing a greater outward flux of nitrogen dissolved in tissues. In the present study, there was no evidence to suggest a beneficial role of helium in the resolution of severe symptoms, specifically if we compare the outcomes between both 4-ATA table regimens using either Nitrox mixtures or Heliox mixture (Table 5). For instance, its use in therapeutics remains debated [32].

Not surprisingly, the number of additional HBO received by the patients reflects the severity of spinal cord DCS with a significant risk of worse outcome when more than 5 treatments were required (Table 2). The strong correlation (r = .78) with the initial severity score confirms that this information could be useful for the physician's treatment planning and the assessment of hospitalization duration. Similar result has been yet demonstrated in a previous work using a retrospective review of 100 cases for validation of the Royal New Zealand Navy scoring system with a linear relationship between the number of HBO sessions and the admission clinical score [33].

Table 5 Outcomes for GERS B table (Nitrox mixture) and Comex 30 table (Heliox mixture)

	Full recovery	Sequelae
GERS B, n = 6	2	4
Comex 30, n = 16	9	7

Because a large number of variables are being evaluated for a relatively small sample size of divers, we cannot exclude the possibility that the lack of association with clinical outcome may be due to a lack of statistical power. Furthermore, it is possible that there was no discernable time to treatment effect because delay may have been highly correlated with initial severity score (ie, there may not have been sufficient numbers of severely injured divers with long times to treatment). We believe that our findings should be confirmed by a prospective work with a large cohort of injured divers to better define the appropriate treatment table for severe DCS. Future experiments involving animal DCS models are also needed to assess the benefit of immediate recompression and to explore the optimal delay before bubbles start to grow and lead to tissular damages.

In conclusion, this study reveals that a simple score evaluating initial clinical course before treatment may be useful in predicting severe myelopathy with incomplete recovery. Delay of recompression with oxygen therapy less than 3 hours and enhancement of pressure for initial treatment table do not seem associated with a better outcome of spinal cord DCS.

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