Risk factors and clinical outcome in military divers with neurological decompression sickness: influence of time to recompression

Jean-Eric Blatteau, Emmanuel Gempp, Pascal Constantin and Pierre Louge

Key words
Military diving, decompression sickness, hyperbaric oxygen therapy, recompression, outcome, treatment sequelae

Abstract
(Blatteau J-E, Gempp E, Constantin P, Louge P. Risk factors and clinical outcome in military divers with neurological decompression sickness: influence of time to recompression. Diving Hyperb Med. 2011;41(3):129-34.)
Background: This study was designed to examine the influence of short delay to recompression and other risk factors associated with the development of severe neurological decompression sickness (DCS) in military divers.
Methods: Fifty-nine divers with DCS treated in less than 6 hours from onset of symptoms to hyperbaric recompression were included retrospectively. Diving parameters, symptom latency and recompression delay were analysed. Clinical symptoms were evaluated for both the acute event and one month later.
Results: Median delay to hyperbaric treatment was 35 min (2–350 min). Resolution was incomplete after one month in 25.4% of divers with DCS. Multivariate analysis demonstrated that severe symptoms, classified as sensory and motor deficits or the presence of bladder dysfunction, were predictors of poor recovery with adjusted odds ratios (OR) of 4.1 (1.12 to 14.92) and 9.99 (1.5 to 66.34) respectively. There was a relationship between a longer delay to treatment and incomplete recovery, but the increased risk appeared negligible with an adjusted OR of 1.01 (1–1.02).
Conclusion: Our results suggest that neurological severity upon occurrence is the main independent risk factor associated with a poor outcome in military divers with DCS. Clinical recovery was not dramatically improved in this series when recompression treatment was performed promptly.

Introduction

Divers are at risk of decompression sickness (DCS) caused by bubbles of inert gas that may evolve in the tissues or blood due to supersaturation during decompression. Neurological signs involving the spinal cord and cerebral tissues are predominant in DCS, but the clinical symptoms may vary considerably from minimal subjective sensory abnormalities to paralysis and/or bladder dysfunction that could result in permanent disability.

Numerous, previous studies have been conducted with the aim of identifying possible determinants of outcome in neurological DCS, particularly the clinical presentation before treatment, the symptom latency after surfacing and the delay between the onset of symptoms and recompression treatment.[1–11] However, identification of preponderant factors that might prevent divers from developing severe neurological DCS remains elusive.

The question of whether time to treatment with hyperbaric oxygen (HBOT) influences the clinical outcome in DCS remains unanswered. There is conflicting evidence in the medical literature on whether DCS is more responsive to early rather than late HBOT. It is generally believed that the sooner you treat DCS, the better the outcome will be. However, numerous cases have been observed in which satisfactory outcome was achieved after many hours' delay, and, conversely, poor results were sometimes found in cases treated within the first few hours.[12,13]

In a recent study involving 279 cases of spinal cord DCS, we noted that delay to treatment with a median injury-to-recompression time of 4 h did not influence the recovery after statistical adjustment for confounding variables.[14] However, our analysis revealed that the optimal threshold value may be quoted as 6 h with less beneficial effect on outcome if HBOT was applied thereafter, thus supporting the assumption from previous works.[5,15] Preliminary results in animal studies have shown that DCS might be more responsive to recompression in the first minutes rather than after hours have elapsed. However, to date, there are no data on the potential beneficial effect for shorter delays to treatment in divers.[16]

The aim of this study was, therefore, to investigate the influence of time to treatment in French military divers with neurological DCS promptly recompressed in a hyperbaric chamber available at the diving site. We also sought to identify the role of other risk factors in clinical recovery.

Methods

PROCEDURE

We retrospectively reviewed the available clinical and diving data on 112 military divers treated for DCS in the French Navy from 1980 to 2007, including 66 cases with symptoms indicative of neurological DCS. Diving parameters such as bottom time, maximum depth, inadequate decompression with rapid ascent and repetitive dives within 12 h were

recorded. The following clinical data were also analysed: age, elapsed time between surfacing and initial signs, evolution of symptoms before recompression and time to recompression.

Two investigators reviewed the clinical data in order to ascertain the diagnosis of spinal cord or cerebral DCS, based on the presence of symptoms indicative of CNS injury (paraesthesiae, numbness, motor weakness, ataxia, visual disturbance, altered higher function or speech). Patients believed to have pulmonary barotraumas with cerebral arterial gas embolism (CAGE) were excluded. CAGE was diagnosed in the event of fast ascent or omitted decompression stops, associated with an early onset of altered consciousness, confusion, focal cortical sign or seizure after surfacing.

On the basis of the last neurological examination following all hyperbaric treatment, clinical outcome was grossly determined by the recovery status one month post-injury, i.e., full recovery or presence of residual neurological symptoms defined as persistent objective sensory, motor or urinary disorders.

First-aid normobaric oxygen was routinely administered after detection of initial symptoms. After complete clinical evaluation, all patients underwent recompression treatment with hyperbaric oxygen and standardised intravenous therapy with administration of methylprednisolone (120 mg) and aspirin (250 mg), according to our treatment protocol. Initial recompression complied with French Navy tables, mainly GERS B table, breathing 40% oxygen with 60% nitrogen at 405 kPa followed by staged decompression to surface with 60% and 100% oxygen breathing during 8 hours.[17] Additional HBOT sessions (253 kPa for 70 min), twice daily, were given until the patients fully recovered or until no further improvement could be observed after three further sessions. The study design was approved by the local ethics committee (HIA Ste-Anne Toulon).

STATISTICAL ANALYSIS

Clinical outcome was used as the dependent variable, i.e., full or incomplete recovery. Receiver operating characteristic (ROC) curves were used to find the optimal cut-off level for clinical information, i.e., the level that could discriminate between divers with sequelae and those without sequelae.[19] Univariate analysis was performed with Chi-square or Fisher's exact tests to identify significant variables ($P < 0.05$) predicting incomplete recovery. Variables with a P-value less than 0.20 were used as covariates in multivariate analysis with backward elimination logistic regression to control for potential confounders. Odds ratios (OR), adjusted OR and 95% confidence intervals (95% CI) were calculated. In a separate analysis, comparison for two variables between the injured divers with and without sequelae was performed using the Mann-Whitney U test. Calculations were done using the Sigmastat 3.0 software program (SYSTAT Inc., Richmond, CA).

Results

Fifty-nine of the 66 experienced male military divers with an average age (SD) of 31 (5.4) years were retained for analysis. Three divers with a diagnosis of CAGE and four divers with an elapsed time from onset of symptoms to hyperbaric recompression greater than 6 hours were excluded from this analysis. Of these 59 cases, 37 were classified as spinal cord and 22 as cerebral DCS. Seventeen injured divers reported isolated limb paraesthesiae, while 12 patients presented objective sensory symptoms with decreased pain perception and/or light touch skin sensation at multiple sites. The most common motor dysfunction observed initially was limb weakness, systematically associated with sensory loss ($n = 30$). Cerebral DCS involved mainly unilateral sensory and/or motor deficits of the face and arm with five cases of transient blurred vision, dysarthria, and concentration deficit or behavioural disorder.

Air was the breathing gas in 49 divers, whilst 10 subjects made dives breathing nitrox or tri-mix mixtures. Diving profiles were as follows: mean maximum depth (SD) 40 (12.5) metres' sea water (msw) and mean bottom time (SD) 16 (10.2) min. Four divers performed a provocative decompression schedule (i.e., omitted decompression) and repetitive dives were recorded in seven cases. The median time from surfacing to onset of initial symptoms was 15 min (ranging from immediate to 5 h 40 min).

The median time from onset of symptoms to hyperbaric recompression (time to recompression, TTR) was 35 min (range 2 min to 5 h 50 min). Of the 59 injured divers, 15 had incomplete resolution of neurologic symptoms after one month, all involving spinal cord lesions. Nine divers presented mild symptoms, such as residual paraesthesias or leg muscle strain on exertion, while six divers complained of severe disability, with bladder dysfunction, ataxia due to sensory myelopathy and paraparesis of varying degrees from case to case. Divers with cerebral symptoms did not exhibit sequelae after clinical evaluation and psychometric testing at discharge. The median number of additional HBOT sessions was one (range 0–40), with six patients receiving 20 treatments or more. The number of additional HBOT sessions received by the patients reflects the severity of neurological DCS, with a significant risk of a less favourable outcome when more than five treatments were required. Of the 59 cases studied, 17 were declared medically unfit for diving at discharge.

Univariate analysis revealed that the only two variables statistically predictive of a poor outcome were the severity of initial symptoms ($P = 0.017$) and bladder dysfunction ($P = 0.002$) (Table 1). Incomplete recovery was not associated with age ≥ 35 years ($P = 0.26$), depth ≥ 40 msw ($P = 0.41$),

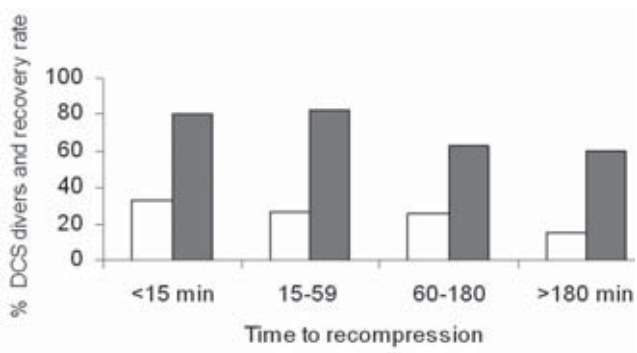
Table1
Analysis of clinical outcome in divers with neurological DCS according to diving data, clinical characteristics and treatment procedures

	Univariate analysis				Multivariate analysis	
Variable	**Full recovery**	**Sequelae**	***P***	**OR (95% CI)**	***P***	**adj OR (95% CI)**
Total (*n* = 59)	44	15				
Age (yr)			0.26	2.64 (0.7–10.1)	N/A	
< 35	37	10				
≥ 35	7	5				
Bottom time (min)			0.94	1.15 (0.35–3.7)	N/A	
< 15	25	8				
≥ 15	19	7				
Depth (msw)			0.41	1.97 (0.6–6.5)	N/A	
< 40	25	6				
≥ 40	19	9				
Repetitive dive			1	0.97 (0.17–5.42)	N/A	
no	38	13				
yes	6	2				
Controlled ascent			1	0.71 (0.07–6.9)	N/A	
no	4	1				
yes	40	14				
Delay onset of symptoms (min)			0.82	1.04 (0.32–3.37)	N/A	
< 15	21	7				
≥ 15	23	8				
Initial symptoms			0.017		0.032	4.1 (1.12–14.92)
Paraesthesia	17	0		N/A		
Sensory deficit	8	4		1		
Motor impairment	19	11		1.16 (0.28–4.76)		
Bladder dysfunction			0.002	14 (2.42–80.95)	0.017	9.99 (1.5–66.34)
no	42	9				
yes	2	6				
Delay onset of symptoms to recompression (min)			0.15	3 (0.82–10.86)	0.03	1.01 (1–1.02)
≤ 90	36	9				
> 90	8	6				
Treatment table regimen (kPa)			0.7	1.32 (0.29–5.92)	N/A	
405	37	12				
283	7	3				

bottom time ≥ 15 min (P = 0.94), repetitive dive (P = 1), rapid ascent (P = 1) or delayed onset of symptoms ≥ 15 min (P = 0.82). Multivariate analysis confirmed severity of initial symptoms and bladder dysfunction as the only independent predictors of poor outcome with adjusted OR of 4.1 (1.12 to 14.92) and 9.99 (1.5 to 66.34) respectively (Table 1).

TTR did not appear to influence the final outcome when univariate analysis was applied with a cut-off level of 90 min determined from ROC analysis (P = 0.15). However, since we observed a P value of less than 0.20, we added the variable "TTR" to the multivariate analysis (Table I). After adjustment, we noted a relationship between TTR and incomplete recovery (P = 0.03), but the increased risk appeared negligible with an adjusted OR of only 1.01 (1 to 1.02). In addition, Figure 1 illustrates the different rates of clinical recovery at one month with respect to TTR, showing

Figure 1
Effect of time to recompression on recovery status at one month post-injury in 59 subjects with neurological DCS; open bar = % subjects; solid bar = % full clinical recovery



that early treatment provided only a small portion of overall recovery.

Separate analysis showed that TTR was not significantly different in divers with full recovery (median time = 29.5 min) when compared with divers with incomplete recovery (median time = 37 min, P = 0.247). Additionally, we compared the prevalence rates of subjects presenting with sequelae who had a TTR of less than 15 min with those of a TTR of more than 15 min. Incomplete recovery was observed in 4 of 21 divers with time to treatment of less than 15 min and 11 of 38 for longer delays, but this difference was not statistically significant (P = 0.6). Among the 30 patients with 'severe' initial symptoms, the median TTR was shorter than for 'mild' cases, (23.5 versus. 60 min, not significant). Moreover, in this subgroup of 30 severe cases, no significant differences were identified after linear regression analysing TTR and presence of residual symptoms (P = 0.103).

Discussion

The prevalence of injured divers with incomplete recovery after neurological DCS is reported between 22% and 29.7% in DAN reports, including large series.[19,20] Our results are in accordance with these data, despite the difference in diving population. We found that variables usually considered as potential risk factors such as age, onset of initial symptoms, depth, repetitive dives or provocative decompression profiles, were not associated with residual deficit. On the other hand, the severity of the presenting symptoms and signs was the only predictor of a poor outcome, consistent with previous reports in other diving populations.[3,4,10] The observation that poorer outcome was associated with the need for more HBOT is consistent with previous reports underlining a linear relationship between the number of HBOT sessions and the admission clinical score.[21]

We observed that the influence of TTR on outcome was not clear and a short TTR had little additional benefit on clinical recovery. Indeed, medical evidence supporting the relative importance of TTR in DCS is controversial (Table 2), notably, if we consider that the usual delay for recompression recorded in most clinical studies is quite long with a median time ranging from 6 to 24 hours.[3,10,11] In a study of 49 cases of spinal cord DCI, it was observed that in the severely injured group, treatment within 12 h was more beneficial than after 24 h.[3] In 1,159 DCS cases of the Divers Alert Network (DAN) database from 1987–1990, long delays were associated with less successful initial decompression and a higher incidence of residual symptoms.[22] However, after logistical regression analysis this association between treatment delay and initial recompression disappeared, and the association between treatment delay and residual symptoms after three months became weak. In a study of 466 cases of "*dysbaric disorder*", analysis did not reveal any relationship between the number of hours' delay and the initial, medium and final outcome.[9]

The short time window, from symptom onset to treatment initiation has been examined in some other studies with differing outcomes. In 847 cases of DCS, a clear relationship was shown between improved outcome and earlier HBOT, including cases treated within 15 min.[2] However, pain symptoms were preponderant in this study (91.8% type 1 DCS). In a series of 50 cases of neurological DCS in military divers (a study comparable to ours), the outcome was no different in those recompressed within one hour compared to those treated thereafter.[8] In another series of 96 divers treated for neurological DCS, the delay between emerging from the water and recompression was not different in divers without sequelae (median = 185 min) when compared with divers with sequelae (median = 203 min).[23] A clinical audit conducted on 390 divers having undergone HBOT in less than six hours in Scotland 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.[5] Finally, the latest surveys, on large series of divers with DCS in Scotland or in France, support the view that the optimal time to recompression should be no more than six hours.[14,15,24] A summary of the findings of these various studies is shown in Table 2.

Our study suggests that military divers presenting with less serious neurological symptoms could wait a few hours for recompression therapy with less concern for ultimate successful recovery, whereas patients with neurological symptoms showing objective sensory deficits and motor impairment should be recompressed in as short a time frame as possible. However, our data also revealed that the shortest TTR, i.e., less than 15 min, was not a guarantor of better recovery. This finding is consistent with a previous experimental study indicating that irreversible Wallerian degeneration of the central nervous system from DCS in pigs occurred within a very short period of time, despite recompression treatment as early as 10 min after surfacing.[26] We are aware that the lack of a statistically significant effect of TTR on recovery, specifically in the severely injured group, may be because the small sample size of our study decreases the ability of the analysis to detect small differences. Whether TTR acts as a gradual progression or as a step change is still unresolved and further prospective work is needed to determine the real impact of short TTR on recovery.

In conclusion, our study suggests that neurological severity at presentation is the main independent risk factor associated with a poor outcome from DCS in military divers. Our data also revealed a statistical relationship between a shorter delay before treatment and higher probability of complete resolution of symptoms, but this association does not seem to be of clinical relevance.

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Table 2
Published case series on the relationship between time to recompression treatment and clinical outcome in divers with neurological DCS; * – this significance was lost on multivariate analysis

Studies	Population	*n*	Recompression delay (h)	% recovery at discharge	*P* - value
Kizer 1982[10]	Neurological DCS	26	12–24	84%	< 0.05
			> 24	54%	
Van Hulst 1990[11]	Neurological DCS	48			
		34	< 12	77%	0.052
		14	> 24	43%	
Méliet et al 1990[8]	Neurological DCS	50			
			< 1	92%	NS
			> 2	86%	
Ball 1993[3]	Spinal cord DCS	49			
		21	< 12	73%	NS
		20	> 24	56%	
	Severe cases	24			
		9	< 12	55%	0.008
		12	> 24	18%	
Blatteau et al 2010[14]	Spinal cord DCS	279			
		197	≤ 6	73%	0.023*
		82	> 6	42.3%	OR 1.8 (1–3.3)
			Median delay (min)		
Boussuges et al 1996[23]	Neurological DCS	96			
	full recovery		185		NS
	incomplete recovery		203		
Stipp 2007[15]	Neurological DCS	343	243		< 0.05

system. The opinions and assertions contained herein are those of the authors, and are not to be construed as reflecting the views of the French Navy.

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Jean-Eric Blatteau, MD, PhD, DSc, is chef de l'Equipe Résidante de Recherche Subaquatique Opérationnelle (ERRSO), Institut de Recherche Biomédicale des Armées (IRBA), Toulon, France.
Emmanuel Gempp, MD, Pascal Constantin, MD, and Pierre Louge, MD, are médecins adjoints du Service de Médecine Hyperbare et Expertise Plongée (SMHEP), Hôpital d'Instruction des Armées Sainte-Anne, Toulon. France.

Address for correspondence:
Dr Jean-Eric Blatteau
ERRSO-IRBA, BP 20545
83041 Toulon cedex 9. France
Phone: *+33-(0)4-8316-2862*
E-mail: *<je.blatteau@infonie.fr>*