Neurological Decompression Illness and Hematocrit: Analysis of a consecutive series of 200 recreational scuba divers.

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Newton HB, Burkart J, Pearl D, Padilla W. Neurological Decompression Illness and Hematocrit: Analysis of a consecutive series of 200 recreational scuba divers. Undersea Hyperb Med 2008; 35(2):99-106. Neurological complications are common in recreational divers diagnosed with decompression illness (DCI). Prior reports suggest that hemoconcentration, with hematocrit values of 48 or greater, increase the risk for more severe and persistent neurological deficits in divers with DCI. Herein we describe our experience with neurological DCI and hematocrit values in a large series of consecutively treated divers. We performed a retrospective chart review of 200 consecutive recreational divers that received treatment for DCI. Standard statistical analyses were performed to determine if there were any significant relationships between diving-related or demographic parameters, neurological manifestations, and hematocrit. In 177 of the 200 divers (88.5%), at least one manifestation of neurological DCI (mild, moderate, or severe) was present. The median hematocrit value was 43, for both male and female divers, with a range of 30 to 61. Hematocrit values did not correlate with diver age or level of diving experience. In male divers, the hematocrit did not correlate with neurological symptoms, including the sub-group with values of 48 or greater. In contrast, female divers with hematocrit values of 48 or greater were significantly more likely to develop motor weakness ($p=0.002$, Fisher's exact test) and an increased number of severe sensory symptoms ($p=0.001$, Kendall's tau statistic). Neurological complications are common in recreational divers treated for DCI. Hematocrit values of 48 or higher were correlated with the presence of motor weakness and severity of sensory symptoms in female divers. The hematocrit did not correlate with neurological DCI in male divers.

INTRODUCTION

Although uncommon, severe injury and death can occur during recreational scuba diving. The Divers Alert Network reports that between 900 and 1,000 divers each year are treated for dive-related complications (1). In these patients, the major symptoms often involve dysfunction of the nervous system. In recreational divers, the most common locations for injury are the spinal cord and the cerebral hemispheres (2-4). Spinal cord involvement that arises during neurological injury from decompression sickness (DCS) is most commonly theorized to occur via nitrogen bubble-induced impairment of spinal venous drainage. Divers with mild or severe neurological symptoms or signs and an appropriate dive profile (i.e., dive depth of 30 fsw or deeper) would be classified as having the type II, or severe, form of DCS (2-4). Damage to the brain is more common after cerebral arterial gas embolism (AGE), with gas bubble obstruction of the neural vasculature. In many divers, the exact mechanism of neurological injury cannot be determined, and so the event

must be classified with the more global term DCI (decompression illness), which includes bubble disease from both DCS and AGE. Previous reports have suggested that divers with hemoconcentration and hematocrit values of 48 or higher are at greater risk of developing neurological DCI, with more severe and persistent manifestations (5).

In a previous report, we described the neurological manifestations of a series of 200 consecutive recreational divers evaluated and treated for decompression illness in Cozumel, Mexico (6). Neurological signs and symptoms were common, affecting more than 80% of the cohort. In the current study, we continue the analysis of this large series of patients, and describe the relationship between neurological manifestations and the hematocrit values noted at presentation to the Emergency Department.

MATERIALS AND METHODS

We performed a retrospective chart review of 200 consecutive recreational divers that received treatment for DCI at the Hyperbaric Medicine Unit in Cozumel, Mexico. The record of each diver was screened for an extensive set of diving-related, demographic, general medical, and neurological parameters. An extensive neurological examination was performed on each patient at the time of admission to the Hyperbaric Medicine Unit, and included an assessment of mental status and speech, cranial nerve function, motor function and reflexes, gait and station, and sensory function; the vast majority of the examinations were performed by a single physician (WP). Repeat neurological examinations were performed during hyperbaric oxygen (HBO) treatment, if there was a question of neurological instability. All patients underwent repeat neurological examinations after the completion of each HBO treatment session.

For the purposes of our analysis, Type I DCS/DCI was defined as divers with symptoms that could include arthralgia, myalgia, dermal manifestations, fatigue, restlessness, and isolated nausea (6). Type II DCS/DCI was defined as divers with symptoms that could include mild (e.g., paresthesias, dysesthesias) or moderate/severe (e.g., paralysis, paresis, gait abnormality) neurological signs and symptoms, and/or cardiopulmonary signs such as cough and shortness of breath.

Hematocrit levels were measured for each diver at the time of admission to the Emergency Department, using standard techniques (7). Blood samples (5 ml) were taken in citrate tubes and subjected to microcentrifugation (4,000 rpm x 5 minutes). The hematocrit values were then measured as the ratio of the globular volume to the volume of total blood.

Standard non-parametric statistical analyses were performed to determine if there were any significant relationships between diving-related or demographic parameters, neurological manifestations, and hematocrit values: the Pearson Rank Correlation Coefficient, Fisher's exact test, Wilcoxon test, and Kendall's tau statistic. Differences between groups were considered statistically significant at $P < 0.05$. No adjustments were made for multiple comparisons in this observational study.

RESULT

Demographic and dive-related information is listed in Table 1. The median age of the cohort was 40 years, with 136 male (68%) and 64 female (32%) divers. The median number of previous dives was 100, with a range of 1 to 18,000, suggesting that the cohort was quite experienced. All of the divers had had at least 1 recent dive before their episode of DCI (median 6 dives). Dive depth data was not available for the entire cohort. The median

onset of symptoms after the completion of the dive was 60 minutes; however, the range was quite wide (ascent – 5,750 minutes), with several divers arriving at the Emergency Department a few days after the completion of their diving activity. Forty-four of the divers (22%) described a rapid ascent at the conclusion of the dive. The median hematocrit value was 43, for both male and female divers, with a range of 30 to 61. Thirty divers (15%) were noted to have hematocrit values of 48 or higher; 22 of this cohort were male (16.2% of all male divers) and 8 were female (12.5% of all female divers).

Table 1. Demographic and Dive Data

Parameter	N	% Total	Median	Range
Age			40 yrs	12-78
Sex	200			
male	136	68		
female	64	32		
Total Dives			100	1-18,000
Recent Dives			6	1-26
Onset Sx			60 min.	ascent – 5760 min.
Rapid Ascent?	44	22		
Hematocrit	200		43	30-61
male			43	
female			43	
Hct ≥ 48	30	15		
male	22	16.2		
female	8	12.5		

Abbreviations: Sx – symptoms, yrs – years, min. – minutes, Hct – hematocrit

The neurological manifestations of the cohort, noted at the time of admission to the Hyperbaric Medicine Unit, are listed in Table 2 (please see page 102 and for a complete description, see reference 6). An overview of the data reveal that in 177 of the 200 divers (88.5%), at least one manifestation of neurological DCI was present, consistent with a Type II presentation of DCS/DCI. Twenty of the remaining 23 subjects had Type I DCS/DCI, with the presence of one or more of the minor symptoms or signs. The remaining 3 divers had predominantly pulmonary symptoms, also consistent with Type II DCS/DCI. The most common neurological symptoms were mild sensory disturbances, consisting of either paresthesias or dysesthesias, or a combination of both. Paresthesias were most common, described by 135 divers (67.5%), and were quite variable in distribution (e.g., hemisensory, single limb). Dysesthesias had a similar, variable pattern of distribution and were noted in 100 divers (50%). Incoordination was observed in 58 divers (29%) and was manifested by gait ataxia and imbalance, and/or limb ataxia. Dizziness (non-vertiginous) was relatively common, described by 47 divers (23.5%). This is in contrast to true vertigo, which was much less frequent and only documented by 15 divers (7.5%). Weakness was present in 42 divers (21%) and had a variable pattern of distribution. Other less common symptoms included nausea and emesis (19%), headache (13%), abnormal nystagmus (12%), reflex abnormalities (13%), visual disturbances (8%), and behavioral disturbances (5%). Painful skin symptoms were noted in 18 divers (9%), and was reported to be highly significantly different between male and female divers ($p < 0.001$, Fisher's exact test) in our previous publication (6). Uncommon neurological manifestations suggestive of severe injury included decreased level of alertness (3%), limb paralysis (2.5%), and limb anesthesia (1%).

Table 2. Neurological Manifestations

Parameter	# Divers	% of total
Paresthesias	135	67.5
male	97	71.3
female	38	59.4
Dysesthesias	100	50
male	69	50.7
female	31	48.4
Incoordination	58	29
male	44	32.4
female	14	21.9
Dizziness	47	23.5
male	33	24.3
female	14	21.9
Weakness	42	21
male	30	22.1
female	12	18.8
Nausea and emesis	38	19
male	23	16.9
female	15	23.4
Headache	26	13
male	14	10.3
female	12	18.8
Nystagmus	24	12
male	17	12.5
female	7	10.9
Reflex abnormality	26	13
male	19	14.0
female	7	10.9
Skin symptoms - painful	18	9
male	2	1.5
female	16	25.0
Visual disturbance	16	8
male	11	8.1
female	5	7.8
Vertigo	15	7.5
male	12	8.8
female	3	4.7
Behavioral disturbance	10	5
male	6	4.4
female	4	6.3
Fasciculations	8	4
Decreased level of alertness	6	3
Hearing loss	5	2.5
Paralysis	5	2.5
Bladder dysfunction	4	2
Anesthesia	2	1

The statistical analysis of diving-related parameters, neurological manifestations, and hematocrit values was applied consistently to all groups (i.e., analyzed the same for male and female divers), and are listed in Table 3. Hematocrit values did not correlate with diver age (rank correlation = 0.053), level of diving experience (rank correlation = 0.003), or gender (p = 0.6, Wilcoxon test). For the cohort as a whole, the hematocrit values were not significantly associated with the risk of neurological DCI (motor symptoms, p = 0.4; sensory symptoms, p = 0.2) or the severity of neurological injury (rank correlation = 0.05). In the cohort of male divers, the hematocrit did not correlate with the occurrence of severe sensory (p=0.27, Wilcoxon test) or motor (p=0.86, Wilcoxon test) symptoms, including the sub-group of 22 men with values of 48 or greater. In contrast, the 8 female divers with hematocrit

Table 3. Statistical Analysis of Diving-Related Parameters, Neurological Manifestations, and Hematocrit.

Parameter	Statistical Result	Statistical Test
diver age	0.053	rank correlation
diver experience	0.003	rank correlation
diver gender	p = 0.6	Wilcoxon test
ENTIRE COHORT		
risk neuro DCI:		
motor Sx	p = 0.4	Wilcoxon test
sensory Sx	p = 0.2	Wilcoxon test
severity of injury	r = 0.05	rank correlation
MALE DIVERS		
Hct ≥ 48:		
severe sensory Sx	p = 0.27	Wilcoxon test
severe motor Sx	p = 0.86	Wilcoxon test
FEMALE DIVERS		
Hct ≥ 48:		
motor weakness	p = 0.002	Fisher's exact test
severe sensory Sx	p = 0.001	Kendall's tau statistic

Abbreviations: Hct – hematocrit, Sx – symptoms, neuro - neurological

values of 48 or greater were significantly more likely to develop motor weakness (p=0.002, Fisher's exact test) and an increased number of severe sensory symptoms (p=0.001, Kendall's tau statistic).

An alternative analysis found that the combination of female gender and motor symptoms was also significant in a multiple regression model for predicting the quantitative hematocrit values (p = 0.006). Due to the relatively low numbers of divers with hematocrit values of 48 or higher (N = 30; male – 22, female – 8), a multivariate statistical analysis could not be performed to adjust for other covariates that might be driving this subset of subjects.

DISCUSSION

These results are in general agreement with other reports and verify that injury to the nervous system is very common in recreational divers treated for DCI, with the majority being mild to moderate in severity (2-4). One or more neurological manifestations of Type II DCS/DCI were present in 88.5% of our cohort. The most frequently observed complications were mild and subjective, and included paresthesias, dysesthesias, and dizziness; however, in other divers, more severe manifestations with objective examination findings were noted, such as weakness, incoordination, and gait difficulty. All levels of the nervous system appeared to be at risk for injury, with the majority of damage localizing to the brain, brainstem, and spinal cord. Overall, the incidence and spectrum of neurological manifestations documented in our cohort was generally consistent with previous reports (1-4,8,9). Some of the most common symptoms, such as paresthesias, dysesthesias, incoordination, weakness, headache, dizziness, and nausea have all been reported in other series of patients with DCI, AGE, and DCS. However, there were several signs and symptoms that had a lower incidence than expected, including seizure activity, confusional states, obtundation and coma, and pure myelopathy (6).

One potential limitation of this dataset is the high overall incidence of Type II neurological DCS/DCI – 88.5%. The incidence of some of the mild, subjective neurological manifestations (e.g., paresthesias, dysesthesias) were higher than some reports in the literature, and suggested that less objective signs and symptoms may have been "overcalled" by the involved hyperbaric physicians. However, this is unlikely since many of the divers had other neurological signs and symptoms, in addition to paresthesias and dysesthesias, some of which could be readily demonstrated on examination (e.g., weakness, incoordination, visual loss).

The hematocrit is a measure of the packed cell volume or erythrocyte volume fraction of whole blood (7). In normal healthy subjects, the hematocrit can vary up to 3% between measurements taken with a sampling interval of 24 hours up to 2 months (10). Taking within-subject and seasonal variation into account, the hematocrit can vary up to 15% in healthy subjects (e.g., from 0.40 to 0.46). The hematocrit is commonly analyzed using various types of automated laboratory devices, as well as by microcentrifugation methods (7,11). Microcentrifugation techniques have been demonstrated to measure hematocrits with the same accuracy as automated analyzers in the Emergency Department setting (r = 0.96) (7). However, there is a slight tendency for centrifuge-based methods to overestimate the true volume of erythrocytes (7,11). This phenomenon is most noticeable when using low centrifuge speeds (i.e., less than 4000 x G) and can be minimized by using faster protocols (e.g., 28000 x G for 1 minute, 12000 x G for 5 minutes) (7). Based on these studies, the centrifuge-based methods for hematocrit determination used in this report appear to be accurate and adequate for statistical analysis.

It has been well documented that plasma volume contraction and hematoconcentration occur in DCI, secondary to increased vascular permeability and plasma leakage (4). This was initially demonstrated in animal models, in particular some of the early dog studies by Bove and Hallenbeck (12-14). In one of these studies, the presence of an elevated hematocrit was associated with a poorer prognosis for recovery (13). Further reports have documented that the hematocrit increases during the first hour or so after exposure to decompression stress, and then slowly normalizes within 24 hours (15). In human experimental subjects and divers, there is considerable evidence linking the presence of an elevated hematocrit and DCI, ranging from 40% to 80% of cases (16-18). However, the hematocrit may remain normal, or even transiently decreased, in asymptomatic divers (19,20).

In the seminal paper by Boussuges and colleagues, data from a consecutive series of 58 sport divers and 16 control divers was analyzed, suggesting a link between hematocrit and neurological DCI (5). All of the sport divers had suffered an incident of neurological DCI, and underwent serial neurological examinations before, during, and after hyperbaric oxygen treatment, including a follow-up examination at one month after the incident. There was no overall difference in hematocrit levels between divers with neurological DCI and controls. However, in the sub-group of divers with persistent neurological sequelae, the median hematocrit was significantly higher in comparison to control divers (47.5 vs. 42.5; $p = 0.01$) and divers without sequelae (47.5 vs. 42; $p < 0.05$). In addition, hematocrit levels of 48% or greater were significantly correlated with the presence of persistent neurological deficits at one month after the incident of DCI, in male and female divers ($p = 0.01$). The hematocrit levels did not correlate with the depth of dive, including the sub-group with levels of 48% or higher.

The data of the current report were not designed to answer the same questions as outlined for the Boussuges study. In particular, neurological examination results were not available at one month after the diving incident for the majority of patients. Therefore, hematocrit levels could not be correlated with the presence of persistent neurological deficits. However, our large consecutive series of patients was able to verify whether or not hematocrit levels, drawn at the time of medical evaluation for DCI, had immediate predictive value for various dive-related parameters and the development of neurological complications. In our series, the hematocrit levels did not correlate with diver age, level of experience, or gender. Similar to the Boussuges study, hematocrit values did not correlate with the overall risk for developing neurological DCI, including motor symptoms, sensory symptoms, or severity of injury (5). However, in contrast to that study, the hematocrit values of our male divers (overall and ≥ 48) did not correlate with the presence of neurological manifestations, such as severe sensory or motor symptoms. For the female sub-group ($N = 64$), hematocrit levels of 48 or higher did have significant predictive value and correlated with the presence of motor weakness ($p = 0.002$) and an increased number of severe sensory symptoms ($p = 0.001$). This type of gender specificity was not present in the Boussuges study, possibly due to the small number of female divers ($N = 10$) in their DCI cohort. In addition, previous studies of neurological DCI and DCS have also never reported a gender bias towards female divers (2-4,8,9). The only report to suggest a gender specific aspect to neurological DCI was our initial analysis of the Cozumel cohort (6). In that report, it was noted that female divers were significantly less experienced than the male divers ($p = 0.025$) and were more likely to develop painful skin symptoms ($p < 0.001$).

It remains unclear why elevated hematocrit values should be more predictive in female divers in regard to developing neurological complications of DCI. There are no reports in the literature describing hormonal effects or other gender-specific characteristics that could mediate a gender bias in the neurological manifestations of DCI. Further research into the possible mechanisms of gender specificity in neurological DCI, using large consecutive cohorts of recreational divers with a significant proportion of women, will be necessary to clarify this issue.

One of the limitations in the data of our study of hematocrit and neurological DCI is the timing of the initial emergency department evaluation and screening bloodwork. An important assumption of the statistical analysis was that all of the hematocrit values were drawn at a time when the patient's vascular compartment continued to reflect any hematoconcentration that might have occurred during the dive. As noted above, hematoconcentration secondary to DCI tends to resolve within 12 to 24 hours after the completion of the dive (15). In our cohort there were ten divers (5%) with onset of symptoms between 12 and 24 hours after the completion of the incident dive (6). This delayed cohort consisted of 8 males and 2 females, with onset of symptoms at approximately 12 and 24 hours, in three divers and seven divers, respectively. It is very likely in this group of divers, if hematoconcentration occurred as part of the process of DCI, that the hematocrit values recorded at the time of evaluation in the Emergency Department were not an accurate reflection of the hematocrit at the conclusion of their dive. The hematocrit values of these divers were either normal or close to normal by the time they arrived for evaluation of their symptoms. Therefore, our dataset may be underestimating the correlation between elevated hematocrit values and the risk of neurological DCI. This may be especially true for males, which comprised 8 of the divers in the delayed group (4% of the total cohort).

These data re-emphasize the potential importance of hydration status in recreational diving, especially for female divers. If the diver is not careful to aggressively re-hydrate in between dives, they may be somewhat dehydrated and intravascularly depleted during the next dive, leading to less efficient off-gassing and greater susceptibility to neurological DCI. In addition, recreational divers should be more cognizant of behaviors that can lead to poor hydrational status and dehydration, in particular the consumption of alcohol. Ethanol induces a diuresis by inhibiting the release of vasopressin (i.e., antidiuretic hormone) from the posterior pituitary gland (21). The mechanism for this affect appears to be ethanol-induced inhibition of calcium currents in the nerve terminals of the neurohypophysis (22). Significant consumption of alcohol, especially in the context of multiple, deep dives over several days to a week, might lead to chronically reduced hydrational status and a higher risk for neurological DCI.

In conclusion, the results of this study indicate that neurological complications are common in recreational divers treated for DCI, and that in female divers, an elevated hematocrit of 48 or higher is associated with an increased risk of developing neurological DCI, in particular motor weakness and severe sensory symptoms. An elevated hematocrit was not predictive of neurological DCI in male divers.

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