Neurological manifestations of decompression illness in recreational divers - the Cozumel experience.

H.B. NEWTON[1,2], W. PADILLA[3], J. BURKART[1], D. K. PEARL[4]

[1]*Division of Neuro-Oncology and Dardinger Neuro-Oncology Center, Ohio State University Medical Center & James Cancer Hospital and Solove Research Institute, Columbus, Ohio, USA,* [2]*Hyperbaric Medicine Program, Ohio State University Medical Center, Columbus, Ohio, USA.* [3]*Hyperbaric Medicine Unit, Subaquatic Safety Services, Cozumel, Mexico,* [4]*Department of Statistics, Ohio State University, Columbus, Ohio, USA*

Newton HB, Padilla W, Burkart J, Pearl DK. Neurological manifestations of decompression illness in recreational divers - the Cozumel experience. Undersea Hyperb Med 2007; 34(5):349-357. Neurological signs and symptoms are common in recreational divers with decompression illness (DCI). The spectrum of neurological manifestations, temporal profile, and laboratory findings are described in a large series of 200 consecutive recreational divers treated for DCI. The Hyperbaric Medicine Unit charts of 200 recreational divers treated for DCI were reviewed and analyzed. The cohort was mainly male, with a median age of 40 years, and quite experienced, with a median of 100 prior dives. In 44 divers (22%) a rapid ascent was documented. The median time to onset of neurological symptoms was 60 minutes after surfacing. One hundred seventy-seven of 200 divers (88.5%) had at least one symptom of neurological DCI at presentation. The most common neurological manifestations were paresthesia, dysesthesia, incoordination, motor weakness, and dizziness. Paresthesias were associated with significantly younger (p=0.003) and less experienced (p=0.03) divers. Similar but less significant correlations were noted for dysesthesias. Female divers were significantly more likely to experience painful skin symptoms (p<0.001). Neurological manifestations are common in recreational divers treated for DCI. Neurological DCI and paresthesias are more likely to occur in younger and less experienced divers.

INTRODUCTION

The Divers Alert Network (DAN) reports that between 900 and 1,000 divers each year are treated for diving-related complications (1). Although uncommon, severe injuries and even death can occur during recreational scuba diving (1-4), and the most severe symptoms often involve dysfunction of the nervous system (5-8). The most vulnerable region for neurological injury in recreational scuba divers appears to be the spinal cord; however, the cerebral hemispheres, deep white matter, brainstem, inner ear, and peripheral nerves are also at risk. Spinal cord involvement that arises during neurological injury from decompression sickness (DCS) has been theorized to occur via nitrogen bubble-induced impairment of spinal

venous drainage. Divers with 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 (6,8). Damage to the brain is more common after cerebral arterial gas embolism (AGE), with gas bubble obstruction of the cerebral vasculature. In many divers, the exact mechanism of neurological injury cannot be determined, and so the event is classified with the more global term decompression illness (DCI), which includes bubble disease from both DCS and AGE.

Although numerous reports have determined that injury to the nervous system is common in recreational divers who suffer DCI, the complete spectrum of neurological

manifestations, temporal profile, and laboratory findings have not been described in a large series of consecutive patients. In this report we describe the neurological manifestations noted in a series of 200 consecutive recreational divers treated for DCI.

MATERIALS AND METHODS

A retrospective chart review was performed 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. Statistical analyses (Wilcoxon test, chi-square test) were performed to determine if there were any significant relationships between diving-related or demographic parameters and neurological manifestations.

For the purposes of our analysis, Type I DCS/DCI was defined as divers with symptoms that could include one or more of the following: arthralgia, myalgia, dermal manifestations, fatigue, restlessness, and isolated nausea. Type II DCS/DCI was defined as divers with symptoms that could include one or more of the following: shortness of breath, cough, and other cardiopulmonary signs; paralysis, paresis, incoordination, dizziness, headache, nystagmus, reflex abnormality, visual disturbance, paresthesias, dysesthesias, behavioral changes, decreased level of alertness, and other signs or symptoms consistent with injury to the nervous system.

An extensive neurological examination was performed on each patient at the time of admission to the Hyperbaric Medicine Unit, including an assessment of mental status and speech, cranial nerve function, motor function and reflexes, gait and station, and sensory function; the vast majority of these examinations were performed by a single physician (WP).

Hematocrit was measured by standard blood microcentrifugation for each diver at the time of admission to the Emergency Department, before transfer to the Hyperbaric Medicine Unit (9).

RESULTS

The cohort consisted of 200 consecutive recreational scuba divers treated for DCI while diving in Cozumel (see Table 1). There were 136 males (68%) and 64 females (32%), with a median age of 40 years (range 12 to 78 years). The diving experience of the cohort was advanced, with a median of 100 dives before the DCI incident (range 1 to 18,000 dives; 24 divers with 2,000 or more logged dives; mean number of previous dives - 905). The median number of recent dives (i.e. just prior to the incident of DCI) was 6, with a range of 1 to 26. In 44 of the divers (22%), a well-documented, rapid ascent at the end of the dive resulted in DCI (as

Table 1. Demographic and Dive Data

Parameter	N	% Total	Median	Mean	Range
Age			40 yrs	40.2 yrs	12-78
Sex	200				
male	136	68			
female	64	32			
Total Dives			100	905	1-18,000
Dives ≥ 2,000	24[a]	12			
Dives ≥ 1,000	39[a]	19.5			
Recent Dives[a]			6	6.73	1-26
Onset Sx[b]			60 min.	173 min.	ascent – 5760 min
Onset Sx – ≤ 15 min.	64[b]	32			
Rapid Ascent?	44	22			
Hct			43%	43.3%	30-61%

[a]1 diver with missing data   [b]2 divers with missing data

reported by the diver or dive buddy). Although the exact maximum depth of the incident dive was not available for the entire cohort, all of the dives were deeper than 30 fsw. The median time for onset of symptoms at the conclusion of the dive was 60 minutes. However, the range was quite broad and included many divers with symptoms that occurred during ascent and another diver with onset at 5,760 minutes (96 hours) after ascent to the surface. In fact, six divers had a significantly delayed onset of symptoms (range 1,000 to 5,760 minutes). In a sub-group of divers (N = 64, 32%), symptom onset was rapid and occurred during ascent or within 15 minutes of surfacing. The median hematocrit for the cohort was 43%, with a range of 30% to 61%.

Non-neurological manifestations of DCI are summarized in Table 2. The most common non-neurological signs and symptoms were arthralgia (70 divers; 35%), fatigue (53 divers; 26.5%), dermatological signs (usually cutis marmorata; 43 divers; 21.5%), myalgias and muscle pain (39 divers; 19.5%), non-painful skin symptoms (26 divers; 13%), and shortness of breath and chest pain during ventilation (22 divers; 11%). In more than half of the divers

Table 2. Non-Neurological Manifestations

Parameter	# Divers	%
Arthralgias	70	35
Fatigue	53	26.5
Skin manifestations	43	21.5
Myalgias	39	19.5
Skin symptoms – nonpainful	26	13
Pulmonary	22	11
Restlessness	10	5
Lymphedema	3	1.5

with skin-related DCI, there were concomitant neurological manifestations.

Neurological symptoms and manifestations of DCI are listed in Table 3, (see page 352). The most common neurological symptoms were sensory disturbances, consisting of either paresthesias or dysesthesias, or a combination of both. Paresthesias were most common, described by 135 divers (67.5%), and quite variable in distribution. Some divers had a hemisensory pattern (i.e., cortical), while others had only a single limb (monosensory deficit; cortical or spinal cord) or two or more limbs affected (e.g., both legs, both legs plus one arm; spinal more than cortical). Dysesthesias had a similar, variable pattern of distribution and were noted in 100 divers (50%). Dermatomal patterns of sensory dysfunction were uncommon. 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, similar to the sensory abnormalities noted above. The most common forms of weakness were hemiparesis (cortical) and paraparesis of the lower extremities (spinal cord); monoparesis was also noted in some patients. Nausea and emesis (N = 38, 19%), as well as headache (N = 26, 13%), were relatively common and could occur as separate symptoms or, as noted in a few cases, could occur concomitantly. In the 5 divers (2.5%) where these symptoms occurred simultaneously, this was an ominous sign and suggestive of a large bubble load in the brain, with elevation of intracranial pressure. Abnormal nystagmus was present on examination of 24 divers (12%), but the type of nystagmus (e.g., horizontal end-gaze, torsional, up-beat) was not specified. Similarly,

Table 3. Neurological Manifestations

Parameter	# Divers	%
Paresthesias	135	67.5
Dysesthesias	100	50
Incoordination	58	29
Dizziness	47	23.5
Weakness	42	21
Nausea and emesis	38	19
Headache	26	13
Nystagmus	24	12
Reflex abnormality	26	13
Skin symptoms - painful	18	9
Visual disturbance	16	8
Vertigo	15	7.5
Behavioral disturbance	10	5
Fasciculations	8	4
Decreased level of alertness	6	3
Hearing loss	5	2.5
Headache & Nausea and emesis	5	2.5
Paralysis	5	2.5
Bladder dysfunction	4	2
Anesthesia	2	1
Seizures	0	0.0
Bowel dysfunction	0	0.0

in an acute nervous system injury from DCI, reflex asymmetries are slow to develop, as sometimes noted in stroke patients. Painful skin symptoms were noted in 18 divers (9%). Visual disturbances were present on examination of 16 divers (8%), and included monocular loss, cranial nerve III palsy and Weber's syndrome, and hemianopsia. Behavioral disturbances consisted of excessive anxiety, aggressiveness, and emotional lability, and were noted in 10 divers (5%). Uncommon neurological manifestations suggestive of severe injury included decreased level of alertness (N = 6, 3%), limb paralysis (N = 5, variable pattern; 2.5%), and limb anesthesia (N = 2, variable pattern; 1%). Other uncommon manifestations included fasciculations, hearing loss, and bladder dysfunction (i.e., urinary retention). It is important to note that none of these 200 patients exhibited seizures or disturbances of bowel function during their presentation with DCI.

A statistical analysis of the data revealed several interesting relationships, which are summarized in Table 4. The initial analysis revealed that 88.5% of the cohort (177 of 200 divers) had at least one neurological manifestation as defined above, and therefore were consistent with a Type II presentation. Twenty of the remaining 23 subjects had Type I DCS/DCI, with the presence of one or more of the minor symptoms or signs (i.e., non-neurological, non-cardiopulmonary). The remaining 3 divers had predominantly pulmonary symptoms, and so were also consistent with Type II DCS/DCI. Divers with Type II DCS/DCI (i.e., one or more neurological manifestations) were more likely to be younger (median 39 vs. 46 years; p=0.05, Wilcoxon test) and less experienced (median 90 vs. 289 dives; p=0.06, Wilcoxon test) than divers with Type I DCS/DCI. Paresthesias were also significantly associated with younger (median age 38 vs. 48.5 years, p=0.003, Wilcoxon test) and less

reflex abnormalities were also noted during the examination of 26 divers (13%). However, the specific pattern of the reflex abnormality (e.g., hemiparetic, paraparetic, monoparetic) was not specified. In addition, it is unclear why few divers had abnormal reflexes when a larger cohort had objective weakness (N = 47; weakness + paralysis). It is possible that

experienced (median 79.5 vs. 232 dives, p=0.03, Wilcoxon test) divers. This same relationship was true for dysesthesias in regard to age, but to a lesser extent (median age 38 vs. 44 years, p=0.02, Wilcoxon test), while the degree of diving experience was comparable (median 100 vs. 135 dives, p=0.9, Wilcoxon test).

Table 4. Statistical Analysis of Diving-Related and Neurological Manifestations

Parameter	Estimates	P-value
Type II vs. Type I DCS/I		
age difference	median = 7 years younger	p = 0.05[a]
experience difference	median = 199 dives less	p = 0.06[a]
Paresthesias		
age difference	median = 9.5 years younger	p = 0.003[a]
experience difference	median = 142.5 dives less	p = 0.03[a]
Dysesthesias		
age difference	median = 6 years younger	p = 0.02[a]
experience difference	median = 35 dives les	p = 0.9[a]
Female divers		
experience	median = 66.5 dives less	p = 0.025[a]
painful skin symptoms	odds ratio = 5.23	p < 0.001[b]
Hematocrit		
gender	median = no differen	p = 0.6[a]
motor DCI	median = 1% higher	p = 0.4[a]
sensory DCI	median = no differen	p = 0.2[a]
severity of injury	correlation = -0.09	p = 0.23[c]
Headache		
abnormal behavior	odds ratio = 21	p < 0.001[d]
Onset symptoms ≤ 15 mi		
uncontrolled ascent	43.2%	p = 0.1[d]
controlled ascent	28.8%	

[a]Wilcoxon test; [b]Chi-square test; [c]t-test in regression; [d]Fisher's exact test

Female divers had a similar age distribution compared with males, but were less experienced (median 76 dives vs. 142.5 dives; p=0.025, Wilcoxon test). In addition, female divers were more likely to experience painful skin symptoms (41% with painful skin symptoms, 18% with non-painful skin symptoms, 41% with no skin symptoms) than male divers (3% with painful skin symptoms, 27% with non-painful skin symptoms, 70% with no skin symptoms) (p<0.001, Chi-square test). This gender dichotomy was not present for any of the other neurological DCI symptoms.

The presence of headache was only noted to correlate with the occurrence of abnormal behavior. Seven of 26 subjects with headache (27%) exhibited abnormal behavior in comparison to 3 of 174 subjects without headache (1.7%). This relationship was significant, with an odds ratio of 21 (p < 0.001; Fisher's exact test).

The median hematocrit for the cohort was 43%, with a range of 30% to 61%. The hematocrit values did not correlate with gender (p=0.6, Wilcoxon test), risk for developing neurological DCI (motor symptoms, p=0.4, Wilcoxon test; sensory symptoms, p=0.2, Wilcoxon test) or with the severity of injury in affected divers (r=0.05, Rank correlation).

A sub-analysis was also performed on divers that had onset of symptoms within 15 minutes to determine if the timing of symptoms correlated with the reported rapid versus non-rapid ascent rate. There was a trend for divers with a rapid ascent rate to have onset of symptoms in 15 minutes or less, compared with divers without a rapid ascent (43.2% versus 28.8%). However, the difference did not reach statistical significance (p = 0.1, Fisher's exact test).

DISCUSSION

This report describes the neurological manifestations of DCI in one of the largest consecutive series of recreational scuba divers in the literature. Our findings are consistent with previous observations and verify that injury to the nervous system is common in recreational divers treated for DCI (5,6,8). 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 included paresthesias, dysesthesias, and dizziness; however, in other divers, more severe manifestations were noted, such as weakness, incoordination, and headache. 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.

One of the most important observations gleaned from this large dataset was the inexact nature of the neurological information that was recorded for each diver. Although basic neurological examination data was consistently present, it was still difficult in many cases to differentiate between events primarily involving the brain and brainstem versus damage localized to the spinal cord. Bubble damage involving the brain will most often have a hemi-body pattern of motor loss and/or sensory alteration (i.e., hemiparesis or hemisensory deficit), with abnormal function affecting both limbs on the same side (e.g., arm and leg weakness or alteration/loss of sensation, increased reflex activity on that side, presence of Babinski's sign). In the spinal cord, bubble damage will usually present with a myelopathic pattern in the mid-thoracic area, with less frequent involvement of the cervical region. The examination typically demonstrates weakness and sensory loss in both lower extremities (all four limbs if involving the cervical spinal cord), overactive patellar

and Achilles reflexes bilaterally, crossed adductor reflexes in one or both of the lower extremities, unilateral or bilateral Babinski's sign, and the presence of a sensory level. To more consistently allow treating physicians and researchers to differentiate between brain and spinal cord presentations of neurological DCI, there will need to be refinement and standardization of the data capture forms for use in the field and associated hyperbaric facilities. With the development of the new Scuba Diving Epidemiological Report Form (SERF) by DAN, efforts are underway to ameliorate this problem (10). The SERF was developed to improve data capture for Project Dive Exploration, a large prospective investigation of DCI in recreational scuba divers using downloadable dive computers and depth-time recorders (1). Thus, more extensive information can be captured regarding neurological symptoms and examination findings. However, to maximize the benefit of new forms such as SERF, dive masters, instructors, on-site treatment teams, hyperbaric technicians, and hyperbaric physicians will require further education on the neurological manifestations of DCI and the performance of the neurological examination.

For the recent evaluation of 2002 DAN data, case reports of injured divers were reviewed and classified as either AGE or DCS; ambiguous cases were designated as DCI (11). The criteria for AGE included a rapid ascent, symptom onset in less than 15 minutes post-dive, cases with prominent cerebral neurological symptoms or signs, and symptom duration of greater than 15 minutes. For DCS, the criteria included cases with a dive depth of at least 30 fsw (9 msw), type I DCS symptoms (e.g., joint pain, skin changes, lymphatic changes, fatigue), and type II DCS symptoms (e.g., mild or severe neurological, cardiopulmonary, in addition to pain and constitutional complaints). Based on these criteria, a mechanism most consistent with AGE would account for approximately 25% to

30% of our cohort (rapid ascent 22%, N = 44; post-dive symptom onset less than 15 minutes 32%, N = 64). Therefore, the majority of our divers (70% to 75%) had a clinical and dive profile compatible with the criteria for DCS. This is not surprising, since diving in Cozumel commonly involves multiple deep dives per day, often over multiple days. These dive profiles would be associated with significant residual nitrogen loads within the tissues, as well as the possibility of an increase in the baseline levels of tissue nitrogen over the course of several days of continuous diving. However, based on the recent DAN data, consecutive daily diving is not a prerequisite for injury from DCI/DCS, since many divers become injured on the first day of a diving trip (11).

The spectrum of neurological manifestations found in our cohort was generally consistent with previous reports (1,5,6,8). Some of the common symptoms—paresthesias, dysesthesias, incoordination, weakness, headache, dizziness, nausea—have been reported with similar frequency in other series of DCI patients; however, several signs and symptoms had a lower incidence than expected. In patients with AGE, confusion and coma are common (~40%), and associated with seizures in almost 20% of cases (6,8,12). In the current series, there were no divers with coma and only 8% of patients had a behavioral disturbance or decreased level of alertness that might be equivalent to stupor or confusion. In addition, none of the divers had a seizure as part of their presentation of neurological DCI. Since 25% to 30% of our cohort had a dive profile and symptom onset consistent with AGE, as well as other symptoms suggestive of cerebral involvement, it is difficult to explain why the incidence of coma and seizure activity should be so low. Similarly, since 70% to 75% of our cohort had a dive profile and clinical picture consistent with DCS, it remains unclear why there weren't more divers with an obvious spinal cord presentation (i.e., thoracic or cervical myelopathy). As already mentioned, some of the problem may be related to a lack of detail and clarity in the capture of the neurological examination data of our cohort. Because of this limitation, our findings may not generalize to other large cohorts of injured divers. However, it is also possible that in a large consecutive cohort of divers, such as the current series, that spinal cord damage and myelopathy may be less common than previously suspected.

Several neurological manifestations were related to the age and experience of the divers in this cohort (see Table 4). Divers with Type II DCS/DCI (i.e., one or more neurological manifestations) were more often younger and less experienced than divers with Type I DCS/DCI. This is consistent with DAN data showing that diving injury and death are more likely in less-experienced recreational divers (11). The data is also similar to a report from Sweden that evaluated risk factors for DCS in a population-based, retrospective study of dive masters and instructors (13). Of the 1,742 divers that answered the survey, 190 (10.9%) reported symptoms compatible with DCS. In that cohort, a higher level of education, training, and dive experience seemed to reduce the incidence of DCS. However, like the DAN experience, neurological DCI can also occur in very experienced divers. In fact, 39 of our 200 divers (19.5%) had logged 1,000 or more dives prior to the episode of DCI. In terms of specific neurological manifestations, paresthesias were also linked to younger and less-experienced divers, while dysesthesias were only more likely in younger divers. To the best of our knowledge, this is the first report to find a significant relationship between sensory DCI symptoms, age, and level of diving experience.

Analysis of the data also revealed several gender-related issues. Female divers were significantly less experienced than

male divers and were more likely to develop painful skin symptoms. A gender effect could not be demonstrated for any of the other DCI symptoms. It remains unclear why female divers should be more likely to have painful skin symptoms. There are no reports in the literature suggesting hormonal effects (i.e., estrogen, progesterone) that could mediate a gender difference in the skin manifestations of DCI; further investigations will be necessary.

Headache was noted in 13% of our divers during the presentation of DCI. This is similar to the recent report by DAN, in which headache was noted in 8% to 10% of their series of injured divers (11). In the literature, the incidence of headache in retrospective studies can vary widely, ranging from 0.5% to 42% (14). The lowest incidence of 0.5% was from a study of 935 cases of DCS, the majority of which were military divers (15), while the highest (42%) was noted among 133 aviators evaluated for severe altitude-related DCS (16). Among our DCI patients, headache was significantly correlated with abnormal behavior, and this manifestation was most suggestive of AGE with bubble damage to the brain and increased intracranial pressure. Although headache can occur in both AGE and DCS, it is generally thought to be more common and severe in cases of cerebral embolism (14). The pathophysiology of headache in divers with DCI has not been completely elucidated, but may be related to multiple factors. A large cerebral load of gas bubbles may lead to a general increase in intracranial pressure (6,14). The presence of intravascular bubbles often causes further changes in the brain which exacerbate the process, including reduced blood flow, damage to endothelium, activation of complement and inflammatory cytokines, and regional ischemia to neurons and supportive tissues (17). In addition, arterial bubbles may damage the blood-brain barrier, leading to vasogenic edema and further increases in intracranial pressure.

Once the activity of intracranial inflammatory pathways and cytokines becomes significant, the trigeminovascular system may become activated and mediate headache pain (18). The trigeminovascular system is now thought to be the predominant mediator of pain in patients with migraine and cluster headaches, but has also been implicated in headaches resulting from other intracranial inflammatory processes.

The median time to onset of symptoms in our cohort was 60 minutes, with a broad range that included divers with onset during ascent and others with onset after several days. Overall, this data is consistent with the DAN injured diver cohort, which also had a median onset of symptoms in the range of 1 to 2 hours (11). In addition, we had six divers (3%) in whom symptoms did not manifest for 42 hours or more. This is similar to the DAN experience, in which approximately 5% of their injured divers had delay of symptom onset until 25 to 48 hours or more after surfacing. This is in contrast to the report by Francis and colleagues, who studied the symptom latency in 1,070 cases of central nervous system DCS gleaned from the literature (19). In this series, over 50% of divers became symptomatic within 10 minutes of surfacing, and in only 15% was symptom onset delayed for more than an hour. In fact, only 5 divers (0.47%) had a delay in symptom onset for 48 hours or more. Divers with cerebral DCS tended to have a shorter latency of symptoms than divers with spinal DCS. However, we could not discern a difference in the onset time between cerebral and spinal DCS.

For our cohort, hematocrit was not related to the likelihood of developing motor or sensory DCI, or to the severity of injury in affected divers. This is in contrast to a previous report suggesting that divers with high hematocrits (48% or higher) were more likely to develop neurological DCI and have persistent deficits (20).

CONCLUSION

Neurological signs and symptoms were very common in our consecutive series of recreational divers with DCI. The majority of these complications were mild and included paresthesias, dysesthesias, dizziness, and other sensory alterations. However, in a smaller percentage of divers, neurological complications were quite severe and manifested as weakness, incoordination, vertigo, and other focal findings. The collection of neurological examination data at dive sites and hyperbaric treatment centers will need refinement as we continue to study this important issue. Type II neurological DCS/DCI and paresthesias are more likely to occur in younger and less experienced divers, while dysesthesias appear to be more common only in younger divers. Painful skin symptoms were more common in female divers, while seizure activity and spinal cord involvement were less common than previously reported.

ACKNOWLEDGMENTS

REFERENCES

1. Divers Alert Network. Report on Decompression Illness, Diving Fatalities and Project Dive Exploration: DAN's annual review of recreational diving injuries and fatalities based on 2002 data. Durham, NC: Divers Alert Network, 2004, pp. 21-45.
2. Melamed Y, Shupak A, Bitterman H. Medical problems associated with underwater diving. *New Engl J Med* 1992;326: 30-35.
3. Clenney TL, Lassen LF. Recreational scuba diving injuries. *Am Fam Phys* 1996;53: 1761-1766.
4. Hardy KR. Diving-related emergencies. *Emergency Med Clin N. Am.* 1997;15: 223-240.
5. Greer HD, Massey EW. Neurologic injury from undersea diving. *Neurol Clin* 1992;10: 1031-1045.
6. Newton HB. Neurologic complications of scuba diving. *Am Fam Phys* 2001;63: 2211-2218.
7. Todnem K, Nyland H, Skeidsvoll H, et al. Neurological long term consequences of deep diving. *Br J Indian Med* 1991;48: 258-266.
8. Barratt DM, Harch PG, Van Meter K. Decompression illness in divers: A review of the literature. *The Neurologist* 2002;8: 186-202.
9. Bartfield JM, Robinson D, Lekas J. Accuracy of microcentrifuged hematocrits in the emergency department. *J Emerg Med* 1993;11:673-676.
10. Freiberger JJ, Smerz RW, Denoble PJ, Vann RD. Hawaii deep treatment diving injury data collected with the new DAN injury report form, the scuba diving epidemiological report form (SERF) (abstract). *Undersea Hyperb Med* 2005;32: 304.
11. Divers Alert Network. Report on Decompression Illness, Diving Fatalities and Project Dive Exploration: DAN's annual review of recreational diving injuries and fatalaties based on 2002 data. Durham, NC: Divers Alert Network, 2004, pp. 46-76.
12. Moon RE. Treatment of diving emergencies. *Crit Care Clin* 1999;15: 429-456.
13. Hagberg M, Örnhagen H. Incidence and risk factors for symptoms of decompression sickness among male and female dive masters and instructors – a retrospective cohort study. *Undersea Hyperb Med* 2003;30: 93-102.
14. Cheshire WP, Ott MC. Headache in divers. *Headache* 2001;41: 235-247.
15. Rivera JC. Decompression sickness among divers: an analysis of 935 cases. *Mil Med* 1964;129: 314-334.
16. Wirjosemito SA, Touhey JE, Workman WT. Type II altitude decompression sickness (DCS): U.S. Air Force experience with 133 cases. *Aviat Space Environ Med* 1989;60: 256-262.
17. Barak M, Katz Y. Microbubbles: pathophysiology and clinical implications. *Chest* 2005;128: 2918-2932.
18. May A, Goadsby PJ. The trigeminovascular system in humans: pathophysiologic implications for primary headache syndromes of the neural influences on the cerebral circulation. *J Cereb Blood Flow Metab* 1999;19: 115-127.
19. Francis TJR, Pearson RR, Robertson AG, Hodgson M, Dutka AJ, Flynn ET. Central nervous system decompression sickness: latency of 1070 human cases. *Undersea Biomed Res* 1989;15: 403-417.
20. Boussuges A, Blanc P, Molenat F, Bergmann E, Sainty JM. Haemoconcentration in neurological decompression illness. *Int J Sports Med* 1996;17: 351-355.