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APNEA DIVING: LONG-TERM NEUROCOGNITIVE SEQUELAE OF REPEATED HYPOXEMIA

Lynne Ridgway and Ken McFarland
University of Queensland, St. Lucia, Brisbane, Australia

This article examines the neurocognitive sequelae of repeated exposure to hypoxemia in apnea (breath-hold) divers. A brief review of the literature on the physiological and neurological adaptations involved in the "human diving reflex" is presented. The results from a neuropsychological investigation of N = 21 elite apnea divers are evaluated. Standard neuropsychological tests, with known sensitivity to mild brain insults, included speed of visuo-motor responding, speed of language comprehension, response inhibition, and visual and verbal attention and recall tasks. Results indicated that the breath-hold divers performed tasks within the average range compared to norms on all tests, suggesting that 1–20 years of repeated exposure to hypoxemia including multiple adverse neurological events did not impact on performance on standard neuropsychological tasks. The results are discussed in relation to implications for clinical conditions such as sleep apnea, respiratory disorders, altitude sickness, and recreational apnea activities.

INTRODUCTION

The increasingly popular apnea sports provide neuroscience with an interesting, naturalistic opportunity to examine the effects of repeated acute and chronic hypoxemia in otherwise healthy individuals. The 2002 world freediving championships in Hawaii attracted 92 elite apnea divers from 22 countries participating in dynamic (depth-diving) and static (at rest) events. With the current world record for an unassisted, constant weight dive at 93 meters, and the static breath-hold record at 8 minutes 10 seconds, and the record holders demonstrating apparently intact physiological and cognitive functioning, there is much to learn about how the human brain and body adapt to low oxygen states. This article briefly reviews apnea sports and compares the neurocognitive profiles observed in 21 apnea divers to observations from clinical, occupational, and recreational conditions involving a reduction in the supply of oxygen to the brain.

Apnea Sports: Background

Underwater apnea activities include synchronized swimming, underwater hockey, underwater rugby, big wave surfing, underwater photography, monofin

Address correspondence to Lynne Ridgway, Ph.D., Scholar Clinical and Neuropsychology, c/School of Psychology, University of Queensland, St. Lucia, Brisbane, 4072, Australia. Tel.: +61 7 33935756. E-mail: lynne@psy.uq.edu.au
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swimming, spearfishing, and competition freediving. The sports group currently utilizing the most rigorous training methods, and setting apnea records in dynamic and static conditions, are competitive freedivers.

Adverse neurological events reported during apnea sports include "*shallow water blackout*" (loss of consciousness, LOC) from rapid atmospheric pressure changes encountered at different depths, cognitive difficulties, and occasional temporary loss of motor control (LMC). Competitive freedivers have coined the term "*samba*" to describe the phenomena of temporary LMC. As observed by the first author, the brief (5–10 second) LMC can occur immediately post breath-hold and involves a bilateral motor tremor accompanied by a rapid, fine bobbing of the head. During a *samba*, or LMC, the freediver is conscious, aware of the tremor, and is able to respond to verbal commands (can give a compulsory "OK" signal to judges). The etiology of this temporary LMC post breath-hold has not been studied in elite freedivers. Possible explanations include changes to the interneuronal GABAergic pathways and a tendency for seizure activity in the post-ischemic or post-hypoxic brain (Sloper, Johnson, & Powell, 1980). Alternately, the *samba* may be synonymous with the "jerky tremor" observed in carbon dioxide retention (Kelman, 1980).

Many clinical, comparative, and experimental studies have been conducted examining the physiological aspects of extreme human breath hold activities (Ferretti, 2001; Ferretti & Costa, 2003, for a comprehensive review). However, no specific studies have been published that examine the acute or chronic neurocognitive sequelae of repeated apneas with occasional temporary LMC or LOC in otherwise healthy adults. Clinical evidence that LOC or epileptic activity is observed when brain damage is in the severe spectrum (Auer & Sutherland, 2002) would suggest poorer neurocognitive outcomes in divers who had experienced multiple episodes of LMC or LOC.

Apnea Sports: Physiological and Neurological Considerations

A careful distinction needs to be made between ischemia, hypoxia, and hypoxemia (see Auer & Sutherland, 2002, as an excellent text reference). Ischemia refers to impaired blood flow (as occurs in heart failure); consequently, oxygen delivery is impaired and waste products are not removed from cells. Hypoxia is a non-specific term meaning low oxygen in the atmosphere, blood, or tissues. Hypoxemia refers only to low levels of oxygen in the blood from whatever cause (Auer & Sutherland, 2002).

With pure hypoxemia (as occurs in diving mammals), only oxygen delivery is impaired, not waste removal, as cerebral blood flow is maintained or, more usually, increased during hypoxic events (Auer & Sutherland, 2002). This distinction helps explain why uncomplicated hypoxemia in a living organism does not by itself give rise to brain cell death. During ischemia, neuronal necrosis appears, and hypoxia then modulates the degree of damage (Miyamato & Auer, 2000). This robust finding is demonstrated using the often-cited Levine rat model (Levine, 1960) where, following unilateral carotid artery ligation and subsequent exposure to hypoxia (8% oxygen), the rat brains exhibited neuronal necrosis ipsilateral to the ligation only (Nagata et al., 2000).

Clinically, in humans, the difference between hypoxemia and ischemia is considerable. Cerebral ischemia of only 2 minutes can cause neuronal death, whereas a pure hypoxemic coma of 2 weeks is usually followed by complete recovery (Auer & Sutherland, 2002). Conditions that may lead to pure hypoxemia, uncomplicated by ischemia, may include environmental causes such as rapid exposure to high altitude or clinical disorders such as sleep apnea. Of all the organs in the body, the brain, representing only 2% of total body weight, is the most metabolically hungry, consuming 20% of the body's oxygen (Sokoloff, 1976). This high metabolic usage renders the brain extremely sensitive to rapid changes in oxygen pressure. Table 1 illustrates the clinical changes occurring at varying levels of alveolar pressures of oxygen (P_AO_2) from changes in inspired air at different altitudes above sea level.

Several studies have demonstrated that cerebral oxygen metabolism remains normal in humans experiencing arterial oxygen pressures (P_AO_2) of 35–40 mmHg (Cohen, Alexander, Smith, Reivich, & Wollman, 1967; Kety & Schmidt, 1948) and even at arterial oxygen pressures of ($P_AO_2 < 30$ mmHg) sufficient to cause loss of consciousness and slow EEG waves (Shimojya, Scheinberg, Kogure, & Reinmunth, 1968). Thus, if neither neuronal necrosis nor impaired neuronal metabolism can explain changes in clinical status during hypoxemia, the logical question becomes, What are the mechanisms of changes to behavior and cognitive functioning during hypoxemia? Recent investigations have focused on the role oxygen plays in neurotransmitter functioning and synaptic changes. In particular, GABAergic (Sloper et al., 1980) and acetylcholine (Gibson, Pulsinelli, Blass, & Duffy, 1981) transmitters appear to be affected, although these changes are reversible in days to weeks, coinciding with clinical recovery from pure hypoxic events (Auer & Sutherland, 2002). Gibson and colleagues conclude, "... mild hypoxia impairs brain function *because* it impairs the metabolism of central neurotransmitters..." (Gibson et al., 1981).

Comparative physiology studies have concluded that the physiological and biochemical traits important to surviving low oxygen states evident in diving animals such as seals and sea lions are highly conserved in all vertebrates (Hochachka, 2000). These traits, collectively known as the "diving response," include the body's ability to hold breath (apnea), slow the heart rate (bradycardia), differential constriction or dilation of blood vessels throughout the body according to importance (peripheral vasoconstriction), and the ability to shift to alternate metabolic pathways

Table 1 Effect of hypoxia on function of human brain (Adapted from Gibson et al., 1981, with permission)

Altitude (ft)	P_AO_2 (mm Hg)	Clinical status
Sea level	95	Normal
5,000	85	Impaired dark adaptation of retinal rods and cones
10–15,000	60–45	Impaired concentration & short term memory; hyperventilation
15–20,000	45–35	Lethargy, euphoria, irritability, hallucinations, impaired critical judgment, muscular incoordination
>20,000	35	Loss of consciousness

Note. P_AO_2 = Alveolar oxygen pressure.

according to oxygen availability (hypometabolism of hypoperfused tissues). In all aquatic mammals and birds, blood flow during a dive is restricted or eliminated in peripheral organs (eg., skin, hands, and feet), allowing blood, and therefore oxygen, to be distributed preferentially to the central nervous system (Zenteno-Savin, Clayton-Hernandez, & Elsner, 2002).

Thus, we know that during hypoxemia, peripheral vasoconstriction acts like the body's own triage system, allowing blood, and therefore oxygen, to be redirected according to most critical need, to the heart, lungs, and brain. Similarly, within the brain, there are several compensatory mechanisms utilized when challenged by hypoxemic conditions (see Auer & Sutherland, 2002 for a review). First, general cerebral blood flow is increased through vasodilation. Second, more oxygen is extracted from the blood, and third, hemoglobin gives up its oxygen more readily (the Bohr effect). Finally, increased capillary density has been observed in brains exposed to chronic hypoxia as in adaptation to high altitude (Boero, Ascher, & Arregui, 1999; LaManna, Vendel, & Farrell, 1992). Together, these adjustments mean that the brain's chemical balance achieves a steady state and events during hypoxemia "... do not progress beyond a reversible pathophysiological state of electrical failure and early energy failure ... the consequence that the stage of tissue necrosis is never reached in the brain in hypoxemia" (Auer & Sutherland, 2002, p. 245). In a review of extreme breath-hold diving, Ferretti (2001) concluded that elite divers demonstrated the occurrence of some adaptive mechanisms that allow the prolongation of apnea and the preservation of oxygen stores during dives.

Apnea Sports: Cognitive and Behavioral Considerations

While the neuropathologists (Auer & Sutherland, 2002) declare that there is no neuronal death from prolonged hypoxemia without ischemia, evidence primarily from aviation and respiratory medicine suggests that there are some disturbing acute cognitive and behavioral changes evident when humans are exposed to low levels of oxygen.

First, with a rapid drop of alveolar oxygen pressure to severe levels (<30 mmHg) there may be a dulling of consciousness and memory impairment unless there is sufficient time for adaptation, with inspired oxygen lowered gradually (Auer & Sutherland, 2002; Gibson et al., 1981). Second, there is some evidence of post-hypoxic seizures (Auer & Sutherland, 2002). In addition, there is anecdotal evidence of people claiming to experience changed cognitive functioning during even mild (alveolar pressure of oxygen 60–45 mmHg) hypoxia (Gibson et al., 1981). Table 2 illustrates some popular terms used to describe changes in behavior and cognition observed in humans in etiologically different low oxygen states.

Early reviews on the psychological effects of hypoxia concluded that there was conflicting evidence of sensory, performance and cognitive difficulties during hypoxic states (Tune, 1964). Reviews of the neuropsychological effects of hypoxia have concluded that acute and chronic exposure to hypoxia reveals a range of cognitive and behavioral deficits (Caine & Watson, 2000; Rourke & Adams, 1996). There is some evidence for impaired cognitive functioning associated with acute low oxygen levels occurring in recreational activities like high altitude mountain climbing (Cavaletti & Tredici, 1993; Hornbein, Townes, Schoene, Sutton, & Houston, 1989;

Table 2 Some qualitative descriptors of cognitive and behavioral changes observed in humans during low oxygen states

Conditions/descriptor	Origin	Cognitive or behavioral changes reported	Reference
Clinical			
Chronic obstructive pulmonary disease	Patient populations	Inattentiveness, poor, judgment, motor, incoordination	Adams et al., 1997; Grant et al., 1987
Sleep apnea	Patient populations	Persistent short-term memory impairment. Problems of sustained attention and verbal ability	Gozal et al., 2001; Lewin et al., 2002; Naegele et al., 1998; Redline et al., 1997
Occupational			
Altitude decompression sickness	Commercial and defence forces pilots	Psychomotor difficulties. Frequent tracking errors, poor concentration, vision, and coordination	Ernsting, 1984; Russell, 1948; Sausen et al., 2001
Commercial (breath-hold) abalone diving	Abalone divers (New Zealand)	"Feel distant and fatigued"	A. Drake, personal communication, April 13, 2003
Japanese ama divers	Retrospective neurological interview	9/16 had stroke-like accidents, motor and sensory abnormalities	Kohshi et al., 2001
Recreational			
"HAS" high-altitude stupid	Mountaineers	Judgment, decision errors. Poor memory, orientation, mood, consciousness	Auer & Sutherland, 2002; Hornbein et al., 1989; Ryn, 1971
"AMS" acute mountain sickness		Headache, fatigue, lassitude, dizziness	Roach & Hackett, 2001
"Taravana" to fall crazily	Polynesian pearl divers	Dizzy, visual disturbance, paralysis of extremities	Cross, 1965
"Shallow water blackout"	Apnea divers and spearfishers	Loss of consciousness often associated with depth pressure changes	Sipperly & Mass, 1998
"Samba" loss of motor control	Elite apnea divers	Brief bilateral motor tremor, esp. hands and head, and unfocused gaze	Author observations. Freediving World Championships 2002
"Apnea brain" or "Mooglie"[a]	Elite apnea divers	Poor verbal fluency and production errors, slow information processing	Canadian Freedive Team, personal communication, October 29, 2002

Note. [a]Verbal production error. A fully conscious Canadian diver emerged post long-duration breath-hold and had meant to say "Man, that was ugly." Instead, the neologism "Mooglie" was uttered and has been adopted by the team to describe any "uncomfortable" breath-hold.

Townes, Hornbein, Schoene, Sarnquist, & Grant, 1984), in occupational pursuits such as high altitude flying (Ernsting, 1984; Sausen, Wallick, Slobodink, Bower, & Clarke, 2001) and apnea diving (Kohshi, Katoh, Abe, & Okudera, 2001), as well as in animal models and medical conditions (see Table 2 for summary).

While there are obvious differences between a reduction of oxygen in inspired air at altitude and breath-holding at sea level, there are also significant differences in the onset and duration of hypoxic conditions between mountaineers and apnea divers. Mountaineers experience gradual onset (hours to days), long-duration hypoxic episodes (days to weeks), whereas apnea divers have rapid onset (seconds to minutes), shorter duration (minutes) acute, repetitive (approximately 5–20 per day) epochs. The gradual onset, longer duration would seem to favor the mountaineers as having better opportunity for adaptation to low-oxygen conditions. However, the absolute level of hypoxemia experienced across both conditions is similar. Data from 3 elite apnea divers found hemoglobin oxygen saturation levels (S_aO_2) dropped to between 60 and 57% for the final 60 seconds of 5-minute breath-holds (Stewart, Bulmer, & Ridgway, 2003), making the severity of hypoxemia equivalent to alveolar oxygen pressures (P_AO_2) of around 35 mmHg or the blood gas levels experienced by mountaineers at heights greater than 20,000 feet above sea level.

The existence of persisting or cumulative effects of hypoxia is unclear, with some studies reporting persisting cognitive impairment after resolution from hypoxic conditions (Hornbein et al., 1989; Naegele et al., 1998; Regard, Oelz, Brugger, & Landis, 1989; Townes et al., 1984), and others reporting no significant cognitive impairment once hypoxic conditions have resolved (Clarke, Heaton, & Wiens, 1989; Jason, Pajurkova, & Lee, 1989). In a retrospective interview survey conducted on 16 professional Japanese breath-hold divers, researchers found evidence for transient neurological symptoms occurring during or after dives (Kohshi et al., 2001). The most common symptoms reported were unilateral motor weakness, (7 of 16 cases) and sensory abnormalities (4 of 16 cases), with 13 of the divers reporting occasional episodes of dizziness, nausea, and/or euphoria. The study concluded that deep breath-hold dives may be harmful and cause brain involvement (Kohshi et al., 2001). This study did not report on the long-term neurocognitive status of the divers.

There is additional evidence for impaired neurocognitive functioning in chronic clinical conditions, such as obstructive sleep apnea (Beebe, Groesz, Wells, Nichols, & McGee, 2003; Gozal, Daniel, & Dohanich, 2001; Lewin, Rosen, England, & Dahl, 2002; Redline et al., 1997), and chronic obstructive airways disease (Adams, Victor, & Ropper, 1997; Grant et al., 1987).

The common diagnostic criteria for sleep apnea is if the person experiences apneas of at least 10 seconds each at a frequency of more than 30 times per 7-hour sleep period (Association of Sleep Disorders Centers and Association for the Physiological Study of Sleep, 1979). In severe sleep apnea, patients may experience periods of >60 seconds without a breath resulting in an oxygen saturation (S_aO_2) level of <50% (Kaplan & Sadock, 1998). Some of the cognitive sequelae reported include memory difficulties (Ewing et al., 1980; Stuss, Peterkin, Guzman, & Troyer, 1997; Wilson, 1996), executive dysfunction (Wilson, 1996), praxic disorders, affective disregulation, poor verbal fluency (Armengol, 2000), slowed psychomotor processing and motor speed, (Berry et al., 1989), and vigilance or attentional difficulties (Ewing et al., 1980; Stuss et al., 1997). However, in a comprehensive metanalysis

of studies of people with untreated obstructive sleep apnea Beebe and colleagues (2003) found evidence for impairments in vigilance, executive functioning, and motor coordination but not for intellectual, verbal, or visuo-perceptual skills.

One area of the brain often espoused to be selectively vulnerable to transient alterations to normal functioning is the hippocampus (Lishman, 1998; Zola-Morgan, Squire, & Amaral, 1986). With the hippocampus involved primarily in new learning and memory (Adams et al., 1997), difficulties in those aspects of functioning during hypoxic events would be expected. Several animal models have linked neurotransmitter changes in the hippocampus as an explanation of hypoxic memory deficits (Furling, Ghribi, Lahsaini, Mirault, & Massicotte, 2000; Mazer et al., 1997; Row, Goldbart, Gozal, & Gozal, 2003; Wang, Zhou, Shao, & Tang, 2002).

The complication with many published neuropsychological studies and reviews of deficits supposedly caused by hypoxia is that they utilize patient samples with differing etiological factors including CO poisoning, cardiac arrest, respiratory arrest, near hangings, and drownings. These conditions clearly involve mechanisms such as cerebral ischemia or toxicity in addition to reduced oxygen supply to the brain. Thus, it is hard to conclude what the exact etiology of any revealed neuropsychological deficits could be. Aside from the clinical mechanisms that differ between these conditions, additional psychological factors experienced in chronic life-threatening illnesses confound these studies by potentially impacting on the results of neuropsychological testing (McSweeny & Labuhn, 1996).

Thus, the current research into the long-term neurocognitive sequelae from repeated hypoxemia in a group of otherwise healthy sports persons provides an excellent opportunity to examine any deficits occurring in the absence of ischemia and other medical variables or psychological distress. One goal of this current study was to examine whether repeated apneas, with occasional adverse neurological events, of high frequency and long duration would lead to poorer cognitive functioning on standardized tests. Anecdotal reports from the divers suggest that there are a variety of acute cognitive and neurobehavioral changes experienced during and immediately post breath-holding but that there are no recognized long-term effects.

Standardized, valid, and reliable neuropsychological tests were selected to attempt to capture the anecdotally and previously reported cognitive and behavioral difficulties summarized in Table 2. Selection of baseline neuropsychological assessments was guided by the following criteria. Tests had to be brief enough to allow multiple cognitive domains to be covered in around 60 minutes, have known sensitivity to minor interruptions to neurological functioning, be objectively reliant on diver performance thus reducing possible experimenter bias, and have multiple equivalent forms allowing follow-up testing for later studies examining the acute effects of hypoxemia. A list of the tests selected with references for administration and psychometric properties is provided in Table 3. A new task, a brief explicit memory task, was developed following observations during a feasibility study indicating that divers had difficulty laying down new memories during, and in the first minutes after an extended breath-hold. Equivalent forms of a 4-item visual display were constructed, each depicting three pictures from a standardized, matched, familiar set (Snodgrass & Vanderwart, 1980) and a single common 4-letter word printed in size-36 font.

Table 3 Information and assessment tools in baseline protocol

Assessment protocol	Details/reference for psychometric properties
Demographic data collection	Age, sex, education, medical, and sport history
Basic neurological observations	Visual-motor tracking, coordination/balance
NART-II National Adult Reading Test	Nelson & Willison, 1991
Modified STROOP	Spreen & Strauss, 1998
WAIS-R Digit Span Test	Wechsler, 1981
Symbol Digit Modalities Test	Smith, 1982
Trail Making Test part A & B	Reitan & Wolfson, 1985
Controlled Oral Word Assoc. (FAS)	Spreen & Strauss, 1998
SCOLP—Silly Sentences	Baddeley, Emslie, & Nimmo-Smith, 1992
4-Item explicit memory tas	Psychology Dept., University of Queensland

Additionally, basic neurological observations with known sensitivity to hypoxia were devised (Dr. R. Boyle, personal communication, August 2002). These included asking the divers to perform visuomotor tracking and balance and coordination tasks. Divers were scored according to 3 categories: (a) Able to complete task with no difficulty, (b) Completes task but slow or unsteady, and (c) Has great difficulty or cannot complete. Finally, the National Adult Reading Test-2 (Nelson & Willison, 1991) was selected as an estimate of previous stable intellectual ability. The value of the test lies in the high correlation between reading ability (accuracy of pronunciation) and intelligence in the normal population (Spreen & Strauss, 1998).

It was hypothesized that elite divers with more years practicing apnea diving would score lower on neuropsychological tests compared to standardized norms. Further, a cumulative effect was hypothesized such that the greater the number of negative neurological events experienced (diving-related blackouts, loss of motor control episodes, and previous concussions) by the divers, the worse their current performance would be on standard tests of neuropsychological functioning.

METHOD

Participants

Participants were volunteer elite freedivers recruited via Australian and international organizers of apnea diving competitions (Association International for the Development of Apnea, AIDA). Only participants who spoke English as a first language were included in the study. The group consisted of 10 Australians, 4 Americans, and 7 English freedivers. There were 12 male divers and 9 female divers. Table 4 presents demographic and apnea dive related data for the 21 freedivers.

No payment was offered to divers for their participation. An elite diver was defined as a person who had been selected by their country to compete at an international championship level. With the exception of one 36-year-old diver from the UK, all divers were non-smokers. All divers were in good physical health with no current medical or health concerns. Three divers reported having had a brief concussion during childhood. None of the divers had experienced a negative

Table 4 Demographic information for $N = 21$ (m = 12; f = 9) elite apnea divers

	Range	Mean	SD
Divers age in years	21–68	31	7.2
Years of education	10–18	13.58	2.04
Years breath-hold diving activity	1–20	6.31	6.40
Recent competition dive depth achieved[a]	30–64	47.1	2.53
Recent competition static time achieved[b]	3.4–6.5	4.9	.23
NART-II predicted IQ	85–126	110.70	9.50
LMC[c] episodes reported = approximate	0–20	4.19	5.20
LOC[d] episodes reported	0–5	.86	1.24
Total[e] number of –ve neurological events	0–23	5.1	6.24

[a]Dive depth in meters.
[b] Static time in minutes.
[c]LMC-Loss of motor control. Divers reported their approximate number of "*sambas*."
[d]LOC-Loss of consciousness from "shallow water blackout."
[e]Total number of negative neurological events in life, includes previous concussions, head injury, and dive-related accidents.

neurological event (LOC or LMC) within the 7 days prior to testing. With the exception of a 68-year-old male participant, all were currently engaged in intensive apnea training at the time of testing.

Materials

An information sheet was issued to all divers, coaches, and judges. Divers completed a demographic questionnaire including details of body measurement, education, sporting involvement, and basic medical history.

Procedures

Testing in this study was conducted poolside, in conditions simulating as closely as possible the competition environment, where further assessments examining the acute effects of extended breath-hold activity were planned. Hence, assessments were conducted at public pools with natural background noise. The diver sat opposite the examiner at a desk facing away from the public. A material screen was erected around the testing station to prevent direct visual contact with other divers or officials during the assessment. Divers were asked to follow their usual pre-dive protocol by abstaining from alcohol for 12 hours before testing. Divers had not engaged in any breath-hold activity for 12 hours prior to the baseline assessment. Divers were first asked to read the information sheet and complete the demographic and consent forms.

The order of administration of the neuropsychological tasks was fixed in the sequence indicated in Table 3. Standard neuropsychological tests were administered according to the author/publisher instructions. For the 4-item memory task, divers were asked twice to name each picture and read aloud the word. The stimulus material was removed from sight and divers were asked again to repeat the 4 items and asked to remember them for recall a few minutes later.

Statistical Considerations

As the main neuropsychological tests have standardized published norms, each diver's standard score for each test was calculated. Thus, where available, individual scores were converted to scaled scores to adjust for age, gender, and years of education. Standardized Z scores were calculated for each diver on each test as follows: National Adult Reading Test = Full-Scale IQ predicted from number of errors; Digit Span = total correct Forward and Backward digits recalled; FAS = total correct words generated for each phoneme in 60 seconds; Modified Stroop = time taken on final color-incongruent-words trial; Silly Sentences—Form A = number completed in 2 minutes; Symbol Digit Modalities Test = number of correct substitutions in 90 seconds; Trail Making Test Part B = time taken (including error corrections); Memory task = number of items correctly recalled after a 5-minute delay. For the neurological observations divers were scored according to 3 categories: (a) able to complete task with no difficulty; (b) completes task but slow or unsteady; and (c) has great difficulty or cannot complete.

To allow an examination of differences between divers with less years experience or less number of negative neurological events, two grouping variables were created. First, years of apnea experience was used to create a grouping variable, with Group 1 being divers who had had less than 4 years experience ($n = 9$) and Group 2, divers with between 4 to 20 years experience ($n = 12$). Similarly, a second group variable, negative neurological events (NNE), was formed with 3, approximately equal, groups: Group 1, divers who had between 0 and 2 NNE ($n = 8$); Group 2, divers who had experienced between 3 and 5 NNE ($n = 7$); and Group 3, divers who had experienced 6 or greater NNE in their lives ($n = 6$).

RESULTS

Prior to analysis, data from neuropsychological tests and demographic variables were examined using SPSS. Distributions of scores were found to fit with multivariate assumptions. Missing data for 2 divers on the SCOLP and Stroop were replaced using the series mean (Tabachnick & Fidell, 1996). Univariate outliers detected on age and the number of years engaged in breath-hold activity were retained in the analysis as they were deemed genuine cases from the intended population sample and they did not significantly skew the distribution of scores (Tabachnick & Fidell, 1996). Thus, no cases were deleted from the analysis.

To confirm that the administration of the NART-II was legitimate for the multinational participants, two statistical procedures were used. First, an examination of the correlation between the divers' scores on the NART-II and the diver's level of education revealed a significant Pearson's correlation ($r = .64$, $p < .01$). Second, divers were divided into two groups according to country of birth: Group 1, Australia, ($n = 10$), Group 2, UK or United States ($n = 11$), and data for NART-II, education, years engaged in apnea activity, and NNE were analyzed for differences. No significant differences between the groups by country were found ($F_{(1,18)} = 2.72$, ns). There were significant correlations between the divers' scores on the NART-II and scores on other tests of written language, Stroop ($r = .61$, $p = .003$), and SCOLP Silly Sentences ($r = .537$, $p = .018$).

All divers were observed to complete the neurological tasks and the 4-item memory task at a ceiling level without difficulty. Group means and standard deviations for $N = 21$ divers on each of the neuropsychological tests are reported in Table 5. As a group, the divers all performed within one standard deviation of published norms on each neuropsychological test with age, gender, and education adjusted for where possible. At the individual level, no diver's score was significantly below his or her population normative data, again with demographic adjustments made where available.

To examine the hypothesis that more years engaged in apnea activity would impair performance, correlations and group differences (less than 4 years versus 4–20 years) on neuropsychological test scores were examined. No significant correlations (Spearman's rho) or group difference was found ($F_{(1,19)} = .66$, ns). To examine the hypothesis that the total number of negative neurological events over a lifetime would impact on performance on neuropsychological tests, SPSS Multivariate General Linear Model was employed to compare the 3 NNE groups. The means, standard deviations and univariate F values are reported in Table 5. Again, no significant correlations or group difference was found.

DISCUSSION

This study aimed to quantify any long-term neurocognitive sequelae from frequent, repeated long-duration apnea activity. The hypothesis that elite divers with more years engaged in apnea activity would score lower on neuropsychological tests than norms was not supported. This group of 21 elite freedivers with either 1–3 or 4–20 years engaging in apnea activity performed within the average range compared to standardized norms on a variety of sensitive neuropsychological tests. Similarly, the second hypothesis regarding persisting or cumulative effects, such that divers with the greatest number of negative neurological events (NNE) would demonstrate poorer performance on the tests than those with less NNE was also not supported. This group of 21 elite divers with 0–2, or 3–5, or 6–23 NNE over a lifetime did not differ on their performance on standard neuropsychological tests with known sensitivity to minor interruptions to neurological functioning.

We expected that, given the serious nature of some of the NNE reported in this group, there would be some evidence for impaired neuropsychological functioning, especially considering findings by a Japanese neurosurgery group that MRI scans of 3 ama divers with similar neurological histories demonstrated multiple brain lesions (Kohshi et al., 2001), and other research demonstrating persisting cognitive impairment from hypoxic conditions (Hornbein et al., 1989; Naegele et al., 1998; Regard et al., 1989; Townes et al., 1984). Thus, we can conclude that even if there were any cognitive difficulties associated with the changed neurobehavioral functioning (brief episodes of LOC or LMC) immediately post breath-hold, there were no persisting deficits according to the sensitive neuropsychological tests used in this study. As all the divers tested had not participated in apnea activity for the past 12 hours, nor had any negative neurological events in the past 7 days, we can only suggest that any acute effects of apnea appear to resolve within those time frames. Our findings, while surprising, provide some support for studies demonstrating

Table 5 Z score means, standard deviations (in parentheses), and *F*-values on neuropsychological tests according to the number of lifetime negative neurological events reported for $N = 21$ elite freedivers

	Lifetime negative neurological events					
Neuropsychological test variable	Group ($N = 21$)	0–2 ($n = 8$)	3–5 ($n = 7$)	6–23 ($n = 6$)	$F_{(2,18)}$	Sig. p .05
National Adult Reading Test-II	.71 (.63)	.73 (.60)	.51 (.84)	.92 (.37)	.65	.53
Symbol Digit Modalities Test	.83 (1.83)	1.11 (1.10)	.44 (1.43)	.91 (1.54)	.47	.63
Speed & Comprehension of Language Process (Silly Sentences)	.60 (.96)	.88 (1.22)	.19 (.93)	.70 (.47)	.97	.40
Controlled Oral Word Association (FAS)	.87 (1.20)	.64 (.97)	1.19 (1.46)	.88 (1.27)	.48	.62
Weschler Adult Intelligence Scale—III, Digit Span subtest	.88 (1.03)	.66 (.65)	1.33 (1.42)	.66 (.91)	.98	.40
Trails B	.77 (1.00)	.79 (1.14)	.70 (.85)	.80 (.75)	.02	.98
Modified Stroop Trial "C" (color incongruent words)	.39 (.59)	.69 (.40)	.06 (.74)	.38 (.46)	2.47	.11

Note. All variables have been rescaled such that a low *Z* score reflects poorer performance.

no persisting effects from mountaineers who suffer repeated exposure to hypoxic conditions (Clarke et al., 1989; Jason et al., 1989).

Explanations for the intact cognitive functioning of this group of elite divers include the possibility of an adaptive response to low-oxygen states such as found in mountaineers, some clinical conditions, and physiological studies of apnea divers (Auer & Sutherland, 2002; Boero et al., 1999; Ferretti & Costa, 2003; Hochachka, 2000; LaManna et al., 1992; Lindholm, Sundblad, & Linnarsson, 1999). It was beyond the scope of this study to determine the presence of specific adaptations; however, it is possible that the presence of a physiological adaptive response may be neuroprotective.

An alternative account may be that the divers in this sample were a well-educated group (average 13 years). It may be suggested that the level of education and intellectual ability obscured the detection of cognitive impairment. However, given that the current analysis used scaled scores to adjust for age, gender, and education, this explanation appears unlikely. Correspondingly, the NART-II, with its potential for experimenter bias in the scoring, may have incorrectly estimated the group's intellectual ability, especially when administered across groups with different national accents. However, the examination of the multinational results revealed no significant groups by country effect and there was no obvious experimenter bias or administrative difficulty. As a cautious note, the lack of group statistical significance does not rule out lasting neurocognitive deficits in some apnea divers. More power with a larger sample size or using sensitive MRI scanning may have detected more subtle residual deficits in this population.

The current research with a group of healthy elite apnea divers without medical or psychological adjustment problems or sleep disturbances has provided an opportunity to attempt to isolate the etiology of neurocognitive sequelae from hypoxic conditions. The current findings may be of interest to researchers examining the etiology of cognitive and behavioral changes reported in disorders such as sleep apnea and chronic obstructive airways disease (Lewin et al., 2002; Naegele et al., 1998; Redline et al., 1997). It may be a possibility that factors other than pure hypoxemia are responsible for the poor cognitive outcomes reported in clinical research.

This study focused on the long-term effects of repeated, long-duration apneas with accompanying negative neurological events in healthy adults. The selected tests did not detect any impairment in neurocognitive functioning compared with norms matched for age, gender, and education. However, before concluding that long-duration apneas are without consequence, further studies are required to examine any acute effects after long-duration apneas. It would also be worthwhile to simultaneously examine the physiological and neuropsychological correlates following extended apneas.

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