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NEUROLOGICAL INJURY AND A RETURN TO RECREATIONAL DIVING.

Chris Acott

Introduction

The development of guidelines for a return to recreational diving following any neurological injury is hindered by the lack of objective data. Furthermore, the available data are limited to commercial or military divers.

A return to diving should depend upon a negative response to all of the following questions:

a Will the continued diving make the condition of the diver worse?
b Will the condition of the diver compromise the diver's or buddy's safety in the water?
c Will the condition of the diver predispose to or aggravate a diving illness?

In addition, if the neurological injury was caused by diving, negative answers must be obtained to the following questions before any consideration of a return to diving can be given:

a Did the diver suffer pulmonary barotrauma?
b Was the diver's illness commensurate with the diving exposure?
c Did the diver respond well to treatment?
d Has the diver any residual problems from the diving injury?

Decompression induced neurological injury

Permanent neurological damage due to decompression illness has been known for more than a 100 years and while physicians agree that a diver with any objective neurological deficit after a decompression accident is unfit to dive,[1] the suitability of such a diver to return to diving if the deficit "resolves" is debateable. For example animal model data have demonstrated that diving can induce "silent" central nervous system damage.[2] In addition, there is post mortem evidence that lesions may persist in the spinal cord after decompression injury and without clinically evident neurological residua in humans.[3,4] Palmer[5] and Mork[6] in separate studies have also shown a positive correlation between cerebral vasculopathy and diving in post mortem studies of divers with or without a history of decompression injury. These divers studied, as far as it is known, were not incapacitated in any form. Overall, there is a paucity of objective data showing that nervous system damage, that occurs silently (in the absence of both clinical symptoms and signs) after diving and persists, causes any loss of function or impairs activities of daily living. That is, the presence of an abnormality at post mortem does not indicate an inevitable impairment of function. Nevertheless, on the basis of these animal studies, some physicians maintain that any episode of neurological decompression illness permanently disqualifies a diver. This stance may actually delay or suppress the reporting of symptoms, and hence delay treatment, for fear of subsequent disqualification. Although this argument is especially relevant to professional divers, it is still applicable to recreational divers.

The original observation that decompression injury may cause an encephalopathy must be assessed cautiously as the study lacked either suitable controls or established neuropsychological tests and the subjects continued to work in compressed air,[7] it is noteworthy that similar study outcomes are reported. For example, a very recent Norwegian study showed a positive correlation between central nervous system symptoms (problems with concentration, memory, irritability and depression) and un-

treated symptoms following decompression in both commercial and recreational divers.[8]

Neuro-investigative techniques (CT, MRI, SPECT, PET) have been either too sensitive or too insensitive in the identification of decompression injury residua.[9-12] Evoked response studies are equally unrewarding, only being abnormal in those divers with an obvious deficit.[13]

The use of fluorescein angiography to examine the retina of divers has shown a positive correlation between perfusion deficits and retinal pigment changes in divers who have had decompression illness[14] and may be an indication of subtle cerebral lesions. However, these changes are often seen in older non-diving individuals and so the clinical relevance, particularly with regard to fitness to return to diving is not established.

The electroencephalogram (EEG) has been used to study divers with decompression illness.[15-17] These studies have shown an association between dysbaric illness and EEG abnormalities but are associated with both significant false positive and false negative results.

Neuropsychological screening tests have been used to investigate brain injuries after decompression illness, but the frequency of persistent neurological deficits following treatment varies with the type of neuropsychological assay.[13,18-21] Data can not be compared between studies unless the same neuropsychological tests are used, even if the same population is studied.[22-25] For example, conclusions from a study on Australian abalone fisherman-divers depended entirely upon the test used. Furthermore, neuropsychologists are yet to agree on test standards and diagnostic criteria for disorders in higher mental functions.[17] Hence these tests need further testing and standardisation. However, provided the same tests are administered by the same person they can be, and have been, used to establish the return of a diver's cognitive function during recovery from a decompression injury.[13,26]

Consequently, the primary follow up for a diver who has had a decompression injury remains a meticulous clinical neurological history and examination with emphasis on the diver's mental function. The ideal frequency of these reviews is not established, but there should be one done at least one week after treatment to ensure that a treatable relapse has not occurred. It is now apparent that the pathological processes of decompression illness do not return to normal for at least one month, even following successful treatment.[27] It is reasonable to perform another review 4 or 6 weeks after treatment and it is at this review that a decision can be made about a return to diving. A longitudinal study of 25 recreational divers who had decompression illness, showed that 6 of the 12 divers who were neurologically normal at discharge, had overt neuropsychological problems at one year.[26] This study demonstrated that divers need continued assessment for 12 months after a decompression injury. The overall data suggest that at a minimum: divers should be reviewed one week, 4 or 6 weeks and one year after treatment for decompression illness; and that 4 weeks after treatment is the earliest time to consider a return to diving. I suggest further reviews be conducted 3 and 6 months after treatment and that fitness to continue diving be assessed at each of these reviews. A return to diving should be contingent upon a decompression injury that is commensurate with exposure, a good response to treatment, the absence of sequelae and the absence of any identifiable risk factors for decompression illness. Residual deficits should exclude the diver from future diving and indications that there is persistent tissue damage despite the recovery of function (incomplete initial response to treatment and a gradual resolution over months) are relative contraindications to further diving as they represent an increased risk of further damage, due to a decreased neuronal pool, if a decompression injury recurs.[15,28]

Divers with a history of pulmonary barotrauma, with or without neurological sequelae and whether "deserved" or "undeserved", should be excluded from further diving. The precipitating cause is thought to be an abnormality of the diver's lungs and because of the demonstrated inability of available investigative techniques, such as radioisotope scanning,[29] pulmonary computerised tomography[30] and methacholine challenge testing,[31] to sensitively or specifically identify someone at risk from pulmonary barotrauma, these divers have to be assumed to be at considerable risk of recurrence. A recommended distinction between "deserved" (eg a rapid breath hold ascent) and "undeserved" (eg a controlled slow ascent) barotrauma with respect to diving fitness[32] is unacceptable because of the subtle undetectable pulmonary scarring that may occur after injury.[1]

Divers whose injury is thought to have been associated with a cardiac shunt should also be advised not to return to recreational diving. Although the significance of a patent foramen ovale (PFO) in decompression illness is established, the utility of bubble contrast echocardiography in assessing future fitness to dive is controversial.[27,33,34] For example, could it be justifiable to use a contrast medium that contains a suspension of bubbles to demonstrate a shunt when there is the possibility that these same bubbles will flow to a cerebral circulation that has already been damaged by the passage of earlier bubbles? In addition, a negative test for a PFO does not exclude a pulmonary or any other anatomical shunt. However, once established, a PFO may not exclude professional divers from continuing to dive because they may be able to avoid diving patterns likely to result in venous gas emboli, which is unlikely in recreational diving.

Divers who have suffered neurological symptoms despite a seemingly trivial exposure and any recreational diver who has returned to diving and suffers another decompression injury without any apparent cause should be advised to cease diving because they may be inherently at risk of decompression illness due to risk factors that have yet to be defined.

Non-decompression induced neurological problems

Traumatic brain injury will affect fitness for diving and consequently considerable information about the nature of the injury is necessary. Details of localising signs, the period of unconsciousness, the duration of post traumatic amnesia, the presence of intracranial bleeding or of a penetrating injury or skull fracture and the likelihood of future seizures must be known. A study of closed head trauma in 1,000 patients reported the lowest incidence of traumatic epilepsy were in an uncomplicated head injury with post traumatic amnesia of less than 1 hour.[35] A history of a loss of consciousness of greater than 10 minutes, the presence of localising signs, an amnesic period of greater than one hour, the presence of intracranial bleeding, any skull fracture other than an uncomplicated linear one and of a penetrating injury would disqualify from future diving because of the risk of post traumatic epilepsy. However, a five year seizure free period without medication and after a meticulous history and examination may allow a diver to return to recreational diving if one uses idiopathic epilepsy as a guide,[36] although some may consider this too conservative because 80% of post traumatic epilepsy will present within 2 years of injury.[37] My view is that any risk of an underwater convulsion is a contraindication to further recreational diving.

There are different opinions about the fitness of divers who suffer migraine headaches. Hickey describes these as an absolute contraindication,[38] while Greer has an opposing view.[36] Diving may precipitate a migraine headache by hypercarbia, hyperoxia, hypothermia, cold water exposure, stress and arterial bubbles. The development of a migraine headache may impair a diver's safety underwater with the development of vertigo, visual disturbances, nausea and vomiting or produce a post dive diagnostic dilemma. Obviously each diver needs to be assessed individually with attention focused on prodromal symptoms and syncope or any loss of consciousness.

A peripheral neuropathy may interfere with the diver's dexterity and can be confused with symptoms of decompression illness. These divers should not continue to dive.

The development of a degenerative or demyelinating disease of the nervous system, spinal cord trauma or intracranial bleeding, with the risk of epilepsy, should disqualify the diver from future diving because any subsequent development of decompression illness may worsen the diver's neurological status.

Remedial spinal surgery (e.g. for an intervertebral disc prolapse with neurological symptoms and signs) should not exclude a diver from continuing to dive; although there is at least the theoretical risk of interference with the para-vertebral venous plexus drainage causing venous stasis predisposing to spinal cord decompression illness. More data are needed.

Any medication that can have central nervous system side effects should disqualify a diver during the course of the prescription. Anti-motion sickness (in particular the belladonna alkaloids) and one anti-malarial medication (mefloquine) are in this category. The belladonna alkaloids are commonly used to prevent motion sickness, particularly preparations containing hyoscine, and when taken as prescribed should not cause any incapacitation.[39] However, side effects such as drowsiness and blurred vision may interfere with the safe conduct of a dive. In addition, when taken to excess, which has occurred on live-aboard dive boats, these drugs can result in an acute brain syndrome.[40] This may be difficult to differentiate from acute decompression illness and/or have fatal consequences underwater. Any physician prescribing an anti-motion sickness medication to a diver should stress the importance of taking it only as directed and of beginning therapy 12 hours before going to sea (both to establish motion sickness prophylaxis and to allow time for any adverse effect to manifest before diving).[41]

The anti-malarial mefloquine (Lariam) has adverse effects even when taken as prescribed (spatial disorientation, vertigo, myalgia, joint pain, syncope, confusion, psychotic manifestations, headache and convulsions) and must never be prescribed for malarial prophylaxis to anyone who wishes to dive.[42] Anyone who is taking mefloquine and wanting to dive should stop taking it four weeks before diving. They should not start on chloroquine as the combination can cause asystole. The risk of convulsions from Lariam is 3%. I think a 3% risk of convulsing underwater is not acceptable. A lot people get symptoms of nausea and vomiting and vertigo, neither of which increase underwater safety.

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WOMEN IN DIVING

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Abstract

The issue of female fitness to dive is really no different from male fitness and both genders should be assessed on an individual basis. The physiological features exclusive to women which have an influence on diving fitness are few. A literature review covering these topics is presented.

Introduction

Female divers are no longer considered a rarity and the image of scuba diving being a male dominated sport is long past. Previously much has been written on the topic of female divers, as if the very presence of oestrogen imparts an ominous prognosis. However, the issue of female fitness to dive is really no different from male fitness and both genders should be assessed on an individual basis. The physiological features exclusive to women which have an influence on diving fitness are few and a literature review covering these topics is presented.

Performance

Females typically have a lower threshold for peak sports performance than males and generally can produce less power, speed and have lower work capacity and stamina. As a consequence a female can not generally achieve a maximal oxygen consumption per kilogram equivalent to a male.[1]

Females possess a higher percentage of body fat which persists despite training. A 20 year old sedentary female has approximately 25% fat, a trained female 10-15% fat and a trained male 7-10% fat. Trained males have relatively more muscle(40% of total body mass) while comparatively fit women have only 23% muscle.[1]

Thermal Stress

Females are able to conserve energy more efficiently than males. Their increased body fat provides better insulation from heat loss as well as increased buoyancy. Females have fewer sweat glands and sweating begins at a higher core temperature, so conserving heat. However, this increases a female's susceptibility to over heating when sitting, fully kitted up, in the sun.

Females have a lower basal metabolic rate (BMR). Overall, women are more susceptible to heat loss than men