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LONG TERM HEALTH EFFECTS OF DIVING

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Key Words

Diving safety, investigations, medical conditions and problems, treatment sequelae.

Introduction

With the improvement in prevention and treatment of diving accidents over the last few decades, attention has now been focussed on the possible undesirable long-term health effects of diving. Much of the investigative efforts in this field have centred on the professional diver, however, for large numbers of recreational divers these concerns are very real. If we, as medical practitioners, are going to assess our patients' fitness to dive, we must also be able to provide advice as to how diving may affect their health.

Long term effects

A long term effect of diving can be defined as an effect outside the range of normal for an appropriately matched population. This effect must be causally related to diving and must persist beyond the acute and rehabilitation phase of a diving accident. There must be no non-diving pathology to explain the effect and it must produce a reduction in the performance or the quality of life of the diver.

There is no dispute that diving accidents can result in permanent sequelae such as dysbaric osteonecrosis, decompression illness (DCI) and pulmonary barotrauma with cerebral arterial gas embolism. A diver who suffers hypoxia from any cause with resultant brain injury may have permanent damage. Other diving related injuries, such as barotraumas, can result in hearing loss and vestibular damage. Gas toxicities and marine animal injuries may also result in permanent sequelae.

A more difficult question to address is, do long-term health effects occur in divers who have not suffered an overt injury or a specific diving accident?

A variety of physiological and pathological changes have been postulated to produce a great variety of long-term health effects, not related to a specific diving accident. This non-exclusive list includes increased environmental pressure, increased gas partial pressure, oxygen toxicity, gas induced osmosis, asymptomatic bubble development with local tissue effects, blood bubble interaction and blood brain barrier disruption, barotrauma damage to surrounding tissues, asymptomatic lipid emboli and adaptive effects of diving. I will specifically discuss dysbaric osteonecrosis (DON), barotrauma, decompression illness (DCI), ^{99}Tc-HMPAO-SPECT scanning (which is an investigation that has been in and out of favour), ophthalmological effects, ear nose and throat problems, pulmonary effects, subclincal pathological deficits (which from their very nature are hard to detect and quantify), neuropsychology, behavioural factors, and finally mention some miscellaneous findings.

Dysbaric osteonecrosis

Dysbaric osteonecrosis is usually assumed to be the direct or indirect result of gas bubbles that form during decompression interfering with the blood supply to vulnerable areas of bone. Juxta-articular, or A, lesions are found near articular joint surfaces and may eventually result in the collapse of the joint. Medullary, or B, lesions are found away from the articular surface, are usually asymptomatic but occasionally result in the development of sarcomata.

DON is often thought of as a consequence of deep diving, although it was first observed in caisson and tunnel workers. It became a problem in the commercial diving world as exploration went deeper.

DON is rare in recreational and military divers who have not been involved in experimental diving. It has been generally assumed that there was no risk of DON in divers who did not descend below 30 m. However, tunnel workers, who work long shifts under pressure, have had a significant incidence of DON, although they were typically working at pressures less than 12 m.

DON has been known to occur after a single pressure exposure.[1] In 1931 a Royal Navy submarine became disabled in the China Sea and three out of the five survivors who escaped subsequently developed DON. They were exposed to a maximum pressure of 4.8 bar (ATA) for 2-3 hours during their escape, their only exposure to increased pressure. This refutes the perception that DON only occurs in people who have had extensive diving careers. A more realistic view is that DON is the consequence of imperfect decompression procedures.

It is known that conservative diving practices offer some protection. By the mid 1980s the British Decompression Sickness Registry, which was responsible for X-raying occupational divers, was closed because the number of new cases were so few, largely due to government regulation of decompression table usage in the North Sea.

While it is presumed that DON is a long term consequence of inadequate decompression and bubble formation, the exact mechanism by which those bubbles cause injury is unknown. Proposed aexplanations include direct bubble injury to bone, infarction secondary to arterial gas embolism, vascular obstruction secondary to platelet aggregation and activation of inflammatory pathways, raised intra-osseous pressure due to bubble formation within bone and raised intra-osseous pressure as a result of gas osmosis during decompression.[2]

There does appear to be an association between DCI and DON although not all divers with DON have a history of DCI. Neither do all divers who suffer DCI develop DON, nor do all divers with a history of high risk exposures develop DON.

Barotrauma

Barotrauma may produce, albeit infrequently, permanent damage to the facial, infra-orbital, maxillary, cochlear and vestibular nerves.

Decompression illness

There is no doubt that DCI causes residual health sequelae. Spinal DCI may result in paraplegia with urinary and bowel disturbance despite recompression therapy. DCI can result in permanent sensory disturbance with paraesthesia or disturbance along the distribution of peripheral nerves. DCI produces psychometric abnormalities with some studies reporting up to 50% of individuals with residual neuropyschological abnormality after experiencing an episode of DCI.[3]

It has also been suggested that when one is diving sub-clinical and cumulative lesions may develop within the brain and spinal cord. Todnem et al. compared 156 divers with 100 non-diving controls and reported that divers had a higher incidence of specific neurological symptoms and episodes of cerebral dysfunction, in non-diving situations.[4] The latter predominantly involved the distal spinal cord and peripheral nerves. There was an independent correlation between these abnormalities and diving exposure, DCI and age.

Whether or not subclinical and cumulative lesions can occur in divers who have never experienced clinical DCI or develop after complete recovery is uncertain. If they do occur, can they be avoided by conservative diving practices and how important are they clinically?

Central neurological damage may be transient, or may be permanent and cumulative. Electro-encephalographic (EEG) changes after diving accidents or incidents may indicate damage, but the alteration in EEG behaviour is sometimes difficult to interpret. CT and MRI scans, as well as evoked cortical potentials may reveal only gross abnormalities and may miss small areas of multi-focal damage. Newer radiological and scanning techniques have been used in the investigation of divers, yet many of these techniques, in their own right, have yet to be adequately standardised.

^{99}Tc-HMPAO-SPECT scanning

Technetium-99 hexamethylpropyleneamine oxime single photon emission computerised tomography (^{99}Tc-HMPAO-SPECT) is a technique used to image regional blood flow. The ^{99}Tc-HMPOA is injected intravenously and diffuses across the blood-brain barrier and remains bound in the tissues for up to 8 hours so therefore it effectively produces a frozen image of the regional brain blood flow at the time of injection.

In 1989 Adkisson et al. first reported the use of this technique in people who had suffered an episode of DCS.[5] They studied 28 patients within one month of presentation. Twenty-three of those were said to have neurological DCS, 4 had cerebral arterial gas embolism (CAGE) and 1 had a limb bend. They reported cerebral perfusion deficits in all cases of neurological DCS and CAGE and a high degree of correlation between the clinical picture and the site of the perfusion deficit. The possibility of occult neurological damage was raised by the appearance of cerebral perfusion deficits in divers who showed clinical signs only of spinal cord involvement. The patient with the limb bend had a normal scan.

However, subsequent investigators have challenged some of these findings. Hodgson et al. compared 10 divers with acute DCS to 10 divers who had been treated some 3-5 years earlier for DCS, 10 divers who had never had DCS and 10 population controls.[6] Although there was a trend towards a larger number of deficits in the individuals who

had had DCS, there was no statistical difference between the groups and there was no apparent correlation between the sites of the perfusion deficits and the clinical presentation. There was also a higher than predicted number of positive scans in both the divers who had never been treated for decompression sickness and the non-diver controls. This study does not therefore support the assumption that asymptomatic divers sustain neurological injury, as the same deficits were seen in individuals who had never dived. The significance of these deficits and the incidence in control populations is yet to be determined.

Following this, Evans et al. studied 54 patients who presented to the Aberdeen Hyperbaric unit with DCS.[7] Of these 62% were said to have abnormal ^{99}Tc-HMPOA-SPECT scans but there was no correlation between the site of the deficit and the clinical presentation. They then extended the study to include divers who had no history of DCS and non-divers.[8] Again they concluded that although the divers with abnormal scans in the group that had DCS tended towards a greater diving exposure, this was not repeated in the diver group. Therefore they could not conclude that a greater diving exposure increases the likelihood of an abnormal ^{99}Tc-HMPOA-SPECT scan.

Ophthalmological effects

Because the eye develops as an extension of the forebrain it has long been recognised that the fundus may reflect changes within the central nervous system (CNS). Polkinghorne et al. studied 84 divers and 23 non-divers using retinal fluorescein angiography to determine whether blood vessel changes are common in the ocular fundi of divers.[9] It was proposed that these changes might indicate the presence of vascular obstruction elsewhere, particularly in the CNS. Twelve of the divers had previously been diagnosed with DCS (9 neurological) although none had visual symptoms. Three presented with joint pain only. The authors reported that the retinal capillary density at the fovea was low in divers and microaneurysms and small areas of capillary non-perfusion were evident. The divers had significantly more abnormalities of the retinal pigment epithelium than the comparison group of non-divers. They also found a positive correlation between the presence of a fundus abnormality and the length of diving history. It was concluded that all changes were consistent with obstruction of the retinal and choroidal circulation and the obstruction was likely to be due to intravascular bubble formation during decompression or to the altered behaviour of blood constituents and blood vessels under hyperbaric conditions. However, no subject had any recorded visual loss as a consequence of diving and other investigators have not been able to reproduce these findings.

Holden et al. performed angiography on 26 divers, who had used safe diving practices for at least 10 years and 7 controls.[10] No significant difference in the incidence of macular abnormalities was found between these groups. It was suggested that adherence to safe diving practices may protect against the effects reported by Polkinghorne.

Murrison et al. studied a cohort of Royal Navy divers with retinal fluoroscopy and angiograms and no difference was found between the divers and the non-divers and the prevalence of abnormalities did not correlate with diving experience.[11]

Ear nose and throat problems

There is no doubt that hearing loss can occur as a result of diving, whether it is noise induced from exposure to noise in compression chambers or as a result of inner ear barotrauma. Exostoses, which are a long-term effect of cold water exposure, are common in divers, but rarely become symptomatic.

Sinus disease is common in divers. It is often a cumulative result of inappropriate diving activities, especially when divers persevere with descent despite the evidence of barotrauma and infections. Chronic inflammation and gradual scarring results in reduction of the lumen of the sinus ostia or ducts.

Pulmonary effects

It has been proposed that long-term effects damage the lung as a consequence of diving. Some divers tolerate high levels of carbon dioxide without increasing their respiratory rate.[12] It is uncertain as to whether this reflects an inborn physiological abnormality or is an adaptation from diving.

It is generally accepted that divers have larger vital capacities than non-divers, with one study suggesting an initial adaptive increase in lung volumes in divers, followed by a progressive decline, presumably age related.[13] It has also been demonstrated that some divers develop a degree of airflow obstruction due to airway narrowing.

It has been suggested that some of these effects may be related to a cumulative pulmonary oxygen toxicity or to repetitive pulmonary gaseous micro-embolisation.[14]

It has also been shown that there is a slight decrease in pulmonary transfer capacity after deep dives which tends to improve in the subsequent weeks, but which may not be totally reversible.[18]

Proposed mechanisms for these changes include lung over-distension, hyperoxia, bubbles, pressure per se and gas and particulate contaminants.[13]

Subclincal pathological deficits

Palmer et al. reported the autopsy of a male sports diver who had recovered almost completely from an episode of spinal DCS 4 years before his death from an unexpected cause.[15] He was clinically asymptomatic, yet at autopsy was found to have extensive changes in the posterior and lateral columns of his spinal cord.

Palmer, Calder and Hughes examined the spinal cords from 8 professional divers and 3 amateur divers who died accidentally.[16] All but one had had a diving medical examination somewhere between 2 and 38 weeks before their death. None had a history of DCS and none had any documented neurological abnormality at the time of their medical examination. Marchi positive tract degeneration was found in the cords of 3 professional divers, variously affecting the posterior, lateral and to a lesser extent the anterior columns. These changes were difficult to see on haematoxylin and eosin sections, but were clearly shown by the Marchi staining technique. Marchi positive material does not appear in degenerating myelin fibres until 7-10 days after the initiating lesion and does not appear intracellularly until some 10 weeks after the insult.

Palmer, Calder and Yates also studied the brains from 12 amateur and 13 professional divers who died accidentally.[17] Only 3 of the 25 had reported a previous episode of DCS. In 15 of the 22 divers who died from diving accidents there were grossly distended, empty vessels in brain tissue presumably due to post mortem decompression artifact. The most striking change observed was perivascular lacuna formation found in the cerebellar white matter of 8 brains (3 amateur, 5 professional). In addition hyalinisation of vessel walls was observed, along with necrotic foci in the grey matter and perivascular vacuolation of white matter in 7 cases.

The authors propose these changes probably arose from intravascular gas bubble formation producing a sudden distension and occlusion of small arterial vessels. With passage of the bubble the vessel returns to its normal size leaving a surrounding area of degenerated tissue within the lacunae.

Hyalinisation of the vessel wall is also believed to occur as a consequence of this rise in luminal pressure. In one previously asymptomatic professional diver, there was also unilateral necrosis of the head of the caudate nucleus.

This study provides evidence of chronic changes (lacuna formation and hyalinisation of vessel walls) in the brains of asymptomatic divers who did not have a history of DCS when compared with control subjects. This is important as it is now generally accepted that bubbles are produced with all but the most innocuous dive profiles.

Neuropsychology

There have been many anecdotal reports in the literature supporting the hypothesis that diving per se produces brain damage and dementia. Reputable studies to support this hypothesis are lacking. Carl Edmonds studied the Australian abalone divers.[18] These divers had dived on average for greater than 16 years, for greater than 5 hours per day, 105 days per year, to depths greater than 50 feet and had suffered DCS on average 4 times. There was no evidence to support a conclusion of chronic mental impairment or diving dementia in this population.

The US Navy studied 421 divers over a 10-year period and found no evidence, in the absence of a specific injury, to support a correlation between altered neuropsychometric tests or function with diving exposure.[19]

Behavioural factors

In discussing behavioural changes as a consequence of diving it is most important to establish what divers are really like. It may come as a surprise to some but most of the literature suggests that divers differ from the normal population![18] Divers tend to be more adventurous; they take risks and are more physically active. They perform quite differently on psychological testing. They appear to have a love of adventure and have reduced levels of anxiety. Divers have a preponderance of traumatic causes of death. They have higher suicide rates and more motor vehicle accidents.

Therefore it is important to consider whether diving causes them to act that way or are they simply a subgroup of risk-taking, adventure loving people. The verdict remains open on this question.

Miscellaneous

It has been claimed that divers are sub-fertile or have a preponderance of female offspring. There is no evidence to support either of these statements.

There are many haematological and vascular changes that have been reported in animal and experimental models but few persist for any length of time post-ascent or in the human population. An increased incidence of chromosomal aberrations in cultured T lymphocytes in divers has been demonstrated but the health effects related to these changes have not been fully evaluated.[20]

It has been postulated that arthritic disorders have a higher incidence in divers than non-divers and a study of over 11,000 US navy divers showed an increased hospitalisation rate for joint disorders in the 23-28 age group without any obvious explanation.[21]

Conclusions

In 1993 an international consensus conference on the long term health effects of diving summarised current knowledge and produced the following statement:[22]

"There is evidence that changes in bone, the CNS and the lung can be demonstrated in some divers who have not experienced a diving accident or other established environmental hazard.

The changes are in most cases minor and do not influence the diver's quality of life. However, the changes are of a nature that may influence the diver's future health.

The scientific evidence is limited, and future research is required to obtain adequate answers to the questions of long term health effects of diving."

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ARTICLES OF INTEREST REPRINTED FROM OTHER JOURNALS

TASMANIA'S AQUACULTURE INDUSTRY: A TEN-YEAR REVIEW OF IMPROVED DIVING SAFETY

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Key Words

Decompression illness, diving industry, diving operations, occupational diving, risk, safety.

Abstract

Tasmania's marine finfish aquaculture industry has developed from humble beginnings in 1986 to become a leading export earner for the state. Marine aquaculture is diving intensive, and divers have made a significant contribution to product quality. The early years of the industry were hampered by significant levels of diver morbidity due to risky diving activities. This ten-year review outlines the major improvements in safety which have been achieved by broad-based changes to diving training, operations and procedures. The number of divers treated annually for decompression illness has fallen from 5.5 per 2,100 dives in 1988-90 to 0.5 per 8,768 dives in 1996-98. The industry now has a decompression illness incidence of 0.57 cases per 10,000 dives and is in line with world's best practice.

Introduction

Tasmania possesses a rich maritime heritage. Since European settlement, the Tasmanian economy has been reliant on its close links with the sea for trade. The unpolluted waters off its coastline support a substantial wild fishing industry. Tasmanians have always enjoyed access to quality seafood. Until the 1970s, wild fisheries were the only significant source of revenue from fishing in the State. A natural progression of the Tasmanian's close relationship with the sea has been the development of marine aquaculture. After initial success with oyster and mussel farming in the 1970s, Atlantic salmon farming commenced in 1986. More recently, marine farming ventures have explored scallop, abalone and striped trumpeter aquaculture. The aquaculture industry is a major contributor to Tasmania's economy, now producing 35% of total fisheries value of $213.9 million.[1] From small origins, the marine finfish aquaculture industry grew to employ over 500 people directly in 1997, producing exports worth $ 64 million.[1,2]

Figure 1. Diver entering a typical aquaculture pen.

Atlantic salmon farming and ocean trout farming are the main sectors of the aquaculture industry which employ divers. Divers contribute substantially to the quality of these fish, which obtain premium prices on world markets. The fish are farmed in floating pens up to 120 m in circumference, which enclose the fish in a cylindrical net suspended from the surface (Figure 1). Pens vary in size, depending on the type of fish farmed and the size of the farm's operations. An aerial photo of a typical lease is shown in Figure 2. Divers in the marine aquaculture industry undertake many roles including maintenance of mooring lines and farm perimeter nets, supervising the setting of fish pens, and undertaking checking and repairs of individual fish pens with removal of dead fish from the pens. In addition, they perform many "land-based" activities. In 1990, two of the authors provided an overview of the industry and described how salmon were farmed from smolt to the finished product bound for interstate and international markets.[3] The industry is further described elsewhere.[2]