ORIGINAL PAPERS

NEUROLOGICAL INVESTIGATIVE TECHNIQUES IN DECOMPRESSION ILLNESS

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Abstract

This paper reviews the neurological assessment of the diver after completion of treatment for decompression illness (DCI). An impaired neurological status is the factor most likely to prevent a return to diving. Investigative procedures available to assist in delineating the neurological injury include magnetic resonance imaging (MRI), computerised tomography (CT), hexamethylpropyl amine oxime (HMPAO) scans, electroencephalography (EEG), neurophysiological and psychometric testing These techniques rarely provide more information than a detailed clinical examination for the individual patient. Collectively, their application has provided valuable insight into the pathogenesis of DCI.

Pathogenesis of decompression illness

Our understanding of the pathological events in DCI is incomplete. As a reduction in ambient pressure forces the tissues to reduce their dissolved gas load, inert gas bubbles are formed in intra- and extra-vascular spaces. Vascular bubbles arise predominantly from the venous side, although de-novo arterial bubble generation is possible. Venous gas emboli, which are produced in many asymptomatic dives, are believed to be filtered out by the lungs up to some unknown critical point. Pulmonary overload or shunting will allow bubbles to enter the arterial system. They then travel by flow and buoyancy to the cerebral circulation in the upright person. Inert gas bubbles form columns which lodge at arteriolar bifurcations producing occlusion, distal ischaemia and neurological manifestations. The majority of these columns clear within 10 minutes, due to a rise in mean arterial blood pressure, but their transit produces endothelial damage with increased permeability of the blood brain barrier.[1] Leucocytes migrate through the endothelium as a result of chemotactic factors and platelet activation occurs.[2,3,4] Activation of complement and coagulation pathways occurs in severe cases and can evolve to disseminated intravascular coagulation.[5] Prostaglandins, histamine and serotonin are released in a manner indistinguishable from the acute inflammatory reaction.[6]

Extravascular, or tissue, bubbles will exert local effects dependent on the volume of the bubble and the type of the tissue affected. Adipose tissue can tolerate large volumes of gas without symptoms. Small bubbles within tightly bound connective tissue will produce pain. The myelin sheath, a prime site for bubbles because of its high fat content, will require only a small critical volume before there is interference with conduction of nerve impulses. In most cases of spinal DCI the production of such autochthonous bubbles would appear a more likely causal mechanism than venous stasis.[7]

These complex pathogenetic mechanisms act to a variable extent, in combination, at the different levels of neurological function (cerebral, spinal, peripheral). The majority of DCI cases suffer a multifocal, central nervous system insult.

Neurological assessment

Assessment of the extent of neurological damage that has been produced by DCI is important if sensible advice is to be given to the diver after completion of treatment. The diver will be interested not only in the likely prognosis, but also in whether a return to diving can be recommended. Causative factors will need to be considered, as will the nature and severity of the insult and the response to treatment. However, the neurological status is the factor most likely to prevent a return to diving.

In those divers where response to treatment is incomplete and who have residual clinical abnormalities, the aim is to determine the site and extent of neurological damage. This information will provide the basis for prognostic advice on potential recovery and outcome. The biggest gains in neurological recovery are seen in the first two to three months, with smaller improvements continuing for up to two years.[8] In some patients gradual deterioration has occurred after a static period of some years. This may be due to natural senescence superimposed on a diminished neurological reserve. If there are residual neurological signs a return to diving is unlikely to be recommended. Any further episode could erode a reduced functional reserve and be devastating.

Where there has been an apparent full clinical recovery, the objective is to elicit signs of subclinical damage. Determination of fitness to return to diving will depend on the assessment of susceptibility to DCI. This is variable both between individuals and for the same individual on different occasions. If DCI was produced without obvious precipitating factors and with minimal decompression obligation, then increased susceptibility must be assumed. Any advice concerning a return to diving would have to be cautious.

Incidents which result in a severe or neurological presentation probably produce subclinical damage, regard-

less of favourable treatment outcome and freedom from clinical signs. A post-mortem study showed spinal cord lesions in a case of recovered DCI.[9] Subtle neurological changes have been found in cases diagnosed as musculoskeletal DCI.[10]

Mounting evidence of neurological damage in DCI has provoked uncertainty about the safety of diving in general. There are concerns that subclinical damage may reduce functional neurological reserve in the asymptomatic and incident free diving population. Retinal fluorescein angiography has been used to examine the eyes of 84 divers, 12 of whom had a past history of DCI.[11] The investigators found low retinal capillary densities at the fovea, microaneurysms, small areas of capillary non-perfusion and increased abnormalities of the retinal pigment epithelium. The extent of these abnormalities correlated with the length of the diving history. However, no subject had any demonstrable visual loss. But since the retina is generally felt to be "the window into the CNS", the authors suggested that their findings may imply that asymptomatic CNS injury occurs as a result of diving. Post-mortem studies of the CNS of divers have demonstrated abnormalities in some who have no recorded history of DCI.[12,13]

As doctors and divers become more aware of the potential for neurological damage, there is an increasing trend to investigate. The delineation of the neurological injury will depend on the limitations of the available investigative techniques. This review evaluates the role of magnetic resonance imaging (MRI), computerised tomography (CT), hexamethylpropyl amine oxime (HMPAO) scans, electroencephalography (EEG), neurophysiological and pyschometric testing in the assessment of the diver.

Magnetic resonance imaging

MRI provides detailed resolution of the brain and spinal cord. A study of 14 patients with barotrauma demonstrated brain MRI abnormalities in three out of the four patients with cerebral presentations.[14] Two of the abnormalities detected corresponded to the neurological deficits on clinical examination. There were 12 patients with spinal cord presentations, but only three had abnormal spinal cord MRI scans.

The Duke University Medical Center experience is similar, with abnormal brain MRI in 56% of cerebral DCI cases and abnormal spinal MRI in 17% of cases with clinically suspected spinal cord damage.[15] Abnormalities of T_2-weighted images were found, compatible with regional oedema.

Two divers with severe neurological DCI demonstrated the difficulty in isolating the level of injury.[16] Both were reported to have a Brown-Sequard pattern of deficit at the thoracic level with bilateral lower extremity weakness. Thoracic cord MR images were normal in both cases, with the one case where a cervical MRI was performed, also normal. This suggests that either the cord was spared, the level missed, or that the resolution of the MRI was insufficient to demonstrate isolated tract or partial column damage. There was certainly no evidence of a hemisection of the cord. Diffuse cerebral pathology was suggested in both cases by abnormal brainstem and somato sensory evoked potentials. One case showed slowing of the EEG waking background rhythm. The brain MRI showed multiple foci of high signal intensity in the peri-ventricular white matter of the parietal region in both cases. One patient also had right lentiform nucleus and internal capsule foci.

A MRI study of 105 divers and 49 controls showed no significant difference between the two groups, despite the fact that 51% of the divers had a history of DCI.[17]

CT scan

CT scanning techniques are less sensitive than MRI for detecting foci of cerebral ischaemia and in the spinal cord are unable to provide adequate definition of the soft tissues. In a study of 47 CT scans performed within one month of DCI, the concordance between the initial CT report and a blinded independent radiologist was 87%.[18] Only one scan had abnormalities reported by both radiologists; small low density areas. A retrospective review of the case notes disclosed 24 cases with symptoms suggestive of cerebral involvement. No CT abnormalities could be correlated with the clinical presentation. Although small low density areas have been separately demonstrated in two serious cases of dysbaric illness,[19] the CT scan is not a cost effective tool for the post-treatment evaluation of DCI. The majority of neurological DCI cases are not sufficiently severe to produce areas of cerebral infarction.

Electroencephalography

As part of a follow up study of 72 post DCI patients, EEGs were performed after completion of treatment, and one month and one year later.[10] EEGs were reported as normal, doubtful or abnormal. Slow wave abnormalities were detected with increased theta wave activity. Focal abnormalities were not found. A definite trend of improvement in the 48 (67%) who returned at 1 month was found with eight of the 11 reported as abnormal improving. At one year, five of the 23 who returned had shown improvement. Problems with the study, included epidemiological concerns about the high drop out rate and lack of independent and blinded EEG reporting. Recommendations for a further study included objective, more frequent analysis of the EEG and quantification of subjective assessment of slow wave activity in the treatment and early recovery phase following DCI.

Psychometric testing

Psychometric evaluations were conducted as part of a neurological sequelae study.[10] The Australian Council of Education and Research Word Learning Test provided expected performance parameters for the Benton Visual Retention Test. This is a test of visuo-spatial perception and short term memory. Of 56 patients tested in the month following treatment for DCI, 16 showed significant impairment. Only four of the 16 returned at one year, an unacceptably high drop out rate, when two were normal and two abnormal. A further analysis found the percentage of EEG abnormalities was the same in both normal and abnormal psychometric groups. This study's preliminary findings[20] are much quoted, yet are inconclusive.

Neuropsychological tests have mostly been used in saturation diving for the assessment of the effects of extreme hydrostatic pressure and the high pressure neurological syndrome.[21] They have been used to monitor recovery following DCI and have also been applied to diving operations where concerns about neuropsychological safety are held.[22] Their sensitivity is far greater when repeated assessments of the individual are made. However pre-morbid results have rarely been available in the clinical setting. More frequent assessment after the incident would also help to delineate the role of this form of testing.

Despite the concerns of the pathologists[12], no convincing evidence of long term psychometric deficits has been demonstrated in abalone divers who are known to dive well beyond standard tables and who have a high incidence of DCI [23]

HMPAO scan

This nuclear medicine technique demonstrates cerebral blood flow. Hexamethyl propylene amine oxime (HMPAO) labelled with $^{99}T_c^m$ is injected and measured with single photon emission computerised tomography (SPECT). $^{99}T_c^m$ HMPAO crosses the intact blood brain barrier with 3-8% becoming fixed to neuronal nuclei in the grey matter in proportion to blood flow at the time of injection. The technique gives an instantaneous picture of cerebral perfusion. Scans were performed on 28 cases of neurological DCI.[24] Cerebral perfusion deficits were reported in all 23 cases of neurological DCI and in all four cases of cerebral arterial gas embolism (CAGE). No deficits were present in the single case of musculoskeletal DCI. As cases of presumed spinal cord disease showed these deficits, it was concluded that neurological DCI is a diffuse multifocal CNS disease. A small follow up series of 18 patients who were rescanned at varying intervals found that the majority had persistent deficits.[25] Criticisms of both studies included the absence of a control population and incompletely blinded interpretation of the scans.

A case control study was performed to investigate further the use of the HMPAO scan.[26] Four groups of 10 subjects were studied:

a Divers scanned days after treatment for neurological DCI
b Divers scanned years following neurological DCI
c Diver controls
d Population controls

All groups were matched for age and the divers were further matched for general diving experience. The scans were randomised and reported blind to the history. Despite a trend towards larger numbers of deficits in individuals who had suffered DCI, the four groups were statistically indistinguishable. Furthermore no correlation was found between the location of the perfusion deficits and the clinical presentation. This particular technique requires further evaluation before significance can be ascribed to perfusion deficits found in divers.

Somatosensory evoked potentials

Somatosensory evoked potentials (SEPs) were measured on a sample of 30 divers who had received recompression treatment for DCI at the Royal Australian Navy School of Underwater Medicine.[27] The median nerve at the wrist and the posterior tibial nerve at the ankle were stimulated and SEPs recorded. Median nerve studies were normal, but nine of the 30 subjects had abnormal tibial SEPs, with an increase in the interpeak latency for N20-P37. This suggested pathology affecting the postero-lateral and posterior columns between the lumbar and low cervical regions. Two divers had bilateral and seven had unilateral abnormalities. Six had normal neurological examinations. In two of the three with abnormal signs there was a correlation between the SEP abnormalities and signs of weakness or sensory loss. Despite the inconclusive numbers it was suggested that SEPs were more sensitive than normal methods of neurological examination for detecting neurological sequelae of DCI.

A more recent study performed SEPs on 23 divers with varying degrees of residual disability from spinal cord DCI.[28] Only the four wheelchair bound patients who had absent SEPs were statistically significantly different from normal. The nine with obvious neurological deficits had SEPs within the normal range. The authors conclusion was that careful neurological examination is a more sensitive measure of residual deficit and SEP studies may only be useful in very severe cases.

Conclusion

Neurological investigative techniques have been important in the development of our current understanding

of DCI. CT and MRI scans have demonstrated lesions in cases with a severe cerebral insult, but not in the majority of cases. Psychometric testing, EEGs and HMPAO scans have all indicated potential sensitivity but require further development and evaluation to determine their roles. For the individual patient, none of these techniques is as sensitive as a careful neurological examination. Sensible advice can be given based on clinical findings and analysis of the initiating factors without the need for detailed investigation. However, in major centres where a large number of patients are being reviewed, it is important for new and refined techniques to be applied as they become available.

There is exciting potential for further research. Positron emission tomography (PET) will probably become the "gold standard" of functional imaging. The resolution of PET is better than that of SPECT because it detects the pair of gamma photons emitted simultaneously from the positron-electron interaction. Metabolic activity can be monitored by labelling biological substrates. Activation studies can demonstrate real time metabolic reactions as the nervous system is stimulated. Evolution and refinement of the technique is continuing, but limited by the expense of the cyclotron necessary to produce the short half-time isotopes.

Computerised EEG mapping techniques should overcome the difficulties encountered with subjective interpretation of EEGs. It also provides a far more comprehensive and reproducible assessment. Evoked potential physiological studies are becoming more sophisticated and sensitive. It is only through this continuing research for clearer elucidation of the neurological insult that we will further our understanding of the pathogenetic mechanisms in DCI.

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NEUROPSYCHOLOGICAL PROBLEMS IN 25 RECREATIONAL DIVERS ONE YEAR AFTER TREATMENT FOR DECOMPRESSION ILLNESS

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Abstract

Twenty-five recreational divers were treated for decompression illness at the Royal New Zealand Naval Hospital in 1987 using the the United States Navy treatment algorithm. Twenty-three of these divers were reviewed one year later. At discharge from hospital, 11 (48%) had obvious neuropsychological sequelae. None of these had recovered fully by one year. In contrast, 6 of the 12 who had no problems at the time of their discharge either developed or were noted to have problems during the next year. These late sequelae were mostly in the form of personality changes. The overall morbidity rate at one year was 74%. Alternatives to the United States Navy treatment alogrithm should be developed and tested, and a review as late as one year after DCI may be needed to assess outcome accurately.

Introduction

The treatment of Australasian recreational divers with decompression illness (DCI) using the United States Navy (USN) treatment algorithm[1] is associated with treatment failure rates at discharge from hospital and one month later of between 32 and 45%.[2-5] However, it is believed that most sequelae of decompression illness will resolve during the subsequent year.[3-6] To test this belief, a defined population of recreational divers who were treated for DCI were surveyed one year later to determine the progression and prevalence of neuropsychological sequelae.

This survey was based on questionnaires and clinical examinations, as careful neurological examination appears a more sensitive measure of outcome than available evocative, recording or imaging techniques of the nervous system.[7,8]

Methods

In 1987, a total of 25 recreational divers were treated at the Royal New Zealand Naval Hospital (RNZNH) for DCI that developed after one or more air dives. The USN treatment algorithm was universally employed. One year after their discharge from RNZNH, letters were sent to all these patients, requesting general information about their invalidity, time off work, compensation or insurance claims, and, if any, specific disabilities. Any replies suggesting problems were followed by further contact with the patient (including an examination, if possible), their spouse, family, family doctor and/or diving physician.

Two patients who claimed significant neuropsychological disability were further assessed by extensive psychometric review[9] at Auckland Public Hospital's post-concussion clinic.

Results

Twenty-three of the 25 patients (92%) responded to the questionnaire. The 2 patients who could not be contacted had no overt problems when they were discharged