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SINGLE PHOTON EMISSION COMPUTERIZED TOMOGRAPHY (SPECT)
New Insights into the Pathology of Cerebral Decompression Sickness

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Background and Development

The Undersea Medicine Division at the Institute of Naval Medicine, Alverstoke, England, in conjunction with the Royal Naval Hospital, Haslar, began a series of studies in late 1987 to evaluate the use of a newly developed method of measuring cerebral perfusion in the investigation of pressure related accidents.

First applied to cerebral arterial gas embolisms (CAGE), it was soon extended to divers suffering from various degrees of decompression sickness (DCS). Work is continuing but the initial studies have provided new insights into the pathology of cerebral DCS.

$^{99}TC^{m}$-HMPAO

Hexamethylpropyleneamine oxime(HMPAO), marketed under the brand name Ceretec by Amersham International Plc in England, was developed in an effort to allow for routine imaging of cerebral perfusion following a variety of cerebral ischaemic events. While agents have existed previously, they have been difficult to use or expensive. HMPAO proved to be a simple and reliable ligand for use in such studies. When combined with Tc-99m, an isotope used in 80 percent of all nuclear medicine procedures, HMPAO penetrates the blood-brain barrier, distributes in proportion to cerebral blood flow and becomes fixed in cerebral tissue with no significant redistribution over the imaging period. Due to its rapid distribution but prolonged half-life, imaging can be performed within minutes or be delayed several hours. Imaging gives a picture of cerebral perfusion pertaining at the time of injection rather than at the time of imaging. Single photon emission computerized tomography (SPECT) is performed using an orbiting gamma camera of the type found in most nuclear medicine departments. A three-dimensional sinogram is acquired that can be examined in axial, saggital or coronal planes. Repeat studies can be performed to monitor changes in perfusion patterns.[1]

Initial Studies

The outcome of a patient with CAGE or DCS is often dependent upon the rapidity with which treatment is instituted. Recompression therapy should not be delayed while secondary examinations and testing are conducted. HMPAO seemed to be an ideal agent for evaluating diving related accidents in both the acute and follow up settings.

A patient could be injected with HMPAO without delaying treatment and subsequently scanned in the controlled setting of a nuclear medicine department. The scan, however, would tell us what was occurring prior to the treatment. In the long run, this proved to be less valuable than first imagined but may be important for future studies.

The first three patients examined with HMPAO were found to have cerebral perfusion deficits that appeared to correlate with their clinical signs and symptoms.[2] Over the next year, 50 divers were studied following incidents of DCS, CAGE or where an unusual event occurred which left the diagnosis in question. 47 patients showed positive scans correlating with suspected injury. 3 patients with negative scans had suffered from Type I DCS or were diagnosed as being non-diving related injuries. 28 of these first 50 patients were reported on showing significant correlation with their diving injury and their cerebral findings.[3]

Specific criteria were established for evaluating this first series of divers. There had to be a definite diagnosis of DCS or CAGE treated by a recognized USN or RN recompression table and the patient had to have been studied with HMPAO within one month of the incident. One month was chosen to avoid the possible effects of resolution over a longer period of time.

22 divers were excluded based on the following reasons: unconscious or rapid ascent with disputed diagnosis (5), scans performed at greater than 1 month (4), definite incidents with no or inadequate initial treatments (10), oxygen toxicity (1) and cases determined to be non-diving illnesses (2).

The 28 patients selected represented a range across the spectrum of dysbaric illness. Of the 23 reported cases with neurological DCS, 4 had severe manifestations with 1 paraparesis, 1 paraplegia and 2 hemiplegias. 3 patients showed widespread motor and sensory symptoms and signs, 5 had evidence of mild motor and sensory involvement and 5 had subjective sensory changes only. 5 of the 8 patients with altered mentation had vague or absent associated symptoms and 1 patient had inner ear DCS.

In all patients with either CAGE or neurological DCS, there appeared to be a significant correlation with the cerebral perfusion deficits noted on the HMPAO scan and their presenting symptoms and signs. In the few cases of non-neurological DCS, the scans appeared normal.

Pathology of DCS

The similarity of scan results in the CAGE and neurological DCS groups may indicate a similar etiology. While the presence of autochthonous bubbles cannot be ruled out, the pattern suggests a microembolic event of homogeneous nature affecting selected areas of the brain, primarily the regions supplied by the anterior and middle cerebral arteries.[4] This does not preclude the presence of spinal cord involvement but, rather, expands upon our understanding of the often vague cerebral manifestations. In CAGE, the cerebral insult acts alone to cause clinical manifestations while in DCS, it may be that spinal and cerebral insults act alone or in combination.

Astrup and Symon proposed a model where cerebral hypoxia, due to decreased cerebral flow, could lead to reduced or absent function without cellular demise. Reversible and non-reversible areas of damage occur depending upon the degree and duration of the hypoxia.[5,6] Hypoperfusion, caused by either CAGE or DCS might lead to reduced function with subsequent symptoms and/or signs. Recompression with increased perfusion may lead to clinical resolution of a diver's symptoms whilst subclinical cortical hypoperfusion remains.

Follow Up Studies

In a follow up study of 18 of these divers, it was shown that the perfusion deficits shown on initial scanning were remarkably persistent.[7] While some improvement occurred, and indeed complete resolution in a number of cases, it was not uncommon for lesions to worsen or remain unchanged over periods of a year or more. Several divers showed worsening scans with apparent extension of their initial deficits. These findings increase concern that neurological damage caused by diving may be more significant than previously believed and may be of a more permanent nature despite prompt and clinically effective therapy. Underlying damage remains and raises the question of what further diving will do to an already damaged brain. Are these divers at increased risk of further incidents? If injured a second time, will their injury be harder to treat or is it likely to leave greater residual damage? No one can answer these questions at the present time. People have been diving for years following repeated incidents of CAGE and DCS without revealing any definite trend. Long term neurological changes have been documented in the spinal cord[8] and have been suspected to occur within the brain for some time. As our methods of studying these divers becomes more sensitive and accurate, these questions may be answered.

References

1 Ell PJ, Jarritt PH, Cullum I, et al. A new regional cerebral blood flow mapping with Tc-99m labelled compound. *Lancet.* 1985; ii: 50-51.

2 Macleod MA, Adkisson GH, Fox MJ and Pearson RR. $^{99}TC^{m}$-HMPAO single photon emission tomography in the diagnosis of cerebral barotrauma. *Br J Radiol.* 1988; 61: 1106-09.

3 Adkisson GH, Macleod MA, Hodgson M, et al. Cerebral perfusion deficits in dysbaric illness. *Lancet.* 1989; ii: 119-122.

4 Pearson RR, Hodgson M and Adkisson GH. *Preliminary findings on the use of "HMPAO" scanning to study divers and submarine escape trainees who have suffered from dysbaric illnesses. INM Report 7/89.* Alverstoke, England: Institute of Naval Medicine, April 1989.

5 Astrup J, Symon L, Branston NM and Lassen NA. Cortical evoked potential and extracellular K^+ and H^+ at critical levels of brain ischemia. *Stroke* 1977; 8: 51-57.

6 Symon L and Branston NM. Ischemic brain edema following middle cerebral artery occlusion in baboons: Relationship between regional cerebral water content and blood flow at 1 to 2 hours. *Stroke* 1979; 10: 184-90.

7 Adkisson GH, Hodgson M, Macleod MA, Sykes JJW and Pearson RR. Cerebral perfusion deficits in dysbaric illnesses, follow up studies in 18 Divers. *European Undersea Biomedical Society (EUBS) Proceedings* 1989; 55-60.

8 Palmer AC, Calder IM and Hughes JT. Spinal cord degeneration in divers. *Lancet* 1987; ii: 1365-66.

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TESTING THE RECREATIONAL DIVE PLANNER

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Summary

In phase 1 M. R Powell, PhD, tested 911 dives, 518 in the chamber and 393 in open water. The broad cross section of subjects had wide variations in dive profiles. All dives were past RDP limits. There were no cases of decompression sickness and minimal bubbling. The increase in vacation diving paralleled development of the Recreational Dive Planner (RDP). As multi-day diving was largely unstudied Diving Science and Technology (DSAT)