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NEUROLOGICAL DEFICITS AFTER DIVING

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Abstract

The residua of acute decompression illness are relatively obvious, their origin is not in dispute and they are not considered further in this context.

The neurological sequelae which have given rise to recent anxiety are those which might arise in an individual who has never had decompression sickness. The majority of allegations result from studies of neuropsychometrics, neurophysiology and neuro-imaging. Among possible causes for "abnormalities" are the "silent" bubble or the failure to recognise decompression symptoms. The High Pressure Neurological Syndrome (HPNS) has been blamed for some findings after very deep dives as has the "unmasking" of effects from an earlier head injury. Many of the conclusions are, at the very least, debatable. It can be concluded only that, while clinical vigilance must be maintained, there is as yet no evidence of long-term damage among those who dive within recreational limits and adhere to recommended diving procedures.

Introduction

The first case of neurological decompression illness was reported exactly 150 years ago.[1] Since then the neurological deficits which can occur after diving have been all too obvious. When a diver becomes paraplegic as a result of diving, whether he was diving in accordance with accepted procedures or whether he flouted them, the cause of his paralysis can be identified as one particular dive. The subsequent natural history is that, unlike paraplegia due to transection of the cord, the diving caused paraplegic, who has multiple discrete multi-level lesions, has a much greater chance of subsequent improvement. Indeed, the extent of functional recovery can be quite remarkable. Only rarely is there subsequent deterioration and this appears to be confined to those who are severely neurologically injured, whose ability to cope depends so much upon willpower and among whom a few may be unable to continue the required level of effort. The natural history of acute decompression illness (DCI) is that it may lead to neurological deficit, which might improve subsequently.

In the last few years there have been an increasing number of publications in the scientific literature which allege or report neurological deficits in divers as a consequence of their occupation. This has led to a number of articles in journals such as *The Economist* and *The New Scientist* with titles such as "Hidden Cell Damage Puts Divers in Peril" and "Diving Disease Linked to Brain Damage". The implication is that divers who perform normal dives and with no episodes of decompression illness, may progress to subtle neurological deficits.

These two extreme examples may be linked by the concept of "sub-clinical decompression illness" a condition in which bubbling and the associated haematological effects occur but which never produce sufficient symptoms to prompt the diver to seek recompression.

The purpose of this paper is to review the current status of so-called neurological long-term health effects. It is tempting to begin at the beginning, but there is not enough time. An excellent foundation is provided by the review by Edmonds and Hayward[2] which was presented to the Underwater Physiology Symposium in Japan. In this paper the authors demolish with accuracy the design of a number of papers which, nevertheless, are still regularly quoted. They concluded that more assessment is required even though their studies of the Australian abalone divers did not confirm any association between brain damage and excessive air diving.

In this review, the studies based upon the post mortem examination of divers will be summarised first. This is to demonstrate that there are indeed pathological changes associated with diving although, of course, these findings cannot tell us to what extent they might have affected the quality of life for the individual.

Pathology

Excluded from this review are those reports which focus on the changes found following acute decompression illness. Nevertheless it is worth taking as a starting point the case of a scuba diver who, in 1976, developed neurological decompression sickness with improvement upon recompression.[3] He was left with some weakness of his left leg and did not dive again. Some four years later the individual was examined by a neurologist at the University of Newcastle Upon Tyne as part of a study of divers. At this time he had brisk reflexes in both legs with clonus at the ankles and knees and showed an extensor plantar response. Together with some other signs, this led to a conclusion that there was residual cortico-spinal tract damage. A few days later, the patient died accidentally. At post mortem the changes in the spinal cord were more widespread than were suggested by the recent neurological examination, but were entirely compatible with the original manifestations at the time of recompression.

The conclusion is that a good functional recovery following neurological decompression sickness (DCS) could lead to the opportunity to be passed as fit to return to diving but would be associated with persistent damage to the central nervous system (CNS).

However, our concern is more with divers who, as far as one can tell from the records, had never suffered acute decompression illness during life. In a group of 11 divers, all of whom except 2 had died in diving accidents, Palmer et al.[4] described the changes which were compatible with the mode of death. But they did not stop there. The cords were also studied for tract degeneration using the Marchi method in which positive staining is not found until 7 to 10 days after the original insult. In 3 of the professional divers there was extra-cellular Marchi-positive material with a beaded appearance indicating myelin degeneration. However, no Marchi-positive material was found within the macrophages, indicating that the causative insult must have been less than about 10 weeks before death.

The implication that cord damage may follow apparently symptom-free diving was not supported by another study[5] which used immunocytochemical staining. The microscopic examination did not reveal signs of nervous tissue lesions or reactive changes in any of the spinal cord sections from 20 divers. While Mork et al. concluded that diving does not lead to lesions in the human spinal cord, they had not included the Marchi stain in their series. The Marchi stain is one developed for studies of neuroanatomy specifically to detect tract degeneration at an early stage and, until some cords are examined both by this method and by the standard immunocytochemical stains, this apparent contradiction will remain unresolved.

The same groups in Cambridge and Bergen have also examined the brains of divers. In a preliminary study Mork[6] found an increased incidence of corpora amylacea in the white matter, hyalinization of the blood vessels and some ependymal and periventricular changes in the brains of some divers. Similar findings have been reported in the brains of 25 divers by Palmer et al[7] with an additional 10 divers in a more recent study, control brain material was obtained from 15 male airmen who died as a result of flying accidents.[8] Hyaline degeneration of small arteries were found in 12 divers, small foci of necrosis in the cerebral grey matter of 8 and evidence of patchy white matter changes in 10 divers. As far as is known none of these divers had an episode of acute decompression illness.

From the very limited number of examinations performed on such material, the difficulty of adequate controls and some apparent contradictions, it is very difficult to draw conclusions but sufficient has been observed to suggest that the laborious task of histopathology in such subjects must continue. The concern is whether or not such findings were associated in life with any decrement of function.

Clinical examination

The meticulous neurological examination of divers is the foundation for any study of long-term effects. In spite of the earlier reports by Rozsahegyi[9] that there might be a progressive disseminated encephalopathy following acute decompression illness, it does need to be remembered that many of the persons whom he studied continued to work in compressed air. If they had reported subsequent manifestations, they were referred to him only if they lost 3 days' work. Under these circumstances, and in the absence of confirmation from any other sources, the hypothesis of progressive encephalopathy is not proven. The conclusions by Rozsahegyi in both clinical neurology and psychometrics, must be regarded with caution. Nevertheless he does describe two cases where symptoms apparently began with no history of previous decompression sickness, an observation which must cause us to examine divers with even greater care but, as yet, without finding a similar progressive illness.

Lehman[10] examined 23 divers who had had decompression illness and 23 non-diving seamen as controls. He found no evidence of any progression of the physical findings of some two to ten years before. It is also important to note that the 23 controls had "a surprisingly high number of neurological signs" although not so many as the divers. A study by Dolmierski et al.[11] of 150 professional divers does not contain enough detail from which to draw firm conclusions. Certainly, they report minor neurological lesions in some divers who are reported not to have had decompression illness, but the value of this paper is to alert us to a problem which needs to be evaluated.

Norwegian professional divers have been studied in great detail.[12] One hundred and fifty six divers were compared with a 100 age-matched non-diving controls. Unfortunately the examinations were conducted unblinded, after the diving medical history had been taken. Also, the criteria used for the label "DCS" were not those used in other studies. If the divers reported fatigue, mood lability, irritability, concentration or memory problems such as inability to remember appointments, this was considered as evidence of a decompression deficit. Autonomic nervous system symptoms included palpitations, diarrhoea and constipation, excessive sweating and sexual dysfunction and each was also considered evidence of decompression sickness. The physical examination recorded, as positive: increased postural tremor, modified Romberg and reduced sensation in the feet. No specific syndrome was detected but, when all the isolated symptoms and signs were added numerically, there was a preponderance in the diving population. As the majority of these divers continued to dive, the significance to the individual of these findings, though

statistically significant, has yet to be fully understood. Further discussion of these and other papers is available in Evans and Shields[13] and Elliott and Moon.[14]

Neuropsychometric investigations

One must be ruthless and exclude the many anecdotal reports of divers who suffer mental impairment and behavioural changes following deep dives in particular. The stories may be true, but there are many confounding variables and no reliable control studies. Indeed, little that is new has been reported since the review by Edmonds and Hayward.[2] Curley[15] found some transient alterations in 25 Navy divers following saturation but with no evidence of neuropsychological abnormalities. In contrast, Vaernes et al.[16] studied 64 deep saturation divers and 32 experienced divers who were only just commencing saturation diving. The authors found some mild-to-moderate changes which could be interpreted as random variations but they state that these could also represent some specific abnormalities. They conclude that their findings are broadly in agreement with Curley[15] in that no major deterioration was evident. Nevertheless they suggest that their more meticulous examination might indicate the presence of a mild pathological process which cannot be detected by standard neurological examinations.

A study of 282 commercial divers and 182 non-diving controls[17] suggested that there is an impairment of cognitive function in apparently healthy divers who have experienced decompression sickness. In those without previous decompression illness there was some evidence of impairment memory and non-verbal reasoning but these changes were interpreted as related to age and not to diving. There was no evidence of clinical personality change associated with diving experience and they conclude that less than 10% of the total decline in divers with no history of decompression illness is due to their diving. The report does not detail the control of, for instance, IQ, head injury or alcohol history and the previous educational attainments of the divers and non-divers was not matched in the major study.

Thus the evidence relating to neuropsychometric changes in diving is not strong but, once again, there is sufficient "smoke" to justify a properly constructed longitudinal study.

Diagnostic imaging

The advances in diagnostic technology over the last two decades has made available to occupational medicine a number of techniques, designed for hospital use, which permit imaging of parts of the CNS not previously observed. The proper approach to problems in occupational health is to construct an hypothesis on the basis of clinical observations and to use whatever techniques are available for the subsequent investigation. In contrast the approach over the last ten years seems to have been one in which a new technique has become available so "lets try it on a bunch of divers to see what it shows". The results have been rather like going through a hedge backwards: ragged with an inability to see ahead.

A MRC workshop on diagnostic techniques in diving neurology concluded that the use of X-ray computerised tomographic scanning (CT) had no place in screening for long-term neurological effects in divers.[18]

Magnetic resonance imaging (MRI) has a much greater potential. Like CT, it was first used in cases of decompression illness but in a study of 156 divers with 100 controls[19] found that up to 33% of all divers had high signal intensity changes whereas these were present in 43% of the control subjects. A very similar study by Rinck et al.[20] came to a similar conclusion. Rinck[21] has challenged conventional diagnosis in these circumstances. He concludes that, despite the fact that several million MRI brain examinations have been performed all over the world during the last decade, ranges of normality still have not been set. He also says that selecting a control group for clinical studies may be a more difficult task than is generally thought, particularly if such a group's range of normality has not been determined and that the results of such studies may have to be interpreted *cum grano salis*.

The use of single photon emission computed tomography (SPECT) and, in particular the use of $^{99}Tc^{m}$ HMPAO was described by Macleod et al.[22] and Adkisson et al.[23] The first use of this technique was in submarine escape trainees with a known episode of cerebral gas embolism. The subsequent use of this technique in divers following acute decompression sickness has led to some uncertainties about interpretation.[23,24] Basically this is because there appears to be no correlation between the four unusual patterns described in the divers with their decompression history[24] and no adequate control series to determine the range of normality.[25] HMPAO is a lipophilic amine which is bound by the cerebral tissue on the first pass through the cerebral circulation after injection. The images from the gamma camera are an indication of perfusion and not of specific anatomical or functional deficits.

It is unfortunate that the techniques used are not standardised between different diagnostic centres. This means that different studies adopt different diagnostic criteria. So no multi-centre comparisons appear to be valid. Studies using HMPAO in a healthy diving population are not likely to be extensive because most centres regard the use of this radioactive marker in apparently healthy persons as unethical. From this it follows that from evidence of long-term neurological deficits is unlikely to come from using this technique. The higher resolution of positron

emission tomography (PET) is being evaluated in some divers after HMPAO but has yet to be used in divers with no history of decompression illness.

The use of retinal fluorescein angiography to examine the fundi of divers has been reported by Polkinghorne et al.[26] This pilot study investigated 84 divers and 23 non-diver controls. The findings were statistically significant with 22% of divers developing pigment changes in their first year of diving but the significance of this is uncertain. Nevertheless the prevalence of pigment changes was 36% in all divers without decompression illness and 92% in divers with a history of decompression illness. At the posterior pole of the eye in divers, there were also dilated arteriolar terminals and microaneurysms, but none in the non-divers. No subject had any loss of visual acuity and the observed changes are often seen in older individuals. Once again, longitudinal studies are needed.

Electrophysiological investigations

The use of the spontaneous electroencephalogram (EEG) and of evoked action potentials has, like other investigations in this field, began with the study of persons who had suffered acute decompression sickness. To use the electroencephalogram in a study of apparently healthy divers needs careful definition of procedure and of diagnostic criteria.[27] Abnormal signs are at best only a possible indicator of pathology which needs to be supported by other evidence. Nevertheless the finding of some EEG changes in a proportion of symptom-free submarine escape trainees does suggest its potential for the detection of sub-clinical abnormalities due to embolism.[28] A study of 21 divers with a history of decompression illness and 37 naval diver controls in Finland[29] found that 57% of the dysbaric group had abnormal EEG findings compared with 21% of the control group.

In a larger study by Todnem et al.,[19] 18% of the divers and 5% of the controls showed abnormal EEGs. The abnormal EEGs were correlated with saturation diving and neurological decompression illness. Because saturation divers more frequently had abnormal EEGs, even in the absence of a history of decompression illness, led the authors to advocate the use of the EEG in the periodical health examination of deep divers. This may be a useful baseline but they do not offer a definition of pass/fail criteria without which the examination has limited value.

A number of studies using evoked responses during and after acute decompression illness have shown that the changes can be significant, but there have been few studies in divers with no such history. The view that the somatosensory evoked potential (SSEP) is less sensitive than a careful neurological examination in detecting abnormalities in divers with the effects of decompression illness[30] is not necessarily relevant to studies of SSEP in the research laboratory where the technique can be more precisely controlled.

Conclusion

Does diving damage your brain? One must agree with Calder[31] that there is positive proof that cerebral vasculopathy develops in divers who are fit for work. The mechanism is somewhat speculative but one possibility is that a primary provoking agent must be damage to the blood vessel wall by a bubble. These changes are subtle but may contribute to an acceleration of the continuous process of degeneration that occurs with aging.[32]

There is only one conclusion. Although the evidence for functional deficits in divers with no history of decompression illness is very slender, there are theoretical mechanisms that would account for such changes. As there are relatively few professional divers in any nation's working population, the annual medical examination of each needs to be recorded not only meticulously but also centrally for the purposes of longitudinal epidemiology. Indeed a proposal for an international divers' medical registry was made by the European Diving Technology Committee some 20 years ago. Since then, the UK Health & Safety Executive has discontinued the Decompression Sickness Registry in Newcastle so that no longer is one able to see how the changing patterns of commercial diving affect diver health. Of course, such studies, whether within a registry or whether as isolated investigations, would be expensive. Against that expense must be balanced the benefits to the community which are brought by the professional diver who, for the foreseeable future, will not be replaced by unmanned robots.

For sports diving, the data is equally unknown but, the risks of long-term damage are probably less than those of an acute incident. So, in closing, may I wish that all your bubbles remain silent.

References

1 Pasley (1843) quoted by Bevan J. *The development of the diving helmet and dress in the UK during the 19th century.* PhD Thesis: University of London, 1991

2 Edmonds C and Hayward L. Intellectual impairment with diving: a review. *Proc 9th Int Symp Underwater and Hyperbaric Physiology*. Eds. AA Bove, AJ Bachrach and LJ Greenbaum. Bethesda: Undersea and Hyperbaric Medical Society, 1987: 877-886

3 Palmer AC, Calder IM, McCallum RI and Mastaglia FL. Spinal cord degeneration in the case of "recovered" spinal decompression sickness. *Br Med J* 1981; 283: 888

4 Palmer AC, Calder IM and Hughes JT. Spinal cord degeneration in divers. *Lancet* 1987; ii: 1365-1366

5 Mork SJ, Morild I, Eidsvik S, Nyland H, Brubakk AO and Giertsen JC. Does diving really damage the spinal cord? A neuropathological study of 20 professional and amateur divers. *Undersea Biomed Res* 1992; 19 (Supp): 111

6 Mork S. CNS findings from 40 diver autopsies. In: *Report of an open meeting of the MRC Decompression Sickness Panel's Long-Term Health Effects Working Group at the University of Hawaii, 2nd International Symposium "Man-In-The-Sea"*. London: Medical Research Council, 1988

7 Palmer AC, Calder IM and Yates PO. Cerebral vasculopathy in divers. *Neuro Pathol Appl Neuro Biol* 1992; 18: 113-124

8 Palmer AC and Calder IM. Brain pathology in divers - a review of recent findings. *Undersea Biomed Res* 1992; 19 (Supp): 75

9 Rozsahegyi I. Late consequences of the neurological forms of decompression sickness. *Br J Ind Med* 1959; 16: 311-317

10 Lehmann HJ, Held K and Werner G. Neurologische Folgezustande der Taucherkrankheit. *Nervenartz* 1970; 41: 189-193

11 Dolmierski R, Kwiatkowski SR, Nitka J, Palubcki J and Laba L. Neurological, psychiatric and psychological examination of divers in the light of their professional work. *Bull Inst Marit Trop Med Gdynia* 1981; 32: 141-152

12 Todnem K, Nyland H, Kambestat BK and Aarly JA. Influence of occupational diving upon the nervous system: an epidemiological study. *Br J Ind Med* 1990; 47: 708-714

13 Evans SA and Shields TG. *A critical review of the literature on the potential long-term neurological consequences of diving*. Aberdeen: RGIT report, 1992

14 Elliott DH and Moon RE. Long-term health effects of diving. *The Physiology and Medicine of Diving, 4th Edition.* Eds. PB Bennett and DH Elliott. London: Saunders, 1993: 585-604

15 Curley MD. US Navy saturation diving and diver neuropsychologic status. *Undersea Biomed Res* 1988; 15: 39-50

16 Vaernes R, Klove H and Ellertsen B. Neuropsychologic effects of saturation diving. *Undersea Biomed Res* 1989; 16: 233-251

17 Morris PE, Leach J, King J and Rawlins JSPR. *Psychological and neurological impairment in professional divers* . University of Lancaster report, 1991

18 Elliott DH and Halsey MJ Eds. *Diagnostic techniques in diving* neurology. Workshop of the Long-Term Health Effects Working Group of the MRC Decompression Sickness Panel. London: Medical Research Council, 1987

19 Todnem K, Skeidsvol H, Svihus R, Rinck PA, Riise T, Kambestad BK and Aarly JA. Electro-encephalography, evoked potentials and MRI brain scans in saturation divers: an epidemiological study. *Electroencephalogr Clin Neurophysiol* 1991; 79: 322-329

20 Rinck PA, Svihus R and de Francisco P. MR imaging of the central nervous system in diving *J Magnetic Resonance Imaging* 1991; 1: 293-299

21 Rinck J. What is normal? *Hospimedica* 1992; Jan-Feb: 20-21

22 Macleod MA, Adkisson GH, Fox MJ, Pearson RR and Houston AS. ^{99m}Tc-HMPAO Single Photon Emission Tomography in the diagnosis of cerebral barotrauma. *Br J Radiol* 1988; 61: 1106-1109

23 Adkisson GH, Hodgson M, Macleod MA, Sykes JJW, Smith F, Strack C, Torok Z and Pearson RR. Cerebral perfusion deficits in dysbaric illness. *Lancet* 1989; ii: 119-122

24 Evans SA, Thornson LF, Smith FW and Shields TG. ^{99}Tc^{m} HMPAO-SPECT imaging in divers. *EUBS 1991 Proc XVII Annual Meeting of Diving and Hyperbaric Medicine* Ed. NS Trikillis Crete: EUBS, 1991; 177-185

25 Hodgson M, Smith DJ, Macleod MA, Houston AS and Francis TJR. Case control study of decompression illness using ^{99}Tc^{m} HMPAO-SPECT. *Undersea Biomed Res* 1991; 18 (Supp): 17

26 Polkinghorne PJ, Sehmi K, Cross MR, Minassian D and Bird AC. Ocular fundus lesions in divers. *Lancet* 1988; ii: 1381-1383

27 Torok Z. Potential contributions and problems of spontaneous EEG to safety in diving. In: *Diagnostic Techniques in Diving Neurology*. Eds. DH Elliott and MJ Halsey. London: Medical Research Council, 1987: 49-60

28 Ingvar DH, Adolfsson J and Lindemark CO. Cerebral air embolism during training of submarine personnel in free escape: an electroencephalographic study. *Aerospace Med* 1973; 44: 628-633

29 Sippinen SA and Halonen JP. Effects of recompression treatment on EEG in diving accidents. In: *9th International Symposium on Underwater and Hyperbaric Physiology*. Bethesda: Undersea and Hyperbaric Medical Society, 1987: 887-892

30 Overlock R, Dutka A, Farm F, Okamoto G and Susuki D. Somato-sensory evoked potentials measured in divers with a history of spinal cord decompression sickness. *Undersea Biomed Res* 1989; 16 (Supp): 89

31 Calder IM. Does diving damage your brain? *Occup Med* 1992; 42: 213-214

32 Spencer PS. Fatal, long-latency neurodegenerative disorders triggered by selective environmental toxins. *Asia-Pacific Symposium on Environmental and Occupational Toxicology* 1987.

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OUTCOME AFTER TREATMENT FOR DECOMPRESSION ILLNESS IN AUSTRALASIA

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Abstract

In the decade, 1983-1992, there were at least 20 published reports of series of Australasian recreational divers who were treated for decompression illness. These series have been reviewed. With one exception they were retrospective and none were controlled. Only two series reported the type and severity of sequelae. Nevertheless, it would appear that conventional treatment regimens are often unsuccessful in controlling such decompression illness and that many divers are left with depressed mood and disordered higher functions.

Introduction

Australasian Hyperbaric Units still use algorithms based on United States Navy (USN) Treatment Table 6 (USN-6) to treat divers suffering from decompression illness (DCI). Although the USN has demonstrated a final success rate of more than 90% for divers treated with a USN 1, 1A, 2, 2A and 3,[1] and also with USN 5 and 6,[2,3] a review of reported outcomes from Australasian Hyperbaric Units suggests that overall success in treating DCI with a USN 6-algorithm may be less than 70%.[4]

Some fundamental differences between these groups (military versus recreational divers, time from onset of symptoms to treatment, type of post-treatment assessment) may invalidate this comparison. Therefore a review of the series of DCI treated in Australasia, and published in the decade from 1983 to 1992, is presented.

Clinical Series of DCI in Australasia, 1983-1992

AUCKLAND, NEW ZEALAND, 1967-1989.

A retrospective review of 23 years clinical experience at the New Zealand Naval Hospital identified records of 125 treated cases of DCI.[5] In most of these, a USN-6 algorithm was used. At discharge, only 57 (46%) had recovered fully; this frequency was not significantly changed in those reviewed subsequently. The timing of these reviews was not detailed, nor was the distribution or severity of sequelae described. However, outcome did not appear to be related to the delay prior to treatment. Some of these patients have been subsequently described in detail. One of these developed severe depression and intellectual impairment despite numerous hyperbaric oxygen treatments.[6] Twenty-five of the divers were treated in 1987; they were carefully monitored over the following year.[7] Two were lost to follow-up. Forty four percent had persistent problems approximately one month after discharge, increasing to 68% at the (about) one year review. In decreasing frequency, the reported problems were depression, problems with higher functions, motor and sensory disorders. It must be noted that these data were acquired retrospectively and (perhaps consequently) conflict with an earlier report of the same population.[8] A subgroup of these divers was also reported elsewhere,[9] but outcome was not described.

CHRISTCHURCH, NEW ZEALAND, 1979-1988

A retrospective review of 10 years clinical experience at Princess Margaret Hospital.[10] showed that 59 divers had been treated for DCI. However the outcome of these divers was not described.

SYDNEY, NEW SOUTH WALES, AUSTRALIA, 1983-1986

Patients admitted to the Royal Australian Navy School of Underwater Medicine (RANSUM) with DCI, from 1983 to 1986, inclusive were treated with a USN-6 algorithm and intravenous hydration.[11] These patients have also been reported on since then and by different authors,[12,13] but subsequent reports add little new data or analysis. Of the 87 entered into the study, 3 left RANSUM after treatment with persistent problems. Forty six presented for a review both at one week and one month after treatment. The frequency of abnormality changed significantly in that time, increasing from the time of discharge to the one week review (46 reviewed; 10 had overt neurological deficits, 22 had an abnormal EEG, 20 had poor psychometric performance), and then decreasing at one month (46; 2, ? and 8 respectively). This study suggested that :

a the time of measuring outcome after DCI is treated is critical and discharge morbidity will over-estimate treatment efficacy;

b the natural history of DCI sequelae is for early resolution;