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Risk of misclassification of decompression sickness

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ABSTRACT

Decompression sickness (DCS) is classified on the basis of which organ system is affected, and neurological DCS is considered more severe than DCS in joints and skin with respect to response to recompression treatment and risk of long-term sequelae. Gas bubble formation interstitially in the tissues or in the circulation is considered to be the mechanism for all types of DCS. Ten patients diagnosed as having DCS in joints or skin, by doctors experienced in diving medicine, underwent clinical examination by a neurologist and had an electroencephalogram. Eight of the ten subjects had findings suggesting central nervous system deficits. The findings indicate that DCS of the central nervous system often accompanies DCS of the joints and skin, and that local skin and joint symptoms may draw attention away from cerebral symptoms. We recommend that all cases with DCS should initially be treated as neurological DCS.

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Key words: bends, diving, neurological decompression illness, recompression treatment

INTRODUCTION

Gas bubble formation interstitially in the tissues or in the circulation is considered to be the mechanism for all types of DCS. Neurological decompression sickness (DCS) has been regarded as more severe than DCS of the joints (bends) or skin. The incidence of neurological DCS seems to have increased, while the overall incidence of DCS adjusted for diving activity has been relatively stable [1, 2]. This may reflect a change in the classification of DCS, or a real increase in the incidence of neurological DCS [3].

Decompression sickness can involve any part of the nervous system. The symptoms and neurological signs vary from mild to severe, and there is a high risk of residual symptoms [4]. Electroencephalography (EEG) may be useful in supporting the diagnosis and the effect of recompression treatment [5]. There are conflicting results concerning the sensitivity of magnetic resonance imaging (MRI) in detecting cerebral changes in DCS [6, 7]. The diagnosis of neurological decompression sickness remains a clinical diagnosis based on assessment of symptoms and a clinical neurological examination.

In this study, patients who were diagnosed as having DCS of the joints or skin, by doctors experienced in diving medicine but who were not neurologists, were examined by a neurologist to evaluate the extent to which the nervous system might be involved.

MATERIAL AND METHODS

Ten divers (9 men) referred for DCS in joints or skin in 1999–2002, and recompressed according to US Navy treatment table VI [8], were included in the study. Median age was 30 years (range 19–41

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years). None of the subjects were considered to have neurological deficits by the physicians who initiated the treatment, all of whom were experienced in diving medicine.

All subjects were examined by the same neurologist (ES) and EEG was recorded within 24 hours after the first recompression treatment. Clinical neurological examination of cranial nerves, sensory system, motor system, co-ordination/cerebellar system, and reflexes including the deep tendon reflexes, and abdominal and plantar cutaneous reflexes, were performed [9].

ELECTROENCEPHALOGRAPHY

The EEG was standard 21-channel recorded at rest, after hyperventilation and during photostimulation [10]. All recordings were evaluated at the same neurophysiological laboratory.

STATISTICS

The prevalence of neurological signs in the general population was assumed to be less than 10% [8, 9]. The probability of obtaining the observed or more extreme numbers of neurological signs was calculated as if the prevalence of neurological signs in the general population were 10, 20, and 30%.

RESULTS

All ten divers had symptoms which might be associated with neurological DCS (Table 1). Seven divers had clinical signs indicating neurological deficits when examined after the first recompression treatment (Table 2). Assuming a chance occurrence of neurological signs in 10, 20, and 30% of subjects, the occurrence in this group of patients was significantly higher ($p < 0.001$, $p < 0.001$, and $p = 0.01$, respectively).

Table 1. Symptoms of acute decompression sickness reported when interviewed after the first recompression treatment

Pain in joints	6
Numbness	4
Headache	3
Dizziness	2
Paraesthesia	1
Itching	1
Loss of concentration	1
Fatigue	1
Poor balance	1
Impaired vision	1

Five divers had abnormal EEG. Assuming the same chance occurrence of abnormal EEG as for neurological signs, the occurrence of abnormal EEG was larger than expected ($p < 0.001$, $p = 0.006$, and $p = 0.047$, respectively). Among these five divers four had abnormal EEGs in terms of increased generalised theta/delta activity (Figure 1), and one diver had a focal abnormality. One diver had normal neurological status in spite of a pathological EEG. Four divers had positive clinical neurological signs as well as abnormal EEGs. Only two divers had both normal neurological status and normal EEGs.

DISCUSSION

In 10 patients initially considered to have DCS in joints or skin only, a trained neurologist found clinical neurological deficits in seven, and there were EEG abnormalities in five.

The findings indicate that DCS of the central nervous system often accompanies DCS of the joints and skin, and that local skin and joint symptoms may draw attention away from cerebral symptoms. The EEG abnormalities are not specific and may indicate multifocal central nervous system lesions. A weakness of the study was that we had no information on previous neurological examinations and EEG, neither before the dive nor before the first recompression treatment.

There are few studies addressing the prevalence of clinical neurological signs in the general population. In the study by Skre [9] the prevalence of neurological signs in the age group 20–50 years can be estimated at less than 10% based on the prevalence of abnormal findings regarding reflexes, sensibility, motor function, and coordination. In the cross-sectional study of professional divers by Todnem et al. [11] the control group consisted of offshore workers and policemen. The prevalence of neurological signs and EEG abnormalities in the control group was less than 5%.

Although the neurologist was not blinded with respect to diagnosis and treatment given, these data suggest a considerable misclassification of decompression sickness, underestimating the proportion of

Table 2. Clinical neurological signs after the first recompression treatment

Sensory disturbance	5
Asymmetrical reflexes	5
Motor dysfunction	2
Unsteadiness	1
Positive Babinski	1

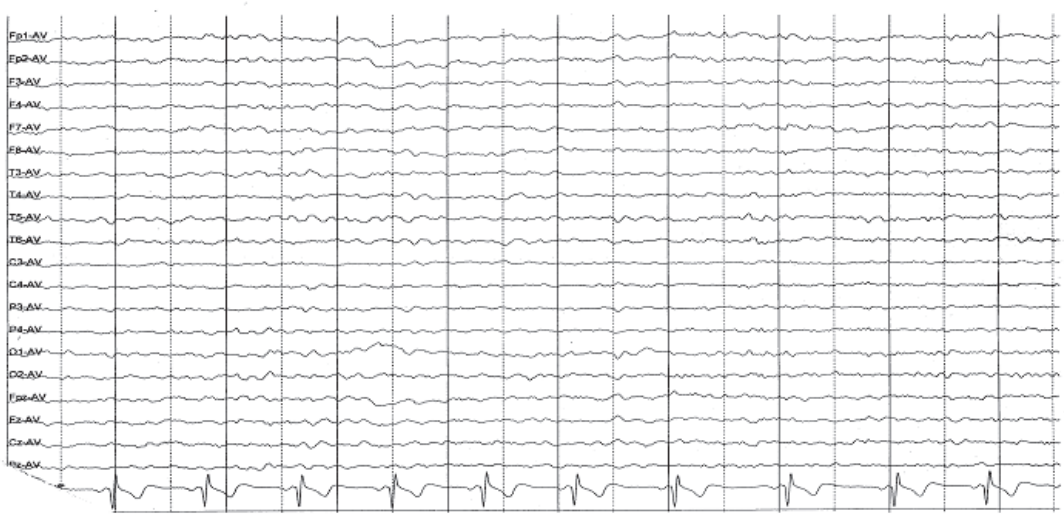

Figure 1. Pathological EEG from a diver. The EEG has continuous, generalised 4–6 Hz theta activity

neurological DCS. Results from previous studies may add support to such a point of view. For example, Staff et al. [12] found significant textural differences in the brains of divers having suffered episodes of DCS and diving controls using ^{99}Tcm-HMPAO SPECT. No differences were found between the two groups of divers who had a history of neurological DCS or joint and skin DCS.

Despite the weakness of such a small study, we recommend that all cases with DCS initially be treated as neurological DCS with US Navy table VI, and followed up by a neurologist.

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