Electroencephalography and magnetic resonance imaging after diving and decompression incidents: a controlled study

S. A. SIPINEN, J. AHOVUO and J.-P. HALONEN

Research Institute of Military Medicine, Naval Department; Department of Radiology, Central Military Hospital, Helsinki; and Department of Clinical Neurophysiology, University of Turku, Finland.

Sipinen SA, Ahovuo J, Halonen J-P. Electroencephalography and magnetic resonance imaging after diving and decompression incidents: a controlled study. Undersea Hyper Med 1999.; 26(2):61–65.—Diving incidents with symptoms of decompression sickness (DCS) and/or arterial gas emboli (AGE) might increase the degree of pathologic change in the electroencephalogram (EEG) or magnetic resonance imaging (MRI) of the supraspinal central nervous system (CNS). Diving itself, even without known symptoms of DCS and/or AGE, has been proposed to increase the number of CNS lesions using either EEG or MRI. In the first part of a two-part study we examined the effects of recompression treatment on EEG in decompression incidents in a group of sport and professional divers compared with a control group of healthy naval divers. In the second part we recorded brain MRI from three groups of volunteers: 1) divers who were treated for DCS in pressure chamber, 2) divers who had never had symptoms of DCS (and/or AGE), and 3) healthy normal controls who were not divers. Our results indicate that DCS increases the incidence of pathologic EEG recordings, whereas recompression treatment decreases them. The results of MRI do not verify evidence of increased numbers of CNS lesions in normal divers as compared to non-diving, healthy control subjects, whereas some of the divers treated for DCS in a pressure chamber had hyperintense lesions in brain white matter. None of them had any abnormalities in EEG, neurologic performance, or psychologic behavior. Both EEG and MRI are sensitive and non-specific methods for judging suspected evidence of brain lesions from diving or diving accidents.

diving, diving accident, decompression sickness, air embolism, magnetic resonance imaging, electroencephalogram, recompression treatment, neurologic sequelae, long-term health effects

Long-term health effects have been examined in several studies dealing with both a normal diving population and diving accidents. Recently, Wilmshurst (1) speculated that "Diving itself may cause brain damage, but we need more evidence". We surveyed our diving accidents in Finland and presented the results of electroencephalogram (EEG) recordings in 1987 (2) and the overall treatment results in 1994 (3). Our follow-up studies have shown a clear effect of decompression sickness (DCS) on EEG and an improvement in it after recompression treatment.

Self-reported, long-term effects of diving and DCS have been published in recreational scuba divers (4) and in an epidemiologic study in occupational divers (5). The first magnetic resonance imaging (MRI) studies of the brain after saturation dives were presented in 1991 by Todnem et al. (6) with a high percentage of abnormal EEGs but no difference in MRI pathology compared to a control population. The same findings were seen concerning neurologic symptoms in professional divers after DCS and successful recompression treatment (7). The first MRI studies of the brain after arterial gas emboli (AGE) and DCS were published between 1988 and 1993 (6,8–10). However the usefulness of MRI techniques in decompression incidents remains controversial.

The aim of the present study was to examine healthy divers, divers after DCS treated or not treated with recompression, and non-diving healthy persons, to correlate the possible pathologic findings obtained from MRI with other neurologic, neurophysiologic, or psychologic symptoms and with other possible long-term health effects.

METHODS

In the first part we called two groups of volunteers to the Central Military Hospital for EEG examination. We examined 21 sport and professional divers who had suffered from DCS and/or AGE between 1976 and 1986 with a standard 22-channel EEG. As controls we examined 37 healthy naval divers who had never had symptoms of DCS or AGE. All 58 EEG recordings were assessed by the same investigator (J-PH), who was unaware of the accident history. EEG classification was based on visual and qualitative inspection of EEG charts recorded on paper (11).

Table 1: Quantification of EEG Findings[a]

	Incident Group	*n*	Controls	*n*	*P* <
Overall normality	1.86 ± 0.19	21	1.27 ± 0.10	37	0.01
General disturbance	1.81 ± 0.18	21	1.16 ± 0.08	37	0.001
Vigilance	1.53 ± 0.19	19	1.11 ± 0.05	37	0.01
Partial irritation	1.05 ± 0.05	21	1.00 ± 0.00	37	NS
IRDA	1.33 ± 0.20	21	1.00 ± 0.00	37	0.05

[a]EEG findings in incident and in control group, mean = SEM; NS = not significant.

Table 2: Effect of Teatment[a]

	Normal EEG			Abnormal EEG		
	n	Percent		*n*	Percent	
Controls	29	(78.4)		8	(21.6)	
Incident group	9	(42.9)	6 with treatment 3 no reatment	12	(57.1)	4 with treatment 8 no treatment

[a]Distribution of normal and abnormal EEG findings in controls and in incident group with or without adequate recompression treatment.

normal EEGs in a small control population (n =12) was 25%. Our present diver population had about the same percentage, although they were in general healthier than Rozsahegyi's group. Of the caisson workers in Rozsahegyi's study (13) who had suffered from a central nervous system (CNS) form of DCS (n = 57), only 25% had a normal EEG.

Ingvar et al. (14) examined submarine personnel undergoing training in free escape and found a pathologic postdive EEG in 12 of 14 cases, with clinical evidence of lung rupture and AGE. Each of these patients was immediately treated in a pressure chamber; untreated controls were, of course, not available. In most of the cases the EEG normalized in a few days or weeks. They concluded that a routine EEG was valuable in following up cases where AGE has occurred.

Irrespective of the small number of decompression incident cases in the first part of our study, the beneficial effects of a recompression treatment were obvious and resulted in a reduced number of abnormal EEGs. The opposite finding was noted when no treatment was provided. Even if the amount of normal EEGs in a general population might be 80–90% (15,16), the high number of abnormal EEGs in clinically healthy divers raised the question of subclinical DCS with CNS involvement. This open question induced us to seek another method to verify if CNS lesions really occur during normal diving.

Neurophysiologic methods, such as recording the somatosensory evoked potentials, visual evoked potentials, or brain-stem auditory evoked potentials had no significant correlation or could not show that neurologic damage had persisted after full clinical recovery from neurologic DCS (17). Comparable results of no objective evidence of retinal damage were obtained using mass screening of blue color vision in divers with the desaturated Lanthony-15-Hue test (18). In 1989 it was reported that after neurologic DCS and cerebral arterial gas emboli (CAGE), cerebral perfusion deficits were present in patients examined using an injection of 99Tcm-hexamethylpropyleneamine oxime and single photon emission tomography (HMPAO) (19). Later, however, another group found the results of the HMPAO-scanning method inconsistent with clinical findings in divers (20).

As a relatively new method, we used MRI for the other part of the study. The study by Reul et al. (21) using MRI to compare normal divers to non-diving controls reported hyperintense lesions of the subcortical cerebral white matter, thus indicating a possibility of CNS lesions in normal divers without history of DCS.

Only 4 of the 78 volunteers in the second study, using MRI, revealed abnormal focal signal intensities in the white matter of the brain. This kind of vascular type lesion may represent an old ischemic injury that occurred during DCS, compared to dysfunction in cerebral neurons demonstrated in the EEG. However, similar white matter lesions are found also in multiple sclerosis, and thus it must be taken into consideration in differential diagnosis. Since psychologic and neuropsychologic examination and EEG were all normal except in one diver, multiple sclerosis can probably

be ruled out. The one patient with minor concentration difficulties during psychologic testing had lost his younger brother in an accident during the previous week, which might explain this result as temporary. Otherwise, he also was normal.

The normal results in brain function tests also let us conclude that focal signal intensities in MRI are not equivalent to brain damage. Even if there has been some damage, the overall capacity of the brain can repair some possible temporary deficits.

The results of this study strongly support our experience, that diving itself does not harm the CNS, assuming that all rules and regulations are strictly followed. The EEG study could only underline the importance of recompression treatment for divers who have suffered from DCS, as seen in a decreased number of pathologic EEG recordings compared to non-treated divers. In a broad review article, the usefulness of EEG and evoked potential studies in the investigation of acute neurologic DCS in man was stated as conflicting (22). Our experience by looking for late effects of recompression-treated divers 3 mo. after HBO_2 therapy supports that, since only 2.4% reported symptoms and at the same time 57.1% had a pathologic EEG (2,3).

Compared to this investigation with small but highly selected study groups, Todnem et al. (6) made a large, epidemiologic study on EEG and MRI mainly in saturation divers. They were all commercial divers who had suffered a high incidence of DCS (51%). Those divers used significantly more alcohol than their controls. The divers also had more abnormal neurologic findings, which might correlate as well with alcohol consumption as with DCS. Their results, however, were rather similar to ours. Pathologic EEG was found significantly more often in divers (with 51% DCS) than in controls, but no correlation was found to other neurologic findings. High signal intensity changes on MRI were found in divers (33%) and in controls (43%). Although both scores are very high, no difference was observed. The reason for such a high percentage of lesions might be explained either methodologically or by a different selection of study volunteers compared to our groups. The results of both of these studies indicate, however, the same sort of differences between EEG and MRI findings.

The study by Reul et al. (21) contains some unpredictable factors concerning patient selection criteria. Also, the pathologic findings in the MR images were only registered, but their significance or clinical correlation to any neurologic or neurophysiologic sequelae was not examined. Our MRI patient material is small but carefully selected and the correlation between similar MRI lesions in brain white matter and clinical neurologic damage was not evident.

Recent reports of Tetzlaff et al. (23,24) of neurobehavioral alterations in professional divers and MRI in diving-related DCS also support the hypothesis that MR lesions do not correlate with neuropsychologic performance and only some DCS patients will have hyperintensities in MRI.

We conclude that EEG alone is a sensible method to record abnormalities in brain electrical activity. It also could be used as an indicator of the effectiveness of recompression treatment in case of DCS. Its correlation, however, to other neurobehavioral performance is not known. An abnormal EEG alone is too common to make conclusions of brain damage or even of fitness do dive.

Magnetic resonance imaging is a sensitive tool to find possible anatomical abnormalities in the CNS after diving and DCS. The results of this study do not exclude that diving itself might cause brain damage, but our results do not support that speculation. The significance of periventricular focal high signal intensity abnormalities in the white matter of the brain is still unclear, as is their correlation to late neurobehavioral performance. More research in this area is required.

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