IS MAGNETOENCEPHALOGRAPHY APPLICABLE IN CLINICAL NEUROPHYSIOLOGY OF DIVING?

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Key Words
Investigations, research.

Introduction

At present neurology uses many brain imaging techniques for diagnosis. In addition to Positron Emission Tomography (PET), Single Photon Emission Computed Tomography (SPECT) and (functional) Magnetic Resonance Imaging (MRI), mostly used to image brain metabolism and cerebral blood flow, multi-channel EEG and MEG (magnetoencephalography) brain mapping are used to quantify, with millisecond time resolution, information processing and spontaneous activity in the human brain.

Since an MEG machine can only be used at normal pressures, physiological research for diving is limited to the application of normobaric gas mixtures, with compositions presumed to mimic, to some extent, the effects of hyperbaric pressure. Possible future applications of MEG in diving medicine are for mapping local brain dysfunction due to severe neurological decompression illness (DCI) and chronic brain disorders due to diving. Nowadays, MRI is used to establish brain damage due to diving,[1] but a negative MRI cannot rule out arterial gas embolism (AGE) or decompression sickness (DCS).[2] As well as showing anatomical damage, dysfunction can objectively be established with MEG and EEG (especially when combined). These techniques can be used for assessing the effects of HBO treatment of patients with brain disorders.

To assess the applicability of MEG we did a pilot study to answer the question whether systemic hypercapnia influences

1 the spontaneous MEG
2 visual MEG responses and
3 sensory cognitive processing.

To provoke systemic hypercapnia we used voluntary breath holding, for convenience called apnoeas in this paper. Apnoeas are required during breathhold diving for e.g. collecting pearl and other sea organisms, underwater photography and underwater games. Systemic hypercapnia was also induced by hypercarbia exposure by breathing CO_2 enriched air (mimicking conditions during long, deep dives, and with failing rebreathers). As far as we know (quantitative) MEG and EEG data during breathholds and systemic hypercapnia has never been published in the recent literature.

Theory and methods

MEG mapping is based on the recording outside the brain of the change of the magnetic field (range 10^{-14} to 10^{-12} Tesla), due to neuronal activity occurring in restricted parts of the brain. This change is measured by the evoked current change in a small sensor, a superconducting coil. Advanced MEG machines have tens of such sensors or channels mounted in a helmet filled with liquid helium.

When a neurone processes synaptic information the highest current density occurs in the main dendritic shaft (if any) and in the axon, for instance, of a pyramidal cell in the cortex. When, in a brain structure, many thousands of such cells have the same orientation, then the activity of this structure can by measured by MEG and EEG. However, MEG is especially suitable when these cellular currents, together represented as an electric dipole, run parallel to the scalp surface. In contrast, EEG is most suitable to record a dipole which is oriented perpendicular on the scalp surface. This means that, when a sensory stimulus is applied, its neurophysiological response can basically be detected by MEG and EEG, but sometimes the response (or a part of the response) is undetectable with the one method whereas it is well detected by the other technique. Table 1 (on page 152) gives the most obvious features of both methods.

On the basis of the features of both techniques it can be concluded that EEG and MEG applied simultaneously will give very complete, detailed, non-invasive, objective information about neurological functioning of the human brain.

The apnoea experiments were done with three male volunteers (45-52 years old, in total 10 sessions) and the hypercarbia experiments with four males (20-22 and 52 years old, each one session). The Declaration of Helsinki tenets were followed. Consent was obtained from the subjects after informing them about the aim of the study, the procedures of the various experiments, the symptoms of hypercapnia and the prevention of hyperventilation.

We used a whole-helmet CTF MEG machine with 62, and later with 151 channels. In addition, in some sessions, EEG (6 or 22 electrodes) was recorded simultaneously. Apnoeas (two or three per session) lasted 120 to 225 seconds and were preceded by normal ventilation (normal apnoeas) or by three deep inspirations (pre-breath apnoeas). There was at least 6 minutes of rest between apnoeas.

Results

Within 150 seconds of normal apnoea, SaO_2 decreased about 4% (finger tip oximetry) and heart rate increased about 4%. However there was large variability in both results across the experiments. Subjects reported

TABLE 1

COMPARISON OF FEATURES OF EEG AND MEG

Feature	Electro-encephalography (EEG)	Magneto-encephalography (MEG)
Signal measured	Potentials at scalp	Magnetic field around head
Temporal resolution	0.1 ms	0.1 ms
Bandwidth	0.1 Hz - 5 kHz	0.1 Hz - 5 kHz
Spatial resolution	1 - 2 cm	0.2 - 0.5 cm
Noise level	0.1 μV	3 x 10^{-15} Tesla
Topographic maps	-	More detailed
Localization source	-	More precise
Radial source	Yes	No
Volume conductance	Yes	No
Influence skull	Yes	No
Preparation time	-	Much shorter
Head movements	Less sensitive	Not allowed
Magnetic protheses (in and at body)	Allowed	Recording impossible
Amalgam fillings and MRI scan made	Allowed	Recording impossible

normal alertness and normal vision during the apnoeas. With closed eyes, apnoeas yield an increase of 0.35 ± 0.06 (mÅSD, 7 sessions) Hz of the peak frequency in the (occipital) alpha rhythm in the spontaneous MEG (and EEG). The peak-amplitude did not change consistently, but the alpha peak became narrower (Q_{3dB} increased 34 ± 14%). Also the (centro-temporal) mu rhythm, occurring in one subject, showed a frequency increase. The effect of normal apnoeas and pre-breath apnoeas seem to be basically similar.

With eyes open the changes of the alpha rhythm and 8-12 Hz alpha band (no alpha peak distinguishable) are inconsistent. The amplitude of the delta rhythm increased under both conditions. The beta (12-40 Hz) and theta (4-8 Hz) bands did not show consistent changes across subjects. The brain maps (2 D representations by iso-fT contours calculated for various time instants of the responses of all sensors) of any band displayed no, or only subtle, changes. The EEG showed basically the same effects.

Hypercarbic exposure (3-4% CO_2 for 35 min or 5% for 5 min) resulted in a transient increase of the frequency and amplitude of the alpha peak (2 subjects) or no changes (2 subjects, 35 min).

Amplitudes of visual evoked responses, to appearing/disappearing checkerboard patterns, applied during apnoeas did not decrease consistently. Also response latencies did not increase consistently. The component in the response related to cognitive functioning, the P300 (investigated with a 'visual odd ball' paradigm), did not change either (2 subjects, in total 3 sessions).

Discussion and conclusion

The brain tissue PO_2 and PCO_2 during apnoeas is not known. The small systemic decrease of SaO_2 does not mean that the PO_2 in the gray matter of the brain diminishes as strongly as in the periphery, where vasoconstriction occurs. In contrast, in the brain, hypoxia as well as hypercapnia produces a vasodilatation, which will increase cerebral blood flow. This will counteract the diminishing of brain tissue PO_2 due to the decrease of arterial PO_2. A further decrease of diminishing tissue PO_2 is caused by the higher heart rate which, however, is supposed to be absent under water (diving reflex). Taking these effects together, it is probable that tissue PO_2 does not change much. The increase of tissue PCO_2, which also induces a small pH decrease, is probably of more importance. Since unpublished data shows that hypoxia alone decreases the alpha frequency, the increase of the alpha frequency (occipital) and the power increase of the delta band (0.5 - 2 Hz, mainly frontal) during apnoeas should be due to hypercapnia.

These preliminary findings about spontaneous activity indicate that the sources of the alpha rhythm (which is located mainly occipitally), the mu rhythm (which is located centro-temporally) and the delta activity (predominantly frontal) are influenced by a long apnoea. At the same time, the invariance of the evoked responses suggest that during apnoeas the occipital cortex and the visual cognitive centres are probably not influenced substantially during subjective normal vision. Possibly, cerebral tissue PO_2 and PCO_2 change insufficiently to establish an obvious effect on vision and cognition.

Therefore, the changes of the alpha, mu and delta rhythms may be caused indirectly.

From this preliminary study we conclude that MEG is applicable in certain aspects of diving research which can be studied at normal pressures. Similarly, as with normobaric EEG examination, it can be used diagnostically in diving medicine. It may also be helpful to elucidate the controversial issue of whether sport diving implicitly has a risk of brain lesions,[3] a notion claimed by several MRI studies.[4,5] However, most promising seems to be its application before and after treatment of neurological DCI with hyperbaric oxygen in a recompression chamber, since MEG (combined with EEG) is a powerful, objective assessment of brain function.

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CAVE DIVING IN AUSTRALIA

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Key Words

Cave diving, deaths, history, safety, training.

Introduction

The first cave dives in Australia were probably dives in the early 1950s in Tasmania and New South Wales. Diving continues in these areas usually as an adjunct to 'dry' cave exploration. The majority of cave dives in Australia take place in the vicinity of Mount Gambier in South Australia and on the Nullarbor Plain. This select history of the cave diving in these latter areas tracks the development of rules, equipment and diving techniques specific to cave diving.

Beneath Mount Gambier is an aquifer in a limestone layer up to 150 m thick. This aquifer is rain fed and flows slowly from the north west to the south east where water exits at springs on the coast, primarily Ewens Ponds. Many sinkholes (cenotes) have formed by dissolution of the limestone at cracks and joints and eventual collapse of the roof of the resulting water filled cavern. Parts of these sinkholes can be dived with either direct access to the surface or with daylight visible.

In the very early 1960s divers in Mount Gambier began to dive the well-known local sinkholes; The Shaft, 10-80, The Black Hole, Kilsby's and Piccaninnie Ponds. By the middle of this decade the word had spread about these enormous caverns filled with crystal clear water and there were many divers from Mount Gambier, Melbourne and Adelaide visiting these sinkholes, it not being unusual to find 6 car loads of divers at any sinkhole on a weekend. For the most part, these dives were conducted within sight of daylight using standard open water diving equipment and techniques. Air supply was typically a single tank with J valve without octopus or pressure gauge. Using wetsuits and usually no buoyancy vests in these deep sinkholes required that weight belts be detached and hooked onto a convenient submerged tree branch. Single, low output lights were used and there were no cave diving reels.

A very few people were exploring the dark zones of sinkholes or entering true caves and experimenting with the forerunners of modern cave diving equipment. There was some experimentation with base fed lines which worked well in the larger sinkholes and much less well in true caves. As a result of this limitation of base fed line the first cave reels were built about 1968. The need to monitor air supply was apparent to the few divers using cylinder pressure gauges. At this time perhaps only one diver was using twin cylinders in some caves. Octopus regulators or truly