Bispectral index with density spectral array (BIS-DSA) monitoring in a patient with inner ear and cerebral decompression sickness

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Abstract

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Bispectral index with density spectral array (BIS-DSA) monitoring during hyperbaric oxygen therapy of a case with inner ear and cerebral decompression sickness is described. During the initial treatment, a particular DSA pattern was found, which resolved after four treatments. Clinical resolution of the symptoms accompanied this improvement. The particular BIS-DSA pattern described in this case is concordant with a potential hypo-perfusion of the cortex related to decompression stress. This case suggests that BIS-DSA monitoring may be an easy, cost-effective, and viable form of neuro-monitoring during hyperbaric oxygen treatment for decompression sickness.

Introduction

Inner ear decompression sickness (IEDCS) is a challenging clinical presentation of decompression sickness (DCS), not just because there can be a confusing differential diagnosis with inner ear barotrauma, but also because of its uncertain pathophysiology and the high percentage of residual symptoms such as hearing loss and vestibular dysfunction.[1,2] Cerebral decompression sickness (CDCS) seems less frequent, and its diagnosis is mainly based on dysexecutive symptoms and sometimes focal manifestations such as weakness and sensory disturbance.[3] What both presentations of DCS have in common is their frequent association with a right-to-left shunt (RLS) which implicates tiny arterialised venous gas emboli in the pathophysiology.[3] The inner ear may be particularly vulnerable to harm from these tiny arterial bubbles because of persistent supersaturation early after diving[4] which allows even small numbers of arriving bubbles to grow and cause harm.[5] Decompression sickness involving the brain whose luxurious perfusion rapidly eliminates supersaturation, may occur when very large numbers of tiny bubbles arrive synchronously causing injury to micro-vessel endothelium with subsequent inflammatory change.[3] The potential vulnerability of the inner ear to arrival of small numbers of tiny arterial bubbles may explain the frequent observation of inner ear but not cerebral symptoms in individual cases.[6]

Electroencephalography (EEG) is a method for monitoring the electrical activity of the brain. It is widely used in the diagnostic workup of patients with chronic neurological disease but is not easily applied on a day-to-day basis for acute neurological patients. Bi-spectral Index (BIS) is based on the continuous monitoring of a processed frontal EEG, and because of the simplicity of its acquisition, it has been shown to be helpful in anaesthetised patients and some neuro-critical settings to monitor depth of anaesthesia.[7] Another important aspect of the processed frontal EEG is its capability to display the obtained data in clinically useful formats like the density spectral array (DSA) and the spectral edge frequency 95% (SEF). For example, SEF values of 8–13 Hz represent an adequate depth of anaesthesia with most anaesthetic drugs.[8]

Even though the use of EEG has previously been described for DCS, this was mostly done for investigational purposes and it is not used in the standard clinical evaluation of patients with DCS.[9,10] The use of BIS in general and specifically of the DSA has not been described up to this moment in relation to management of acute DCS.

Case report

The patient gave written consent for the publication of his case details.

The patient was a 40-year-old otherwise healthy man qualified as an advanced open water diver, with no dives during the six months prior to the diving accident. On the day of the accident, he dived twice, with a first dive to a maximum depth of 22.3 metres of seawater (msw) (327.3 kPa) for 44 minutes and a second dive after a one-hour

surface interval to 22.4 msw (328.3 kPa) for 45 minutes. Both dives were uneventful, with air as breathing gas.

About 20 minutes after the second dive, the patient started to experience vertigo, which worsened during the next hour and was accompanied by vomiting. Because of the severity of the symptoms, he was evacuated by helicopter and admitted to a general hospital. The patient underwent a central nervous and inner ear computed tomography scan, magnetic resonance imaging of the spine, audiometry, and tympanometry. All test results were normal. D-Dimers were within the normal range.

As DCS was suspected, the patient was transferred to our hyperbaric service, receiving oxygen by mask during transport. During the initial evaluation, the patient was completely alert and conscious. Diminished muscle strength was noted in his left quadriceps (3/5), and a strongly positive Romberg test was elicited, with swaying to the right in less than one second. The patient could not stand without assistance and was not able to walk. No signs of middle ear barotrauma were evident on otoscopic examination. No further neurological or dermatologic signs were apparent.

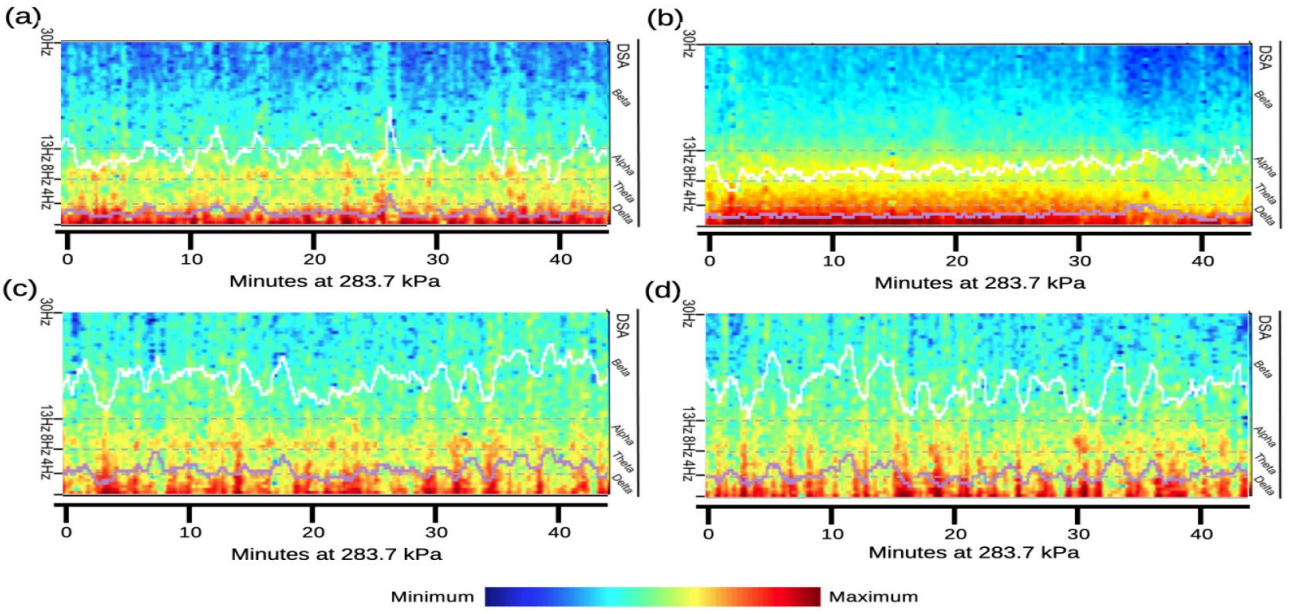
The initial IEDCS risk evaluation showed a value of 11 points on the severity score for inner ear decompression sickness,[11] and the patient was treated with hyperbaric oxygen starting approximately 10 hours after his last dive. The patient was treated in a Sechrist H3300 monoplace hyperbaric chamber (Sechrist Industries, Anaheim – USA). The initial treatment table was based on a traditional United States Navy Treatment Table 6. As the patient reported continuous improvement during the treatment at 283.7 kPa, he received a total of six periods (three extensions) at 283.7 kPa, reporting complete recovery of his muscle strength in his left leg during the last extension and major improvement of his vertigo. He subsequently completed four periods (two extensions) at 192.5 kPa. During the treatment, the patient was monitored according to the monitoring protocol for acute neurological patients of our hyperbaric center, including electrocardiogram and oxygen saturation on a Mindray BeneVision N19 monitor and, for BIS and DSA on a Covidien BIS 4x monitor (Software Version 3.5) using a bilateral sensor (Ref#186-0212) in the standard position recommended by the manufacturer.

For statistical analysis, the SEF values of the first two periods at 283.7 kPa were considered. Each population of values was first checked for normality with a Shapiro-Wilk test, and the comparison between non-normally distributed samples was made using a Mann-Whitney test. Statistical significance was considered in the case of $P < 0.05$. In order to determine the statistical significance of changes in SEF values in air compared to oxygen periods, baseline SEF was recorded every 10 seconds during the last three minutes on oxygen before an air break and compared to the SEF recorded every 10 seconds during the last three minutes of the air break.

During the first HBO treatment, the patient exhibited a reduction of his heart rate by 16% during the first half hour

Figure 1

Density spectral array for each hemisphere during the first 40 minutes of the first and fourth hyperbaric oxygen treatments. The y-axis represents the wave frequency, while the x-axis shows the minutes elapsed at 283.7 kPa. The white line is the spectral edge frequency 95% limit, and the median frequency is shown as a gray line; a – treatment 1, left; b – treatment 1, right; c – treatment 4, left; d – treatment 4, right



of the treatment, stabilising at an average of 82·min^{-1}. The EEG and the DSA showed a predominance of delta waves, primarily in the right hemisphere, generating an asymmetry towards the right side of the DSA. Bearing in mind that mean SEF values of 23 (standard deviation [SD] 4.2) Hz have been reported normal for awake patients,[12] there was a bilateral reduction that was more pronounced on the right side (11–13 Hz left versus 8–10 Hz right) ($P = 0.02$), accompanied by an important reduction of the variability shown in the standard deviation being 0.44 Hz on the right and 1.1 Hz on the left ($P = 0.01$) (Figure 1a and b). No significant changes were found in the DSA during the air breaks. No signs of convulsions were found on the neuro-monitoring. BIS values oscillated between 82 and 97.

The patient was reassessed six hours after the first treatment, showing a complete resolution of the muscle strength deficit in his left quadriceps. The Romberg test remained positive, swaying to the right after 12 seconds. The patient could walk with limited assistance. Approximately 24 hours after diving he received a second HBO treatment on a Table 6 with two extensions at 283.7 kPa and one extension at 192.5 kPa.

There was a second follow-up evaluation 13 hours after the second treatment. At this time (late on day one after diving), he had a positive Romberg test, swaying to the right at 18 seconds, but could walk without assistance. His third HBO treatment was a traditional Table 6 without extensions. No signs of oxygen toxicity were evident either clinically or on frontal EEG.

The patient was reevaluated on day two after diving, and a completely negative sharpened Romberg test was observed. He could walk without assistance but complained about some limited sensation of instability with no positive signs on the clinical neurological evaluation. His fourth HBO treatment was a Brummelkamp table (283.7 kPa for three periods).[13] It is to be noted that all the treatments the patient received had the same structure at the beginning (283.7 kPa for 2–4 periods, each period composed of 20 minutes on oxygen and 5 minutes on air). This is the reason why, for statistical analysis of the measurements, just the first 50 minutes (two periods at 283.7 kPa) of each treatment were considered.

During the last treatment, the patient showed SEF values within the normal range, being significantly higher than in the first treatment on both sides ($P > 0.01$), without asymmetry on the DSA and with a considerably higher variability (standard deviation of 2.7 Hz compared to the measurements during the first treatment [$P < 0.01$]). During the last treatment, there were no significant differences between the left and the right side (Figure 1c and d).

During the evaluation on day three after diving, the patient was completely asymptomatic and was discharged from the hyperbaric treatment. After the initial management, a transthoracic echocardiogram showed an interatrial septal aneurism. On a follow-up call three months after the initial presentation, the patient informed us that he was diagnosed with a persistent foramen ovale.

Discussion

In the past, IEDCS was supposed to be present in 10–20% of patients with DCS and generally associated with other symptoms of DCS.[14] In actuality, the incidence is higher in some contemporary series, and the association with other symptoms of DCS is considered not to be frequent (75% of cases have no other symptoms of DCS).[4] In the current case, the patient had a clinical presentation of right IEDCS associated with symptoms of motor cortex involvement, expressed as muscle weakness in the left leg, and which is compatible with the findings in the right DSA. A predilection for right-sided IEDCS has been previously described, as has the typical short latency of inner ear and cerebral DCS.[15] The association of inner ear and cerebral DCS with right-left shunts is widely reported and discussed.[16] Based on this, the present case may be considered a typical case of IEDCS with right cerebral involvement.

Up to this moment, neuroimaging has not shown to be sensitive at the initial clinical evaluation of these patients[3] but may become more useful at later follow-ups,[16] when ischaemic areas may be found.[17] In contrast to neuroimaging, neuromonitoring is infrequently utilised in DCS, and most reports are limited to EEG in experimental settings. The clinical use of BIS-DSA monitoring during hyperbaric treatment of DCS has not been reported to our knowledge.

Electroencephalography is considered to have low sensitivity for cerebral DCS. Observed abnormalities include slow waves and sharp potentials primarily on the right temporal and occipital regions, with the slow wave being the most typical finding.[9] This predominance of slow waves is compatible with the lower SEF values seen in the present case, as the wave frequency distribution shifts towards lower frequencies, lowering the SEF value.

It is notable that in previous reports, the EEG was obtained after the HBO treatment. A controlled study estimated the incidence of abnormal EEG to be 57.1% before HBO treatment and observed a significant normalisation of the EEG after the HBO treatment.[10] In our case, we used continuous BIS-DSA monitoring during the HBO treatments, initially showing a particular pattern (low average SEF in the alpha range with reduced variability, primarily on the right side) that has not been described before. The pattern resolution was consistent with the clinical improvement during the progress of the treatments.

As previously mentioned SEF values below 13 Hz are typically associated with sedated states. In this case, the patient was completely alert. The pattern shown by the patient is compatible with the loss of high frequency in the EEG that may be seen by low blood flow, causing local

hypoxia in the tissue, mainly related to the oxygen-sensitive layers III, V, and VII of the cortex.[18] In animal models, a reduction of cortical blood flow related to DCS was previously demonstrated.[19] A recent animal study suggested that a neuro-inflammatory state via micro-particles may be involved.[20]

Autopsy reports in cases of cerebral DCS concluded that the patient exhibits alterations of the permeability of the blood vessels and activation of the blood clotting system, resulting in cerebral oedema, perivascular haemorrhages, and oedematous necrosis of the white matter.[21,22] A diffuse perivascular inflammation generated by decompression stress may cause a reduction of local blood flow and/or favour an intracellular hypoxic condition, affecting mainly cortex layers III, V, and VII, potentially causing the BIS-DSA pattern seen in this patient.

Conclusions

The symptoms of the patient indicated a right-sided cerebral involvement that goes in hand with the major changes of the DSA in the right frontal hemisphere.

The fact that the DSA on the left side also deviated from normal values without presenting any clinically evident symptoms points to the hypothesis that CDCS may be a more diffuse and mostly subclinical entity.

The DSA pattern seen in this case is characterised by a bilateral reduction of the SEF with a significant reduction of its variability, both more extensive on the symptomatic right side.

BIS-DSA may be considered a vital monitoring option during hyperbaric therapy in acute neurological conditions and may be a valuable clinical tool in the diagnosis of CDCS.

To our knowledge, this is the first report of BIS-DSA monitoring during HBO treatment in a clinical setting in general and in a patient with inner ear and cerebral DCS in particular.

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