

Effects of Hyperbaric Environment on Human Auditory Middle Latency Response (MLR) and Short Latency Somatosensory Evoked Potential (SSEP)

Shin-ichi WADA^{1,*}, Akira YOKOTA¹, Shigeaki MATSUOKA¹,
Chitoshi KADOYA¹ and Motohiko MOHRI²

¹Department of Neurosurgery, School of Medicine, University of Occupational and Environmental Health, Japan.
Kitakyushu 807, Japan

²Japan Marine Science Technology Center. Yokosuka 237, Japan

Abstract : Hyperbaric chamber dives at 19 ATA with helium-oxygen were performed at the Japan Marine Science Technology Center from November 15 to December 3 in 1988 and from January 25 to February 4 in 1989. During simulated underwater experiments, auditory middle latency responses (MLRs) and short latency somatosensory evoked potentials (SSEPs) were recorded in 3 professional divers (2 divers in each dive) for assessment of brain function. During the saturation dive (180 m below sea level) component Pa on MLR was lost, while component Po remarkably increased in amplitude. These MLR changes rapidly recovered between the beginning of decompression and at about 90 m below sea level. On the other hand, N9-N20 interpeak latency on SSEP slightly or moderately increased in the both divers, but N9-N14 interpeak latency was not affected by the 19 ATA saturation dive. These results suggest that the hyperbaric environment corresponding to 180 m below sea level cause some cerebral dysfunctions, probably between the brainstem and the cortex, but these dysfunctions are only transient.

Key words : hyperbaric environment, brain function, MLR, SSEP.

(Received 15 August 1989, accepted 13 September 1989)

Introduction

Since 1984, in order to investigate the effect of deep sea diving on human health, saturation dives at various levels of depth have been carried out at the Japan Marine Science Technology Center (JAMSTEC). We have participated in some of these experimental series of saturation dives, and have already reported the effects of hyperbaric environment on electroencephalography (EEG) (Matsuoka *et al.*, 1987) and auditory brainstem response (ABR) (Wada *et al.*, 1988). According to our previous report, ABR, which expresses brain stem function, was not affected up to the level of 150 m below sea level, and the hyperbaric environment corresponding to between 150 m and 250 m below sea level caused a transient increase of central conduction time (I-III and/or I-V interpeak latency). On the other hand, auditory middle latency response (MLR) and short latency somatosensory evoked potential (SSEP) also reflect the integrity of a more central brain function,

*Correspondence to Shin-ichi Wada M. D., Department of Neurosurgery, School of Medicine, University of Occupational and Environmental Health, Japan, Iseigaoka 1-1, Kitakyushu 807, Japan

which seems to be more sensitive to such a hyperbaric environment than the brainstem.

The aim of this study is to clarify the effects of the hyperbaric environment with helium-oxygen at the 180 m below sea level on the human brain function by using MLR and SSEP.

Materials and Methods

The experiments in the hyperbaric environment were twice performed in a human diving simulator at JAMSTEC from November 15 to December 3 in 1988 and from January 25 to February 4 in 1989. Three professional male divers participated in the 2 diving tests to 180 m. The divers NH and YK (ages 22 and 21 years old, respectively) participated in the first saturation dive, and divers NH (same person as in the first diving test) and HT (age 27 years old) in second one. MLRs were obtained from the first dive and SSEPs from the second one. The 2 dive profiles were almost the same and are shown in Fig. 1.

The brain electrical activities were recorded between a silver-silver chloride electrode at the vertex (Cz) and the linked electrodes at the left and right mastoids. One hundred μ sec, 'click' stimuli were binaurally administered through a TDH-39 earphone at a rate of

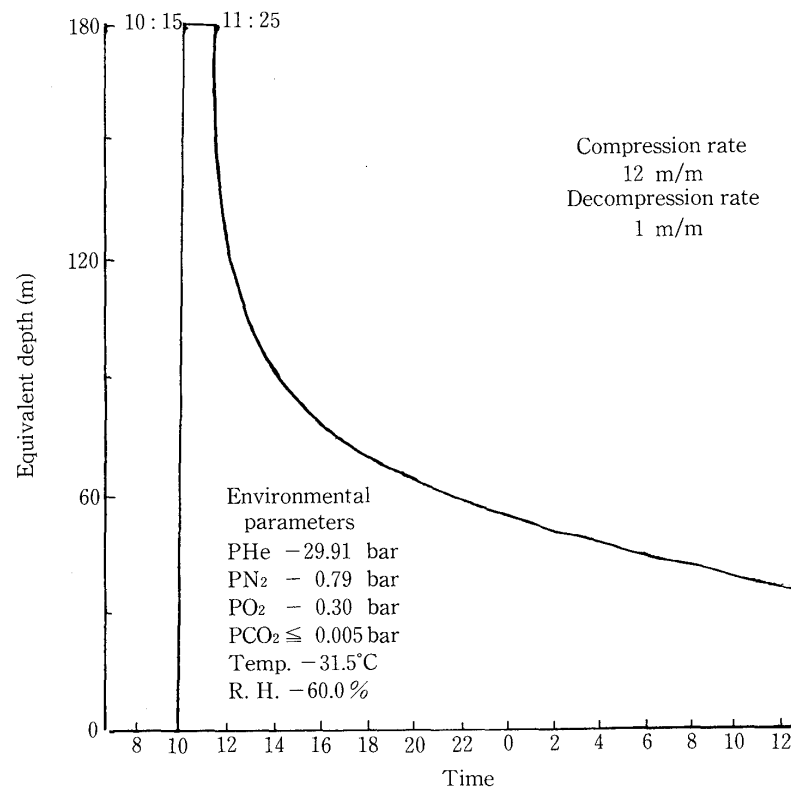


Fig. 1. Dive profile of the second saturation dive performed in 1989. The dive profile of the first one which was performed in 1988 was almost the same. MLRs were recorded during the pre-diving, compression, pressure holding time (180 m below sea level), beginning of decompression and decompression (about 90 m below sea level) periods, while SSEPs were recorded 3 times, i. e., control period, pressure holding time (180 m below sea level) and decompression time (60–70 m below sea level).

5/sec and at an intensity of 80 dB nHL. The brain electrical activities were amplified 50,000 times with a band-pass of 5–3,000 Hz (–6 dB points, 12 dB/octave). The amplified signals were fed into a computer and also monitored on an oscilloscope. In the analysis epoch of 60 msec 1,000 trials were averaged.

Simultaneous 2 channel SSEP recordings were obtained from the contralateral Erb's point to the stimulated hand referred to contralateral hand sensory area (7 cm lateral to the midline and 2 cm posterior to the vertex) and also referred to ipsilateral Erb's point with a gain of 50,000 and a band-pass of 3–3,000 Hz (6 dB down). Responses were analyzed for 30 msec following the stimulus onset. Replicated averages of 500 stimuli each were obtained. The median nerve was electrically stimulated at the wrist at a level just above the motor threshold. The electrical pulse was 200 μ sec in duration, delivered at 4/sec.

Body temperatures of all divers were monitored during the experiments and were between 36.5 and 37.5°C.

Results

Normal MLRs consist of 2 negative and 2 positive components between 8 and 50 msec following stimulus onset and these components were named No, Po, Na and Pa respectively (Picton *et al.* 1974). SSEPs consist of 2 positive peaks (P11 and P14) and 3 negative peaks (N9, N18 and N20) (see Figs. 2 and 3, control periods).

Figure 2 shows MLRs in a case (YK) recorded during 5 periods (see dive profile in Fig. 1). The MLRs during the control period clearly show both Po and Pa (or No-Po and Na-Pa) components. When helium-oxygen compression was rapidly induced, component Po (or No-Po) significantly increased in amplitude (almost 10 times) without significant latency

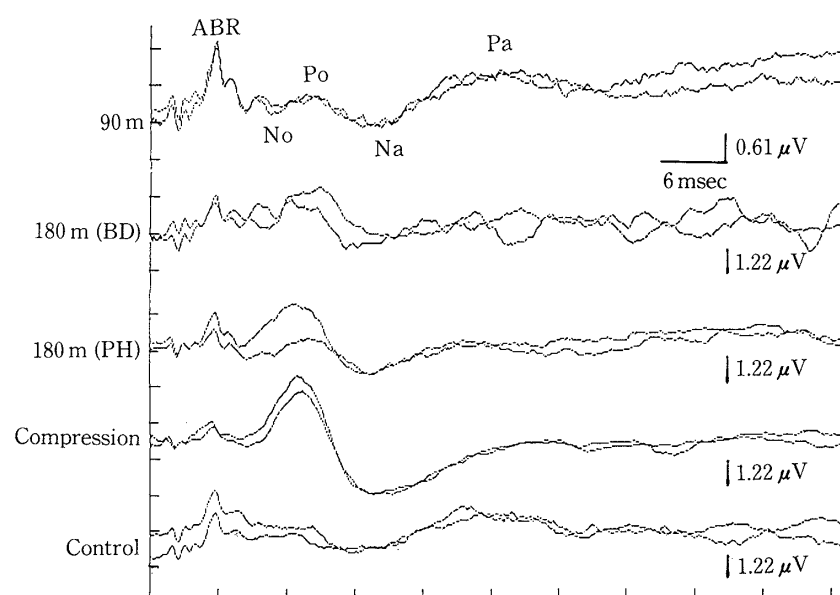


Fig. 2. The results of MLRs (diver YK). BD: beginning of decompression, PH: pressure holding time.

changes, while component Pa decreased in amplitude and increased in latency. During pressure holding time (180 m below sea level), component Po still showed a large amplitude in comparison with its control period, and component Pa could not be detected. This nondetection of the Pa component continued until the beginning of the decompression. When the decompression reached to 90 m below sea level, both components Po (No-Po) and Pa (Na-Pa) were clearly recorded and recovered to almost the same pattern as during the control period. Another diver (HN) showed almost similar MLR changes as diver KY.

SSEPs were recorded 3 times, i. e., pre-dive, pressure holding time (180 m below sea level), and decompression time (60–70 m below sea level) during another saturation dive whose profile was almost the same as the first experiments. SSEPs during the control

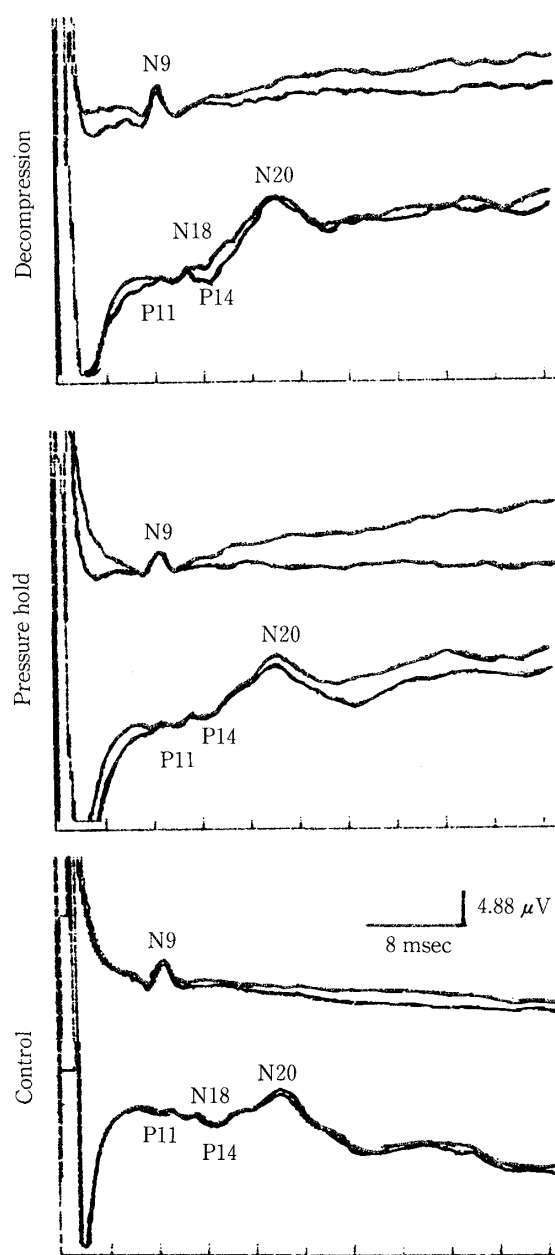


Fig. 3. The results of SSEPs (diver NH).

period in diver NH components N9, P14, N18 and N20 were clearly detected with good reproducibility (Fig. 3), while in diver YK only components N9 and N20 were obtained with good reproducibility because of individual variation, electrode positions and/or EMG or ECG contamination. The interpeak latencies of N9–N20 during the three epochs in diver NH were 9.68, 10.00 and 10.16 msec respectively, which were a slight increase in comparison with the control period. On the other hand, N9–P14 interpeak latency was always the same value (4.0 msec) during the 3 epochs. The N9–N20 interpeak latency in diver YK was moderately increased during the diving period, i. e., 10.8, 12.0 and 11.6 msec respectively.

Divers complained of dizziness, tremor, inattention etc. between 140 and 180 m below sea level, but these symptoms were mild and immediately disappeared following the beginning of decompression.

Discussion

The detection of high pressure nervous syndrome (HPNS), which is considered to occur at the hyperbaric environment of around 200 m below sea level (Bennet & Towse, 1971), and its prevention has been an important problem in regard to working safety at the bottom of the sea. In order to elucidate the mechanism of HPNS various electrophysiological studies have been conducted (Naquet *et al.*, 1984; Hock *et al.*, 1966; Matsuoka *et al.*, 1987). Recently, cerebral evoked potential studies, especially far-field potentials such as auditory brainstem response (ABR) or SSEP have made rapid advances and have made possible the obtaining of the deep brain functions without any invasive procedure. However, there have been few such studies under hyperbaric conditions probably because of technical difficulties. We have already reported the effects of saturation dives in a 31 ATA helium oxygen environment on human ABR and how the environment corresponding to 150–250 m below sea level reversibly affected brainstem function (Wada *et al.*, 1988). On the basis of this previous study we conducted the present study by using SSEP and MLR for examination of telencephalon and diencephalon which seemed to be more easily affected by low pressure than the brainstem. Since MLR is considered to be generated by the auditory pathway between the upper brainstem and the temporal auditory cortex (Picton *et al.*, 1974; Kaga *et al.*, 1980; Kraus *et al.*, 1982; Cohen, 1982; Woods *et al.*, 1987; Kadoya *et al.*, 1988) and since N9, P14, N20 on SSEP by the brachial plexus, cervicomedullary junction and sensory cortex respectively (Lesser *et al.*, 1981; Desmedt & Cheron, 1981; Lüders *et al.*, 1983; Mauguière *et al.*, 1983; Hashimoto, 1984; Urasaki *et al.*, 1985), these evoked potentials were considered as answers to our problems.

The results of this study indicated that the hyperbaric environment corresponding to around 180 m below sea level caused a disappearance of Pa component and an increase of Po amplitude on MLR. Component Po is often enhanced by post auricular muscle reflex (Wada *et al.*, 1987; Kadoya *et al.*, 1988) and the increase of Po amplitude was probably

caused by the diver's tension due to compression. On the other hand, a marked decrease in Pa amplitude or loss of Pa must mean some dysfunction of the cerebral cortex or its surroundings, because the Pa component is considered to be generated in these areas (Kaga *et al.*, 1980; Woods *et al.*, 1987; Kadoya *et al.*, 1988).

The effect of the hyperbaric environment corresponding to 180 m on the SSEPs in diver NH showed only a mild increase of N9–N20 interpeak latency without any changes of N9–P14. Furthermore, the interpeak latency of N9–N20 in diver YK, in which components P11, P14 and N18 could not be clearly detected, increased by about 1 msec. Considering the fact that no changes of N9–P14 interpeak latency took place in diver NH and our previous report which showed that the ABRs were not affected by the hyperbaric environment corresponding to 150 m below sea level or less, these interpeak latency changes may be caused by some dysfunctions of the sensory pathway between the brainstem and the sensory cortex.

In conclusion, the results of this study indicated that the hyperbaric environment corresponding to 180 m below sea level must cause some dysfunctions between the brainstem and cerebral cortex, but these dysfunctions were probably transient because of the rapid recovery of MLR. Moreover, these transient MLR and SSEP changes may be related to the diver's transient symptoms such as inattention, dizziness, and tremor.

References

- Bennet, P. B. & Towse, E. J. (1971): The high pressure nervous syndrome during a simulated oxygen-helium dive to 15,000 FT. *Electroencephalogr. Clin. Neurophysiol.*, 31: 383–393.
- Cohen, M. M. (1982): Coronal topography of the middle latency auditory evoked potentials (MLAEPs) in man. *Electroencephalogr. Clin. Neurophysiol.*, 53: 231–236.
- Desmedt, J. E. & Cheron, G. (1981): Non-cephalic reference recording of early somatosensory potentials to finger stimulation in adult or aging normal man: differentiation of widespread N18 and contralateral N20 from the prerolandic P22 and N30 components. *Electroencephalogr. Clin. Neurophysiol.*, 52: 553–570.
- Hahimoto, I. (1984): Somatosensory evoked potentials from the human brainstem: Origins of short latency potentials. *Electroencephalogr. Clin. Neurophysiol.*, 57: 221–227.
- Hock, R. J., Bond, G. F. & Mazzone, W. F. (1966): Physiological evaluation of Sealab II. Effect of two weeks exposure to an undersea 7 atmosphere helium-oxygen environment Deep Submergence system project. U. S. Navy. Northrop Space Labs, Hawthorne December. pp. 16–18, 41–44.
- Kaga, K., Hink, R., Shinoda, Y. *et al.* (1980): Evidence for a primary cortical origin of a middle-latency auditory evoked potential in cats. *Electroencephalogr. Clin. Neurophysiol.*, 50: 254–266.
- Kraus, N., Ozdamer, O., Hier, D. *et al.* (1982): Auditory middle latency responses (MLRs) in patients with cortical lesions. *Electroencephalogr. Clin. Neurophysiol.*, 54: 275–287.
- Kadoya, C., Wada, S. & Matsuoka, S. (1988): Clinico-experimental studies on auditory evoked middle latency response (AEMLR) with specific reference to generation and auditory dominance. *J. UOEH*, 10: 11–30.
- Lesser, R. P., Lüders, H., Hahn, J. *et al.* (1981): Early somatosensory potentials evoked by median nerve stimulation: Intraoperative monitoring. *Neurology*, 31: 1519–1523.
- Lüders, H., Dinner, D., Lesser, R. P. *et al.* (1983): Origin of subcortical evoked potentials to posterior tibial

- and median nerve stimulation: A comparative study. *Arch. Neurol.*, 40: 93-97.
- Matsuoka, S., Okuda, S., Wada, S. *et al.* (1987): Topographic characteristic of EEG during a saturation dive to 31 ATA helium-oxygen environment. *Clin. Electroencephalogr.*, 27: 584-593. (in Japanese)
- Mauguière, F., Desmedt, J. & Courjon, J. (1983): Neural generators of N18 and P14 far-field somatosensory evoked potentials studied in patients with lesion of thalamus or thalamo-cortical radiations. *Electroencephalogr. Clin. Neurophysiol.*, 56: 283-293.
- Naquet, R., Lemaire, C. & Rostain, J. C. (1984): High pressure nervous syndrome; psychometric and clinico-electrophysiological correlations. *Philos. Trans. R. Soc. Lond., B.*, 304: 95-102.
- Picton, P. W., Hillyard, S. A., Krausz, H. I. *et al.* (1974): Human auditory evoked potentials. *Electroencephalogr. Clin. Neurophysiol.*, 36: 179-190.
- Urasaki, E., Matsukado, Y. & Wada, S. (1985): Origins of human short latency somatosensory evoked potentials by median nerve stimulation. *Kumamoto Med. J.*, 38: 117-141.
- Wada, S., Kadoya, C., Yokota, A. *et al.* (1987): Role of inferior colliculus and lateral lemniscus for auditory evoked middle latency response and its lateralization. *In: EEG Topography 1987.* (Tsutsui, J., ed.). Neuronsha. Tokyo. pp. 121-132.
- Wada, S., Matsuoka, S. & Kadoya, C. (1988): Effects of hyperbaric environment on human brainstem function with specific reference to auditory brainstem responses. *J. UOEH*, 10: 317-324.
- Woods, D. L., Clayworth, C. C., Knight, R. T. *et al.* (1987): Generators of middle and long-latency auditory evoked potentials: implications from studies of patients with bitemporal lesions. *Electroencephalogr. Clin. Neurophysiol.*, 68: 132-148.

高圧環境下におけるヒトの中潜時聴覚誘発電位，短潜時体性感覚誘発電位の変化

和田 伸一¹・横田 晃¹・松岡 成明¹・角谷千登士¹・毛利 元彦²

¹産業医科大学脳神経外科学教室

²海洋科学技術センター

要 旨: 大陸棚開発に必要な海中作業技術の確立と安全を目的とした海洋科学技術センターでの潜水シュミレーターを用いて実施された実験に参加し，二度にわたり水深 180 m での高圧環境が中潜時聴覚誘発電位 (MLR)，短潜時体性感覚誘発電位 (SSEP) に与える影響を観察した。MLR は 180 m の急速加圧後 Po 成分の著明な振幅増大，Pa 成分の潜時遅延，振幅低下および 180 m 到達後の消失をみた。しかし，これらの変化は減圧開始後 90 m までに元に復した。一方，SSEP は N9-N20 間の 1.2 msec 以内の潜時遅延がみられた。これらの結果から先に報告した 150 m 以下では ABR に異常を示さなかった結果と考え合わせて，180 m 程の急速な高圧環境は脳，それも脳幹-大脳皮質間に何等かの機能障害を起こすが，これらの機能障害は一過性のものであることが示唆された。

J. UOEH (産業医大誌), 11 (4): 441-447 (1989)