Continuous Changes in Electroencephalographic Topograms and Auditory Reaction Time during Simulated 21 ATA (Atmospheres Absolute) Heliox Saturation Dives

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The present study was designed to investigate EEG changes during two simulated 21 ATA hyperbaric heliox saturation dives. Four professional male divers were attached to 13 electrodes over the scalp and compressed to 21 ATA by helium. Throughout the dives, EEGs were measured and stored on a magnetic FM tape recorder to calculate electroencephalographic topograms.

Three patterns of slow wave increase were clearly distinguished by the electroencephalographic topogram during compression. The first pattern was caused by trains or bursts of theta waves which spread from the frontal region to the central region of the scalp. This pattern occurred at comparatively shallow depth and showed the slowest recovery during the bottom stage among three patterns. The second pattern found in two divers was caused by frontal midline theta bursts, which showed maximum activity at F_z . The frontal midline theta bursts were augmented by a reaction time task. The third pattern was caused by vertex sharp waves which indicated a transition from sleep stage 1 to sleep stage 2. Neurophysiological meanings of these EEG patterns as well as the relationship between EEG changes and other indices were discussed.

Key words: Electroencephalographic topogram, Saturation diving, HPNS

The increase in theta activity of human EEGs under hyperbaric heliox environment was noted in the first dive beyond 35 ATA (Brauer, 1968). Subsequent studies have confirmed this phenomenon and revealed its characteristics. For example, the increase in theta activity is small if a compression rate is low and it tends to reduce if a depth is held constant for a certain period (Bennett & Towse, 1971; Corril et al., 1973; Fructus et al., 1976). Moreover, the theta activity increase is dominant from the frontal region to the central region of the scalp (Rostain & Charpy, 1976; Rostain & Naquet, 1978). Since then, the EEG change has been extensively utilized as the most important index of HPNS (High Pressure Nervous Syndrome) in many saturation dives.

However, recent studies have questioned reliability and meaning of the theta activity increase as a HPNS index because the other symptoms of HPNS, such as tremor and subjective reports (e.g., dizziness, nausea and vertigo), do not always appear with it (Rostain & Naquet, 1978; Török, 1984; Værns et al., 1982, 1987). We think the discrepancy is caused by the fact that there is still no agreement on fundamental features of EEGs under hyperbaric heliox environment. For example, the scalp region where the theta activity appears most dominantly is not clear because electrodes were attached on various sites of the scalp in previous studies, and consequently it is difficult to compare their results. Besides, the depth at which the theta activity began to increase ranges from 16 ATA (Værns et al., 1982,

1987) to more than 30 ATA (Brauer, 1968; Frustus et al., 1976).

In addition, the problem is furthermore complicated by the recent report that Fm (Frontal midline) theta activity was the sign of HPNS (Matsuoka et al., 1987), because Fm theta activity has been considered as a normal EEG although its appearance is rare in resting states and is usually restricted in specific settings (Mizuki, 1984; Yamaguchi, 1983, a, b, c).

Therefore, the present study was designed to investigate continuous changes in topographic patterns of EEGs, i.e., contour maps of EEGs over the scalp, to clarify the nature of EEG activity under hyperbaric heliox environment. Auditory reaction time and tremors were also measured to examine the relationship among HPNS symptoms.

METHODS

Subjects. Four male professional divers were served as subjects (mean age, 30.5 ± 6.72 years old). They were selected from 12 divers of two simulated 21 ATA dives (diver A and B from DIVE I; diver C and D from DIVE II).

Diving schedule. The schedules and environmental control of the two simulated 21 ATA dives are

shown in Fig. 1. The same compression rate was used in both dives. At first, the chamber was pressurized to 2 ATA (10 msw) by air. Then, it was pressurized up to 21 ATA (200 msw) by helium with intermittent pressure-holding stages at 13 ATA (120 msw) in both dives and 16 ATA (150 msw) in the case of DIVE 1. The compression rate from 2 ATA to 13 ATA and from 13 ATA to 21 ATA were 1.0 m/min and 0.5 m/min, respectively.

The differences between the two dives were a duration at the bottom stage, i.e., the maximum storage depth, and intermittent pressure-holding stages. In DIVE I, divers went to 21 ATA after 8 hours holding at 13 ATA and 36 hours holding at 16 ATA, and stayed for 29 hours at the depth. In DIVE II, divers went to 21 ATA after only 8 hours holding at 13 ATA, and stayed for 90 hours at the depth.

EEG measurement. Thirteen electrodes were placed on the scalp according to the international 10-20 method: the electrode sites were Fp₁, Fp₂, F₇, F₈, C₃, C₄, T₅, T₆, O₁, O₂, F_z, C_z, and P_z. The ground electrode was placed on the forehead. Referential derivations from these electrodes were made by using linked ear lobes (i.e., A₁+A₂) as reference electrode by a electroencephalograph (EEG 4321, Nihon Kohden Inc.) and simultaneously stored on a

Fig. 1 The schedules and environmental control of DIVE I and DIVE II.

magnetic FM tape recorder (A-816, Sony Magnescale Inc.). The time constant was 0.3 seconds and the high-cut frequency was 30 Hz in all channels.

About two hours before compression, the scalp electrodes were tightly attached by collodion and the other electrodes by surgical tapes. Immediately after this procedure, 1 ATA (0 msw) control measurement was done.

Throughout compression, EEGs were continuously measured. The divers were instructed to stay quietly on beds with their eyes closed except that they were asked to report their mental and physical states and to perform tasks. Since the other ends of the electrode leads were connected to a input panel by way of a multichannel connector, the divers were able to move freely inside the chamber by detaching the connector, if necessary.

In DIVE II, EEG measurement was performed on the fourth day of the bottom stage to see if there was any adaptive indication to hyperbaric heliox environment. In this case and also during decompression, ordinary silver cup electrodes and electrode paste were used. Non-volatile skin cleansing jelly (Skin Pure, Nihon Kohden, Inc.) was applied to degrease the electrode sites.

During decompression, EEG measurement was continued at 18 ATA (170 msw), 12 ATA (110 msw) and 7 ATA (60 msw) in both dives. Post-decompression measurement was done two days after surfacing in DIVE I and three days after surfacing in DIVE II.

Processing of EEG topogram. Electroencephalographic topograms were calculated by a medical computer system (ATAK 450, Nihon Kohden Inc.). Thirteen channels of reproduced EEGs from the FM tape recorder were simultaneously digitized by a multichannel 10 bit A/D converter for 5-sec epoch (sampling period, 19.53 msec; frequency resolution, 0.2 Hz) and stored on a floppy disk. This process was continuously repeated for four times, resulting in 20-sec analysis time. Then, power spectra of the EEGs were calculated by using Fast Fourier Trans-

form for each 5-sec epoch.

By averaging the power spectra of four 5-sec epochs, the mean power spectrum was obtained. Then the mean power spectrum was divided into four frequency bands, i.e., delta (2-4 Hz), theta (4-8 Hz), alpha (8-13 Hz) and beta (13-20 Hz). Components less than 2 Hz and more than 20 Hz were not used because these components were easily contaminated by various artifacts. The square root of summed power within each frequency band was defined as an equivalent potential of the corresponding frequency band in a 20-sec EEG. Equivalent potentials at non-measurement electrode sites (i.e., F_3 , F_4 , T_3 , T_5 , P_3 and P_4 according to the international 10-20 method) were defined as the mean value of neighboring three or four measured electrode sites (e.g., $F_3 = (F_{P1} + F_7 + F_z + C_3)/4$). These calculations were performed for every channel.

Then, a distribution of equivalent potentials over the scalp was estimated for each frequency band by a mathematical interpolation using the equation by Ueno and Matsuoka (1976). Finally, the distribution was quantified to ten intensity levels with equal intervals (from 0 to 9 in increasing order), which were represented by ten different colors (blue to red in increasing order. See the scale of Fig. 3, 4, 5 and 6). Thus the EEG topograms were displayed on a color CRT monitor and photographed by a Polaroid camera. Numerical data of each EEG topogram were printed out on a line printer.

Measurement of auditory reaction time. Eighty tone bursts (2000 Hz, 80 dB SPL, 50 msec duration with 10 msec rise-decay time) were presented through binaural headphone speakers. Interstimulus intervals were randomly altered between 2 and 5 seconds. The divers were instructed to respond to these stimuli as quickly as possible by pushing a microswitch. In case that divers failed to respond to these stimuli, additional trials were performed. The control of stimulus presentation and measurement of reaction time were automatically performed by a personal computer (Apple II Plus, Apple Computer

Inc.,).

Many researchers have pointed out difficulties in using auditory stimuli under hyperbaric environment, because high density gas itself causes complex effects on performance characteristics of both human ears and headphone (or earphone) speakers. However, Farmer (1983) have reported that the hearing threshold at 2000 Hz showed little or no change as a function of depth up to 30 ATA in heliox environment. Therefore, we used 2000 Hz tone bursts for this reason and also for easiness of presentation control in auditory stimuli.

Measurement of tremor. Physiological tremors or resting tremors, postural tremors and intentional tremors were examined throughout the dives. The physiological tremors and the postural tremors were measured by a minor tremor pickup (MT-3T, Nihon Kohden Inc.,) attached on the middle finger of the right hand. The postural tremors were induced by the method reported by Bachrach and Bennett (1971). The intentional tremors were checked by a steadiness test and also by handwriting of numbers, alphabets and diver's own name. However, the detailed results were not reported in this paper because there was almost no change in any of these tremors.

RESULTS

(1) Changes in electroencephalographic topograms of each diver

Slow wave activity, i.e., theta and delta activity, increased in all divers during compression, although the wave form and distribution of the slow wave activity during compression were different among the divers. Pressure-holding and recompression also caused different effects among the divers.

①Diver A (Fig. 3. the scale of topograms: less than $3\mu\text{V}$ ~ more than $27\mu\text{V}$)

A marked increase in theta activity accompanied with a slight increase in delta activity was found from the frontal region to the central region during compression. Trains of theta waves often appeared

in the frontal, central and even parietal regions in EEG traces (Fig. 2-d). In some cases, high voltage ($100\sim 200\mu\text{V}$) theta bursts appeared on background activity composed of low voltage slow waves in the same regions (Fig. 2-e).

Pre-compression EEG was characterized by alpha activity which appeared dominantly in the occipital region and spread to the central region. A few theta waves were noted from the frontal region to the central region (Fig. 2-a, Fig. 3-a).

The first change was a decrease in occipital alpha activity during compression from 5 to 7 ATA (Fig. 3-b). Between 7 and 9 ATA, low voltage alpha activity spread to the frontal region along with the midline. At the same time, low voltage theta activity began to appear from the parietal region to the frontal region. The distribution of the theta activity at this time showed slight left hemisphere dominance (Fig. 3-c). Between 9 and 12 ATA, three patterns appeared by turns. The first pattern (Fig. 2-d, 3-d) was characterized by a marked theta activity increase which spread over the scalp, with the maximum activity existing in the central region. The second pattern (Fig. 3-e) was characterized by alpha activity which also spread over the scalp (diffuse alpha pattern), with the maximum activity existing at Fz. The third pattern (Fig. 3-f) was characterized by theta bursts or slow-alpha bursts which appeared at regular intervals on low voltage background activity. Between 12 and 13 ATA, theta activity began to decrease, with only low voltage sporadic theta waves appearing from the frontal region to the central region (Fig. 3-g).

After eight hours of pressure-holding at 13 ATA, theta activity was hardly recognized and the occipital alpha pattern reappeared although its amplitude was slightly low (Fig. 3-h).

Compression from 13 ATA caused a slight theta activity increase in the frontal and central regions as well as a shift of the center of alpha activity from the occipital region to the parietal region (Fig. 3-i). Then, the frontal theta activity furthermore in-

Fig. 2 EEGs before and during compression. Dominant occipital alpha waves were found before compression (2-a~c). However, various types of slow waves appeared during compression (2-d~g).

Fig. 3 Changes in electroencephalographic topograms of diver A throughout the DIVE I. The upper part of each circle represents the frontal region of the scalp and the lower part of it represents the occipital region. The left side of it represents the left hemisphere and the right side represents the right hemisphere. Activity levels of four frequency bands, i.e., delta (2 - 4 Hz), theta (4 - 8 Hz), alpha (8 - 13 Hz) and beta (13 - 20 Hz), were represented by ten different colors (blue to red in increasing order with equal intervals. Blue: activity less than 3 μ V. Red: activity more than 27 μ V).

creased, accompanied with a decrease in parietal alpha activity (Fig. 3-j). This frontal theta activity was caused by theta bursts which appeared at regular intervals on low voltage background activity. In some cases, the theta bursts were accompanied with body movement. These two patterns alternately appeared during compression from 13 to 16 ATA.

EEG returned to the normal pattern after six hours of pressure-holding at 16 ATA: theta activity reduced and occipital alpha activity appeared again although its amplitude was low (Fig. 3-k).

Soon after compression from 16 ATA, theta bursts or slow-alpha bursts appeared at regular intervals on low voltage background activity (Fig. 3-l). Between 19 and 21 ATA, two patterns alternately appeared. The first pattern was characterized by frontal alpha activity with decreased theta activity (Fig. 3-m). The second pattern was characterized by high voltage theta and/or delta bursts which appeared at regular intervals on low voltage background activity in the frontal and central regions (Fig. 3-n).

Although a marked theta increase disappeared, EEG did not return to the pre-compression pattern during the bottom stage at 21 ATA: there remained some theta waves at C_3 and the center of alpha activity existed at F_z (Fig. 3-o). In addition, the reaction time task activated marked theta activity in the frontal region (Fig. 3-p).

During decompression, theta or delta activity did not increase and EEGs gradually returned to the pre-compression pattern. At 18 and 12 ATA, low amplitude parietal or occipital alpha activity was seen (Fig. 3-q, r). The normal EEG pattern was recognized two days after decompression, although occipital alpha amplitudes were still slightly low (Fig. 3-s).

②Diver B (Fig. 4. the scale of topograms: less than $2.5\mu V$ ~ more than $22.5\mu V$)

Theta activity increased in the frontal midline region during compression in diver B and also in diver C. Sinusoidal theta waves (6~7 Hz, 30

~ $150\mu V$) with the maximum amplitude at F_z were clearly recognized on low voltage background activity in EEG traces (Fig. 2-f). Judging from the shape and distribution of the waves, it is concluded that this theta activity was Fm theta activity as Matsuo-ka et al. (1987) have suggested.

Pre-compression EEGs were characterized by slightly low voltage occipital alpha activity (Fig. 4-a). Two Fm theta bursts were found in five minutes at rest, although theta activity was not recognized by the topogram. Moreover, Fm theta activity was slightly augmented by the reaction time task.

Compression to 8 ATA caused no change in EEGs. Between 8 and 12 ATA, two changes occurred in alpha activity. The first change was decreases in amplitudes of occipital alpha activity and the second change was shifts of the maximum alpha activity region along with the midline (Fig. 4-b, d). Occasionally alpha activity spread over the scalp (diffuse alpha pattern), with the maximum activity existing in the occipital region (Fig. 4-c). This pattern was sometimes accompanied with body movement. Between 12 and 13 ATA, alpha activity furthermore reduced its amplitude and then low voltage slow wave activity occupied most parts of the record, indicating a beginning of sleep stage 1. At the same time, low amplitude Fm theta bursts began to appear (Fig. 4-e).

After eight hours of pressure-holding at 13 ATA, the occipital alpha pattern without Fm theta activity reappeared (Fig. 4-f); the reaction time task activated marked Fm theta activity, however (Fig. 4-g). Soon after compression from 13 ATA, low voltage slow wave activity replaced the occipital alpha activity (Fig. 4-h). Then two patterns appeared: the first pattern was characterized by low voltage parietal or central alpha activity (Fig. 4-i) and the second pattern was characterized by Fm theta activity (Fig. 4-j). These three patterns appeared by turns during compression from 13 to 16 ATA.

EEGs returned to the occipital alpha pattern after six hours of pressure-holding at 16 ATA, although

Fig. 4 Changes in electroencephalographic topograms of diver B throughout the DIVE I. See the legend of Fig. 3 for the explanation of this figure (Blue: activity less than 2.5 μV . Red: activity more than 22.5 μV).

slight theta activity remained at Fz (Fig. 4-k). During compression to 21 ATA, almost the same changes observed during compression from 13 to 16 ATA were repeated (Fig. 4-l, m, n).

Low amplitude parietal alpha activity appeared during the bottom stay at 21 ATA (Fig. 4-o). Fm theta bursts were activated by the reaction time task in this period as seen during the pressure-holding at 13 ATA (Fig. 4-p).

EEGs showed occipital or parietal alpha activity throughout decompression; the reaction time task, however, caused a slight increase of Fm theta (Fig. 4-q). Two days after decompression the normal occipital alpha pattern reappeared, although its amplitude was slightly low (Fig. 4-r).

③Diver C (Fig. 5. the scale of topograms: less than $3\mu V$ ~ more than $27\mu V$)

Theta activity increases during compression in diver C were caused by Fm theta activity as already suggested (Fig. 2-f).

Pre-compression EEGs were characterized by dominance of occipital alpha activity (Fig. 5-a). Although neither theta activity nor delta activity was found in the topogram, a few Fm theta bursts were found during five minutes at rest in EEG traces. Moreover, Fm theta activity was slightly augmented by the reaction time task.

No change was found in EEGs during compression to 5 ATA. Beyond 5 ATA, amplitudes of the occipital alpha waves became low and this state lasted up to 7 ATA (Fig. 5-b). Between 7 and 10 ATA, the occipital alpha waves furthermore reduced their amplitudes, and then low voltage slow wave activity which indicated a transition to sleep stage 1 began to occupy most parts of the record (Fig. 5-c). Occasionally alpha activity increased its amplitude in the occipital region and spread over the scalp (diffuse alpha pattern), being sometimes accompanied with body movement (Fig. 5-d). This pattern continued for 20 to 180 seconds, and then it was replaced by low voltage slow wave activity.

Beyond 10 ATA, alpha bursts appeared at regular

intervals on low voltage slow wave activity in the occipital and the central regions (Fig. 5-e): the durations of alpha bursts were about 1 second. This pattern and the low voltage slow wave pattern (Fig. 5-f) alternately appeared between 10 and 11 ATA. Then Fm theta bursts began to appear on low voltage slow wave activity (Fig. 5-g) and they continued to exist until 13 ATA was reached.

EEGs returned to the normal occipital alpha pattern after eight hours of pressure-holding at 13 ATA (Fig. 5-h). The delta activity at the frontal-pole found in the topogram was caused by eye movement artifacts. In contrast with the resting condition, a lot of Fm theta bursts appeared during the reaction time task.

Soon after compression from 13 ATA, EEGs changed into low voltage slow wave activity (Fig. 5-i) and then Fm theta bursts began to appear (Fig. 5-j). Although the Fm theta bursts continued to increase between 15 and 18 ATA (Fig. 5-k), they tended to decrease beyond 18 ATA (Fig. 5-l). Vertex sharp waves as well as Fm theta bursts appeared on low voltage slow wave activity near 21 ATA. The vertex sharp waves caused not only a slight increase in delta activity around C_z but also extension of the distribution of theta activity (Fig. 5-m).

After 16 hours of pressure-holding at 21 ATA, the alpha activity reappeared in the occipital region (Fig. 5-n). However, EEGs did not return to the precompression pattern: alpha activity spread to the central region and slight theta activity, which was caused by slow alpha waves in theta frequency range rather than theta waves themselves, was found in the occipital and central regions. This pattern lasted for only 90 seconds and was replaced by the Fm theta pattern (Fig. 5-o). The reaction time task performed at this time considerably augmented Fm theta activity (Fig. 5-p).

On the fourth day of pressure-holding at 21 ATA, alpha activity was localized in the occipital region. This pattern lasted for five minutes at rest without frontal theta activity (Fig. 5-q). However, the reac-

Fig. 5 Changes in electroencephalographic topograms of diver C throughout the DIVE II. See the legend of Fig. 3 for the explanation of this figure (Blue: activity less than $3 \mu\text{V}$. Red: activity more than $27 \mu\text{V}$).

Fig. 6 Changes in electroencephalographic topograms of diver D throughout the DIVE II. See the legend of Fig. 3 for the explanation of this figure (Blue: activity less than $3 \mu\text{V}$. Red: activity more than $27 \mu\text{V}$).

tion time task still activated Fm theta activity.

During decompression to 18 ATA, dominant alpha activity reappeared in the occipital region (Fig. 5-r) and further decompression caused no change in EEGs. Three days after decompression, EEGs returned to the pre-compression pattern except that the distribution of alpha activity was slightly wider than that of pre-compression (Fig. 5-s).

④Diver D (Fig. 6. the scale of topograms: less than $3\mu V$ ~ more than $27\mu V$)

This diver showed the least EEG change among subjects during compression. Considerable increase in slow wave activity, mainly delta activity, was recognized in the frontal and central regions at the end of compression. The EEG trace at this period showed that the delta activity increase was caused by vertex sharp waves which indicated a transition from sleep stage 1 to sleep stage 2 (Fig. 2-g).

Pre-compression EEGs were characterized by distinct occipital alpha activity without slow wave activity (Fig. 2-c, 6-a). This pattern continued during compression to 9 ATA.

Beyond 9 ATA, the occipital alpha pattern was replaced by low voltage slow wave activity, i.e., the pattern which indicated a beginning of sleep stage 1 (Fig. 6-b). Occasionally alpha activity increased its amplitude in the occipital region and spread to the frontal region along with the midline, being sometimes accompanied with body movement (Fig. 6-c). The durations of this pattern were between 20 and 120 seconds. These two patterns alternately appeared during compression from 9 to 12 ATA. Then EEGs returned to the occipital alpha pattern and remained unchanged until 13 ATA (Fig. 6-d).

EEGs after eight hours of pressure-holding at 13 ATA resembled those of pre-compression (Fig. 6-e). However, it was noticed that the distribution of the occipital alpha activity was slightly wider than that of pre-compression. Soon after compression from 13 ATA, the occipital alpha activity decreased its amplitude and also its continuity (Fig. 6-f). In addi-

tion, a slight increase in theta or delta activity occurred at F_z between 16 and 20 ATA (Fig. 6-g, 6-h). Beyond 20 ATA, the occipital alpha activity furthermore decreased and was replaced by low voltage slow wave activity (Fig. 6-i). Then vertex sharp waves began to appear, which caused a marked increase in delta activity around F_z (Fig. 6-j). Since body movement frequently occurred at this period, EEGs did not change into sleep stage 2.

During the bottom stage at 21 ATA, EEGs returned to the occipital alpha pattern although the distribution of the alpha activity spread to the parietal region (Fig. 6-k, 6-l). The alpha activity tended to be localized in the occipital region during decompression (Fig. 6-m) and EEGs returned to the precompression pattern three days after decompression (Fig. 6-n).

(2) Common changes among divers

Although each diver showed characteristic EEG changes during compression, pressure-holdings and recompression, there were a few common EEG changes among divers.

Occipital alpha activity was depressed or it was replaced by low voltage slow wave activity in all divers under relatively low pressure (less than 10 ATA); on the contrary, it augmented and spread to the frontal region in three divers (B, C, and D). Shifts of the center of alpha activity from the occipital region to the frontal region were noted in two divers (A and B).

Theta or delta activity increase caused by compression reduced during pressure-holding stages. Although EEGs returned to the pre-compression pattern during the pressure-holding at 13 ATA in all divers, considerable individual differences in EEG recovery were observed during the bottom stay at 21 ATA. Compression from 13 or 16 ATA caused reappearance of theta increase in three divers (A, B, and C).

The reaction time task activated theta activity which had already reduced during pressure-holdings in three divers (A, B, and C). Concerning diver D, his

EEG pattern showed essentially no change during the task.

(3) Fm theta activity

Fm theta activity, found in two divers (B and C) during compression, can be observed in normal subjects under 1 ATA. In addition, it is reported that Fm theta activity is augmented by various psychological factors such as mental tasks. Therefore it is necessary to investigate how Fm theta activity changed during compression in detail. For this purpose, percentages of total appearance time of Fm theta activity during five minutes were calculated every 2 ATA throughout compression from 1 to 13 ATA and every 1 ATA throughout compression from 13 to 21 ATA (Fig. 7).

A few Fm theta bursts were found in both divers before compression; moreover they were slightly augmented by the reaction time task. The Fm theta percentage showed no change up to 11 ATA and thereafter it showed a slight increase between 11 and 13 ATA in both divers. Although the Fm theta percentage was reduced during the pressure-holding stages, it was augmented by the reaction time task.

As for diver B, the Fm theta percentage showed a peak around 14 ATA during compression from 13 to 16 ATA. It showed another peak around 18 ATA during compression from 16 to 21 ATA. As for diver C, it showed a peak around 17 ATA during compression from 13 to 21 ATA. In addition, the reaction time task performed at 15 and 18 ATA caused marked increases in it. In both divers, it was also augmented by the reaction time task during the bottom stage at 21 ATA.

Fm theta bursts sometimes appeared with body movement, such as transient increases in muscle activity, opening or shutting the eyes and deep breaths. Moreover, emotional changes (e.g., anger and surprise) accidentally caused by conversations with an experimenter or fellow divers were accompanied with Fm theta bursts.

(4) Changes in simple reaction time

Table 1 shows mean simple reaction time of each diver throughout the dives. Analysis of variance (one-way layout design) indicated that there were statistically significant differences among depth conditions in all divers. Therefore LSD (i.e., least signif-

Fig. 7 Percentages of Fm theta activity during 5 minutes of resting and task sessions throughout the dives.

Table 1. Mean simple reaction time (msec) of each diver throughout the dives.

DIVER	PRE	COMPRESSION PHASE				BOTTOM PHASE		DECOMPRESSION PHASE			POST	F	LSD ^b
	1 ATA	13ATA	16ATA	18ATA	DAY-1	DAY-4	18ATA	12ATA	7 ATA	1 ATA			
A	210.4 (26.22) ^a	229.7 (35.99)	—	—	245.5 (47.98)	—	226.0 (29.65)	220.3 (25.20)	224.6 (28.08)	225.0 (27.14)	10.460*	13.27	
B	160.3 (31.39)	207.6 (54.30)	—	—	203.6 (35.60)	—	223.8 (51.29)	189.4 (39.24)	208.5 (48.49)	185.4 (38.95)	18.126*	17.83	
C	165.4 (23.65)	213.6 (32.55)	185.7 (21.77)	194.8 (30.36)	218.0 (51.92)	221.9 (61.04)	209.1 (36.83)	203.7 (40.68)	202.1 (31.39)	209.9 (31.64)	15.531*	15.58	
D	192.7 (22.61)	208.0 (29.03)	214.29 (41.19)	233.1 (44.49)	252.9 (65.90)	248.7 (44.90)	267.7 (48.98)	240.9 (41.09)	210.8 (38.51)	210.1 (29.15)	37.092*	17.23	

* $P < 0.01$

a Numbers in parentheses indicate standard deviations.

b LSD indicates least significant difference at 1% level.

icant difference), which showed that the difference between any pair of means greater than this value was statistically significant, was calculated. The reaction time at the bottom (21 ATA) was significantly longer than that of pre-compression in all divers. Although the reaction time showed a tendency to recover during decompression, it was still significantly longer in post-decompression than in pre-compression. There was no correlation between the Fm theta percentage and the simple reaction time (Pearson product moment correlation coefficient, $r=0.06$, $p>0.05$).

(5) Tremor and subjective symptoms

Although physiological tremors, postural tremors and intentional tremors were measured during compression and pressure-holding stages, there was almost no change in any of these tremors.

Three divers (B, C and D) reported drowsiness and dullness as subjective symptoms, when occipital alpha activity was replaced by low voltage slow wave activity. These symptoms disappeared during pressure-holding stages; they, however, reappeared during recompression. After four hours of stay at 21 ATA, the divers reported that they returned to normal states. Diver A reported no subjective symptom throughout the dive.

DISCUSSION

(1) EEG changes during compression

When the first heliox dive beyond 35 ATA was performed, a marked theta activity increase was found in EEG (Brauer, 1968). At present, the EEG change, along with tremor increases and subjective manifestations (e.g., drowsiness, nausea, dizziness and so on), was regarded as HPNS. The theta activity increase during compression was such a remarkable phenomenon that only a few EEGs of bipolar derivation have been analyzed usually by FFT in subsequent dives (Bennett & Towse, 1971; Bennett et al., 1986; Rostain & Charpy, 1976; Rostain & Naquet, 1978; Fructus et al., 1971; Török, 1984; Værns et al., 1982, 1987). Consequently, the nature of EEG per se under hyperbaric heliox has not yet being studied in detail. The discordance among researchers concerning the nature of EEG as a symptom of HPNS may be caused by this methodological problem.

The present study analyzed multichannel EEGs over the scalp and showed that theta activity increases were categorized into three patterns by their wave forms and distributions. Although these EEG patterns have different neurophysiological meanings, the FFT analysis of a few EEGs used in previous researches is not able to differentiate these patterns and merely results in a theta activity increase. The confusion of different kinds of theta activity increases may be one of the reasons for the discordance in interpretation of EEG changes dur-

ing diving.

In addition, slow wave activity increase did not show a linear relationship with compression. Rather, it showed a particular change in each diver. For example, the Fm theta percentage showed a peak at 17 ATA as for diver C, whereas it showed two peaks as for diver B (See Fig. 7). This fact indicates that the timing of EEG measurement is a critical factor. Since EEGs were measured at various periods of compression in previous studies, the difference in measurement timing may be another reason for the discordance in EEG changes as a symptom of HPNS. Therefore, it is insufficient to measure EEG at the end point of compression to analyze complex EEG changes during compression.

Although many researchers have reported that the theta activity increase had occurred at more than 20 ATA (Corriol & Chouteau, 1973; Fructus et al., 1971; Rostain & Charpy, 1976; Rostain & Naquet, 1978), the present study showed that it occurred at less than 13 ATA. This depth is shallower than 16 ATA recently reported by Værns et al. (1982, 1987). Since previous dives used rapid compression speed, the theta activity increase might have occurred in shallower depth than that where EEGs were measured. Taking these facts into consideration, it is desirable to measure EEGs as continuous as possible from the start of compression in future study.

(2) Meanings of EEG patterns during compression

①diver A: midline theta pattern

The characteristics of theta activity increase found in diver A are summarized as follows:

- a) the distribution spread from the frontal region to the central region.
- b) the increase began at relatively shallow depth (7 ATA).
- c) the increase was marked and showed very slow recovery during the bottom stage at 21 ATA.

Corriol et al. (1973) have reported a similar theta activity increase which occurred in shallow depth (6 ATA) around the central region in one diver.

Judging from its wave form and distribution, the

theta activity found in diver A resembles midline theta discharges or rhythm reported by Mokran et al. (1971) and Westmoreland & Klass (1986). Westmoreland & Klass have reported that the origin of the midline theta rhythm was uncertain and that the midline theta rhythm did not correspond to specific neurological or psychiatric disorders. However, they suggested that the midline theta rhythm appeared to represent a nonspecific EEG pattern that could occur in a mixed group of patients with various diagnoses. Further research is needed to investigate whether the theta activity found during compression was the same as the midline theta rhythm or not. It is also unknown whether compression or pressure per se caused the theta activity or they merely activated latent activity which the diver naturally possessed. The latter is more probable because slight but similar theta activity was found even before compression in our diver and also in Corriol's diver.

It has been reported that the midline theta rhythm did not accompany with behavioral or subjective disorders (Westmoreland & Klass, 1986). The theta activity found during compression also did not accompany with them. However, this fact does not mean that the theta activity is a negligible EEG change. Considering the facts that the theta activity increase began at relatively shallow depth and that its recovery was very slow, it is probable that the theta activity will change into abnormal activity accompanied with behavioral or subjective disorders in rapid compression or at greater depth.

②Diver B and C: Fm theta pattern

Matsuoka et al. (1987) have reported that Fm theta activity during compression was closely correlated with HPNS. Rostain et al. (1976, 1978) have repeatedly reported that theta activity increased in the frontal region during compression, and have regarded it as an index of HPNS. Although they have not described characteristics of the theta activity in detail, the EEGs they have presented in figures are similar to Fm theta activity. As already suggest-

ed, the frontal theta activity found in diver B and C is Fm theta activity, judging from its wave form and distribution.

Fm theta activity was first found during various mental tasks by Ishihara and Yoshii (1972). Since then, the nature of it has been extensively studied by Japanese researchers (Ishihara et al., 1979, 1987; Mizuki, 1982; Nishijima, 1985; Yamaguchi, 1983a, b,c) and the following characteristics are found:

a) Fm theta activity is a normal EEG which is found in psychiatrically and neurologically normal subjects, although there is a considerable individual difference in its appearance. The cause of the individual difference is yet unknown.

b) Various mental tasks augment Fm theta activity. High motivation of subjects furthermore augment it. These tendencies are clearly found in subjects who have some Fm theta activity in resting states.

c) There may be positive correlation between quantity of Fm theta activity and performance efficiency. However, no correlation between them is found in some cases.

d) Fm theta activity is occasionally found in resting states, especially in a transitional state from wakefulness to sleep. In these cases, mental activity (e.g., thinkings and images), concentration on bodily sensation (e.g., numbness or itching of the extremities) and emotional experiences are reported.

e) From these characteristics, Fm theta activity is considered to reflect a function of central nervous mechanisms which are responsible for attention or retention of high arousal level.

f) Fm theta activity sometimes appears with body movement.

g) Fm theta activity is correlated with anxiety levels of subjects.

The Fm theta activity observed in our divers had almost the same characteristics as those found in normal subjects at 1 ATA. That is, the Fm theta activity began to appear when occipital alpha activity decreased its continuity or it was replaced by

low voltage slow wave activity, sometimes accompanied with body movement. Moreover, it was remarkably augmented by the reaction time task. Rostain and Charpy (1976) have also admitted that the frontal theta activity during diving was augmented by mental tasks, and that there was no clear relationship between the theta activity increase and performance deficiency. From these facts, it can be said that Fm theta activity during diving is not a specific EEG caused by compression or pressure per se, although it is certain that Fm theta activity is augmented by them because the percentage of it during compression is far higher than that at 1 ATA. Moreover, another important characteristic of Fm theta activity during diving is that it was not accompanied with performance deficiency or subjective disorders. These characteristics, along with the reports that Fm theta activity observed at 1 ATA is a normal EEG, indicate that Fm theta activity during diving is not a reflection of malfunction in the central nervous system, contrary to the suggestions by Matsuoka et al. (1987) and Rostain & Charpy (1976).

It seems that the primary effect of compression or pressure per se on the central nervous system is a decrease in EEG arousal, i.e., the transition from wakefulness to sleep. Appearances of Fm theta activity may be the secondary phenomenon caused by the interaction between the primary effect and aforementioned various psychological factors. In our divers, appearances of Fm theta activity showed a tendency to reach its maximum level slightly before pressure-holding stages rather than linear increase with pressure. This tendency may indicate a change in diver's psychological states. As already pointed out, appearance of Fm theta activity per se is not a sign of malfunction in the central nervous system. However, since Fm theta activity certainly augments in some divers, it can be used as a convenient index suggesting that the divers are psychologically or physiologically influenced by compression or pressure per se.

③Diver D: Vertex sharp wave pattern

The EEG change found in diver D is the appearance of low voltage slow wave activity, i.e., sleep stage 1, which is normally seen in the transition from wakefulness to sleep at 1 ATA. The same change is also found in diver B and C at some stages of compression. In diver D, vertex sharp waves which appeared near the end of compression caused not only a marked delta activity increase but also a slight theta activity increase in the frontal and central regions of the scalp.

In general, sleep stage 1 is a transitional state which does not continue for a long time in normal subjects at 1 ATA: it either changes into sleep stage 2 or returns to awaking state in a few minutes. On the contrary, it continued for four hours during compression from 13 to 21 ATA in diver D. Therefore, it is inferred that the unusual prolongation of sleep stage 1 itself reflects the effect of compression or pressure per se on the central nervous system. Subjective reports (i.e., drowsiness and dullness) at this period corresponded to the EEG change well. The transient increase in occipital alpha activity, which spread to the frontal region, might be related with the decrease in EEG arousal. Since the alpha activity increase was sometimes accompanied with body movement, it is inferred that the phenomenon was caused by diver's effort to keep wakefulness against a sleep-inducing tendency of hyperbaric heliox environment.

Almost all studies concerning EEG changes during deep heliox dives have reported the appearance of low voltage slow wave activity during compression. This EEG activity has been sometimes called microsleep or bout of somnolence because divers complained of difficulty in being wakeful and often went to sleep against their will in this EEG pattern (Corriol et al., 1973; Fructus et al., 1971, 1978; Rostain & Charpy, 1976; Rostain & Naquet, 1978). Although previous studies have reported that low voltage slow wave activity had occurred around 30 ATA, the activity was found at less than 13 ATA in

diver B, C, and D. Considering the possibility that EEG had changed before its measurement was done in rapid compression dives, it is inferred that the appearance of low voltage slow wave activity in previous dives might have occurred at shallower depth.

Slow compression dives like our dives also have factors which have to be taken into account in interpreting the appearance of low voltage slow wave activity, because the activity is easily induced by non-specific factors such as relaxation, boredom and fatigue. Considering the facts that divers were required to keep relaxed states during compression, the possibility that the low voltage slow wave activity was caused by such non-specific factors can not be entirely excluded in case of compression to 13 ATA because it took more than an hour before the low voltage slow wave activity appeared. By contrast, since the transition to low voltage slow wave activity occurred immediately after compression beyond 13 ATA, it is certain that this transition was caused by compression or pressure per se.

(3) EEG changes during pressure-holding stages

Previous studies have reported that pressure-holding stages had the effect of reducing increased theta activity, although the effect was not confirmed for stages exceeding 35 ATA (Bennett & Towse, 1971; Corriol et al., 1973; Rostain & Charpy, 1976; Rostain & Naquet, 1978). This effect was found in all our divers in spite of the fact that characteristics of increased theta activity were considerably different among divers. The present study also showed that the reaction time task activated the theta activity even during the pressure-holding stages in three divers. This fact indicates that the effect of pressure on the central nervous system did not vanish during pressure-holding stages; on the contrary, it continued to exist latently. Therefore, compression from the pressure-holding stage immediately activated theta activity, as previous studies have reported.

(4) Relationship between EEG change and other

indices.

The simple reaction time was significantly delayed during compression and at the bottom stage in all divers. This change probably reflects the decreased EEG arousal caused by compression. Sakamoto et al. (1985) have also noticed that the level of consciousness dropped by compression. However, the simple reaction time was still significantly delayed even after decompression when EEGs returned to the precompression pattern. It is inferred that psychological factors, such as fatigue and motivation, might contribute to the delay in simple reaction time during and after decompression.

There is a general agreement on subjective symptoms during deep heliox dives: they are nausea, dizziness, vertigo, drowsiness, dullness, fatigue and so on. The subjective symptoms found in our dives were drowsiness and dullness which were correlated with the appearance of low voltage slow wave activity. Nausea, dizziness and vertigo, which may be the reflection of malfunction in the vestibular system, were not found in our dives probably due to slow compression speed. It is inferred that the vestibular system is affected by compression or pressure per se in a different way than EEG.

Matsuoka et al. (1987) have recently reported that euphoria and laughter occurred with Fm theta activity during a 31 ATA heliox saturation dive and have regarded them as HPNS. In our dives, drowsiness was the only subjective symptom reported by the divers who showed Fm theta activity during compression. This was true even in the period when the appearance of their Fm theta activity reached the maximum level. In addition, the divers looked rather depressed in behavioral observations.

In a few cases, Fm theta activity did occur with emotional changes, such as anger and surprise, during conversations with an experimenter or fellow divers in our dives. However, the same phenomenon has been reported even at 1 ATA (Ishihara et al., 1979). Therefore, it is inferred that euphoria and

laughter reported by Matsuoka et al. (1987) were not a sign of malfunction in the central nervous system but a reflection of transient emotional changes. Fm theta activity in their report might have been caused by emotional changes themselves.

Tremor increase was not found during compression in our dives. Fructus et al. (1976) have suggested that tremor increase was dependent on compression speed. In addition, Yamasaki et al. (1985) have suggested that there was a considerable individual difference in tremor increase. Therefore, the reasons for absence of tremor increase in our dives might be the slow compression speed and/or an individual difference.

In conclusion, there was a dissociation among various indices of HPNS, i.e., EEG, subjective symptoms and tremor. Rostain & Naquet (1980), Török (1980) and Værns (1987) have also reported the dissociation among HPNS indices and have suggested that HPNS was a collection of multiple symptoms of different origins. Our data support their inferences.

ACKNOWLEDGEMENTS

A preliminary report of these findings was made at the Undersea and Hyperbaric Medical Society Annual Scientific Meeting, Honolulu, 1989.

The opinions and assertions contained herein are the private ones of the authors and are not to be construed as official or reflecting the views of Defense Agency, Maritime Self-Defense Force or Undersea Medical Center.

REFERENCES

- Bachrach, A. J. & P. B. Bennett. 1973: Tremor in diving. *Aerospace Med.*, 44: 613-623.
- Bennett, P. B. & E. J. Towse, 1971: The high pressure nervous syndrome during a simulated oxygen-helium dive to 1500 ft. *EEG Clin. Neurophysiol.*, 31: 383-393.
- Bennett, P. B., Janke, N., Kolb, E. & E. Schwieger. 1986: Use of digital filtering and display for

- HPNS diagnosis. *Undersea Biomed. Res.*, 13: 99-110.
- Brauer, R. W. 1968: Seeking man's depth level. *Ocean Ind.*, 3: 28-33.
- Corriol, J., Chouteau, J. & J. Catier, 1973: Human simulated diving experiments at saturation oxygen-helium exposures up to 500 meters: electroencephalographic data. *Aerospace Med.*, 44: 1270-1276.
- Farmer, J. C. Jr. 1982: Otologic and paranasal sinus problems in diving. In P. B. Bennett & D. H. Ellio (Eds.), *The physiology and medicine of diving*. 3rd. ed., Baillière Tindall. 507-536.
- Fructus, X. R., Brauer, R. W. & R. Naquet. 1971: Physiological effects observed in the course of simulated deep chamber dives to a maximum of 36.5 ATM in helium-oxygen ATM. In C. J. Lambertsen (Ed.), *Underwater Physiology*. Academic Press. 545-550.
- Fructus, X. R., Agarate, C., Naquet, R. & J. Rostain, 1976: Postponing the "High pressure nervous syndrome" to 1640 feet and beyond. In C. J. Lambertsen (Ed.), *Underwater Physiology V. FASEB*. 21-33.
- Ishihara, T. & N. Yoshii. 1972: Multivariate analytic study of EEG and mental activity in juvenile delinquents. *EEG Clin. Neurophysiol.*, 33: 71-80.
- Ishihara, T., Izumi, M. & M. Iwashita. 1979: On the mediators of Fm θ induced at rest-Comparison of the introspection data in three conditions of Fm θ , α -activity and α -attenuation. *Clin. EEG*, 21: 420-424. (in Japanese)
- Ishihara, T. 1987: Appearance of Fm θ in at rest and its relation to mental activity. *Clin. EEG*, 29: 522-525. (in Japanese)
- Matsuoka, S., Okuda, S., Ishikawa, T., Yamamoto, S. & M. Mori. 1987: Topographic characteristics of EEG during a saturation dive to 31 ATA helium-oxygen environment. In A. A. Bove, A. J. Bachrach & L. J. Greenbaum, Jr., (Eds.), *Underwater and Hyperbaric Physiology IX. Underwater and Hyperbaric Medical Society*. 405-414.
- Mizuki, Y. 1982: Introduction to the Fm θ . *Jap. J. EEG & EMG*, 10: 203-208. (in Japanese).
- Mokrán, V., Cigánek, L. & Z. Kabátník. 1971: Electroencephalographic theta discharges in the midline. *Europ. Neurol.*, 5: 288-293.
- Naquet, R. & J. C. Rostain. 1980: High pressure nervous syndrome in man: An account of French experiments. In S. Daniels, M. J. Halsey & E. B. Smith (Eds.), *Techniques for diving deeper than 1,500 feet*. Undersea Medical Society. 48-53.
- Nishijima, H. 1985: On the factors that influence the appearance of Fm θ . *Clin. EEG*, 27: 489-494. (in Japanese).
- Rostain, J. C. & J. P. Charpy, 1976: Effects upon the EEG of psychometric performance during deep dives in helium-oxygen atmosphere. *EEG Clin. Neurophysiol.*, 40: 571-584.
- Rostain, J. C. & R. Naquet. 1978: Human neurophysiological data obtained from two simulated heliox dives to a depth of 610 meters. In C. W. Shillings & M. W. Beckett (Eds.), *Underwater Physiology VI. FASEB*. 9-20.
- Sakamoto, K., Usui, T., Yamasaki, M., Masuya, Y., Seki, K. & Nakayama, H. 1985: Influence of hyperbaric heliox environment at 31 ATA on microvibration and electroencephalogram in four divers. *Annals Physiol. Anthropol.*, 4, 137-145.
- Török, Z. 1980: Deep diving. In S. Daniels, M. J. Halsey & E. B. Smith (Eds.), *Techniques for diving deeper than 1,500 feet*. Undersea Medical Society. 54-61.
- Török, Z. 1984: Behavior and performance in deep experimental diving with man-a review of recent work. In A. J. Bachrach & M. M. Matzen (Eds.), *Underwater Physiology VIII. Undersea Medical Society*. 739-760.
- Ueno, S. & S. Matsuoka. 1976: Topographic display of slow wave types of EEG abnormality in patients with brain lesions. *Jap. J. Med. Electronics & Biol. Engineering*, 14: 118-124. (in Japanese with English summary)
- Værnes, R., Bennett, P. B., Hammerborg, D., E.

- Ellertsen, R. E. Petersen & S. Tønjum. 1982: Central nervous system reactions during heliox and trimix dives to 31 ATA. *Undersea Biomed. Res.*, 9: 1-14.
- Værns, R., Klobe, H. & Ursin, H. 1987: Neuropsychologic and neurophysiologic reactions during a heliox dive to 450 MSW. In A. A. Bove, A. J. Bachrach & L. T. Greenbaum, Jr., (Eds.), *Underwater and Hyperbaric Physiology IX*. Underwater and Hyperbaric Medical Society. 565-572.
- Westmoreland, B. F. & D. W. Klass. 1986: Midline theta rhythm. *Arch. Neurol.*, 43: 139-141.
- Yamaguchi, Y. 1983a: Frontal midline theta rhythm (I). *Clin. EEG*, 25: 276-281. (in Japanese)
- Yamaguchi, Y. 1983b: Frontal midline theta rhythm (II). *Clin. EEG*, 25: 352-358. (in Japanese)
- Yamaguchi, Y. 1983c: Frontal midline theta rhythm (III). *Clin. EEG*, 26: 425-431. (in Japanese)
- Yamasaki, M., Sasaki, T., Sakamoto, K., Shidara, F., Taya, Y., Nakano, M., Seki, K. & Nakayama, H. 1985: Power spectral analysis of intentional tremor and postural tremor during a simulated heliox dive to 31 ATA. *Annals Physiol. Anthropol.*, 4: 129-136.

(Received June 28, 1989)