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The Influence of Simulated Saturation Diving on Electroencephalogram of Human at Different Depths

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This paper report on the changes of the EEG of human during simulated saturation diving at different depths with different mixture gases. The results of this experiment show that there appears diffusion slow waves (θ and δ waves). The changes of EEG at 50 m depth were more obvious than those at 36.5 m. With the prolonging of time under high pressure, the EEG had some improvements, for instance, the slow waves decreased and α waves increased. There was a certain relationship between these changes and the physiological symptoms. The chief factor of the changes of EEG is nitrogen narcosis for the oxgen-nitrogen diving. In addition, carbon dioxide retention under high pressure is also a factor because CO_2 rebreathing and hyperventilation (i.e. high and low CO_2 in body) aggravated the changes of EEG. The main changes of the EEG during helium-oxygen exposure at 302 m depth were the increase of θ waves and δ waves, the decrease in α rhythm and the decline of amplitude of α waves. Increased θ index and decreased α index could be seen at the depth of 302 m. Under any of the above-mentioned high pressure condition, when slow waves were taken as characters in the background in abnormal changes of the EEG, the EEG could be temporarily improved by photic stimulation, that is slow waves disappeared and α waves reappeared. When photic stimulation was over, α waves disappeared and slow waves reappeared. It was indicated that abnormal changes of the EEG under high pressure were a kind of temporary and reversible changes of the brain function.

Key words: EEG, Hyperbaric Environment, Nitrogen Narcosis Hypercapnia, Hyperventilation, High Pressure Nervous Syndrome

INTRODUCTION

In order to understand and make full use of the ocean for bringing benefit to makind, we must go to the undersea view the freedom of activity in it. Therefore, the saturation dive is program which is developed quickly and is paid great attention. The results in the last twenty years have been applied in the oceangraphic scientific survey and the exploration of ocean resource, especially in the exploitation of ocean oil. The main characteristic of this diving method is to ensure the long duration, great depth, high work efficiency, and safety, For these reasons, in the exploitation of ocean resources the underwater operations of 80-90 percent are undertaken by using saturation diving technique.

In the process of submerging the ocean, certain limits are the high pressure. The primary factor for the human physiological fouction is the composition of the gas mixtures, for instance, ultra high partial pressure of oxygen could result in oxygen toxicity, nitrogen could cause nitrogen narcosis and high helium could lead to "high pressure nervous syndrome". For this reason,the gas mixtures of different contents must be used for different depths. Compressed air saturation diving could be used at depths of less than 20 m, nitrogen-oxygen could be usually used at 20-50m. Helium-oxygen or helium-nitrogen-oxygen mixtures could be used at the

depths of more than 50 m. The human cerebrum is one of the most sensitive organs to the effects of high pressure oxygen, nitrogen and high pressure helium which could at first cause the hindrance of encephalic function. The hindrance of encephalon causes serious consequence at the depth, meanwhile the changes in encephalic functions reflected on the EEG are important signs of oxygen toxicity, nitrogen narcosis and "high pressure nervous syndrome" [1-5] This paper reports on analytic results of the changes of the EEG when the human bodies are under saturation exposures at different high pressure.

SUBJECTS AND METHODS

This experiment was carried out through three experiments: (1) Simulated nitrogen-oxygen saturation diving at the depth of 36.5 m for 26 days, (2) simulated nitrogen-oxygen saturation diving at the depth of 50 m for 5 day, and (3) Simulated helium-oxygen saturation diving at the depth of 302 m for 43.5 hr. The subjects to be tested in the first and second experiments were 6 male professional divers, their ages were from 19 to 31 years old, and their diving careers were from 2 to 14 years. Their past diving and simulated diving depths reached 45-200 m. There were no wounds on their heads and no epilepsia histories from their parents. All subjects had undergone a physical examination and strict training: the sensitivity to oxygen, bearing increasing pressure and the physiological function. The subjects were all in keeping with the standard demands of the Navy divers of China. The experiments were undertaken in the experimental chamber for saturation diving at the Institute of Navy Medicine. The main ambient parameters are as below: the saturation pressures in the chamber were 4.65ATA (36.5m) and 6.0ATA (50 m) respectively while the partial pressure of oxygen was 0.31 -0.32ATA, the rest was nitrogen with partial pressures of 4.34 ATA and 5.68 ATA respectively. The ambient temperature in chamber was maintained at about 26℃, and the relative humidity was about 70%. During the experiment, the partial pressure of

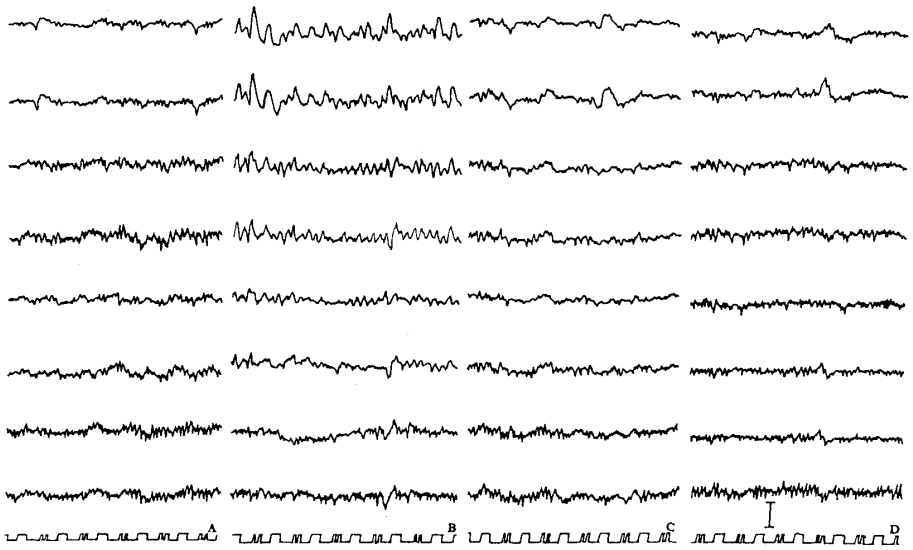

Fig. 1. The EEGs under the conditions of a long exposure to nitrogen and oxygen ambient (at 36.5 m depth)
A. The controls of the EEG. B. 3rd day after compressing to 36.5 m. C. After 15-day stay at 36.5 m. D. After 26 day-stay at 36.5m. The traces from top to bottom are parts of the EEG of right and left lobi frontalis, lobi parietalis, lobi temporalis and lobi occipitalis, and of the time label (one second per measure). The adjusted voltage was 100 μv. It is the same with Fig. 2 and Fig .4.

oxygen was first adjusted to 0.31 ATA, then the chamber was compressed to the saturated pressure with pure nitrogen during 10 min and 13 min respectively. According to the schedule, the subjects stayed at the depth and performed simulated operations with the mechanical work of 600 kg.m/min. At 36.5m depth, the chamber was decompressed at a uniform rate to the surface.

The three subjects who participated in third experiment were well trained and healthy diviers. They had taken part in a number of deep saturation diving. They were 24, 24 and 29 years old, respectively. Two of them had participated in the experiment of deep sea saturation diving at a depth of 200m in France. This experiment was carried out in the compression chamber of saturation dive installed on "South Sea II" platform in the Shan Ya port in China. The procedure of compression was described below: The chamber was compressed to 5 m depth by air in order to maintain partial pressure of oxygen at 0.30 ATA. Then pure helium was used to increase the chamber pressure to the depth of 100 m for 1 hr, and then the compression was conducted to a depth of 302 m at the rate of 12 m/hr. After half-an-hour stay at 100 m depth then for 11 hr at 200 m, we tested the physiological functions for two hours at 200 m depth. The total time of compression was 29.5 hr. Three systematic tests of physiological function were undertaken and two tests of medium physical loads were taken during or at end of stay at 302m for 43.5 hr. The decompression was performed from 302 m to 293 m at the rate of 20 min/m, from 293 m to 61 m at 33 min/m, from 61 to 30 m at 40 min/m, from 30 m to 15 m at 50 min/m and from 15m to surface at 67 min/m. The total time of decompression was 239 hr, which included staying time with sleep every night. The monopolar EEG was recorded using the method of 8 recording electrodes placed symmetrically at lobi frontalis, parietalis, temporalis and occipitalis. Indifferent electrodes were placed at ear lobi. The EEGs of the first and second experiments were recorded by using electroencephalograph Model ND-82, made in China. The EEGs of the third experiment were recorded by using electroencephalograph Model 5109, made in Japan (NIHON-KODEN). The frequency spectrum analyzer Model MAF-5 was used to analyse the integral value of 10 seconds for δ, θ, α, β_1 and β_2 waves of left parietalis and occipitalis. When the EEG was tracing, the subjects, who were

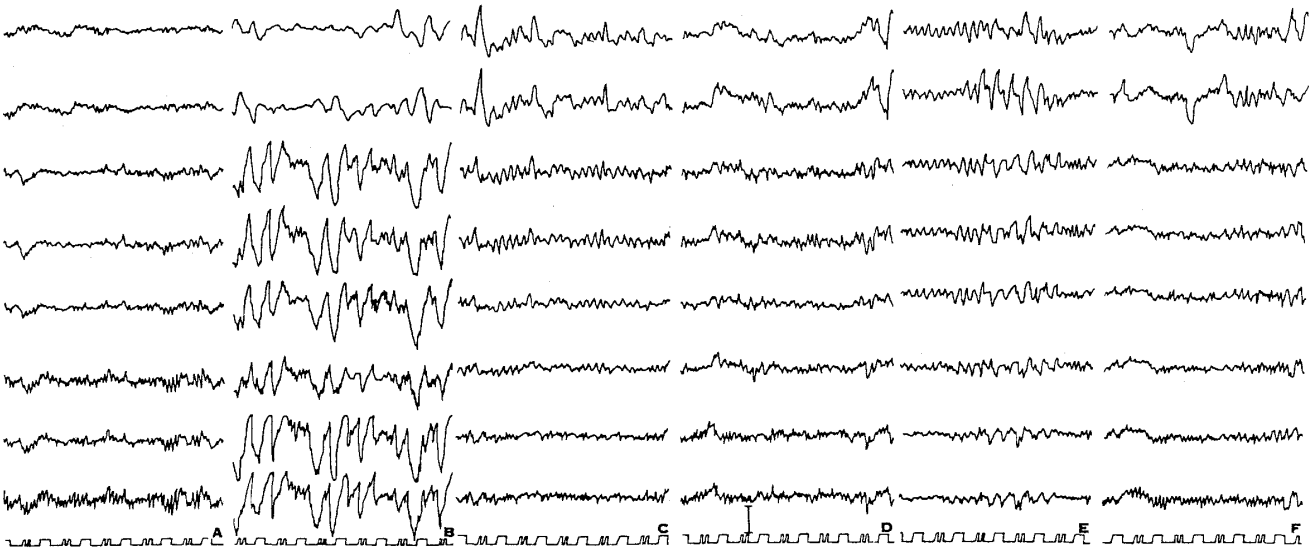

Fig. 2. The effect of hyperventilation on the EEG during saturation exposure to 36.5 m depth and 50 m depth
A. The control of the EEG before compression. B. During hyperventilation before compression. C. 3rd day after compression to 36.5 m. D. During hyperventilation after 3 days of exposure to 36.5 m. E. On the 4th day after compression to 50 m. F. During hyperventilation on the 4th day after compression to 50 m.

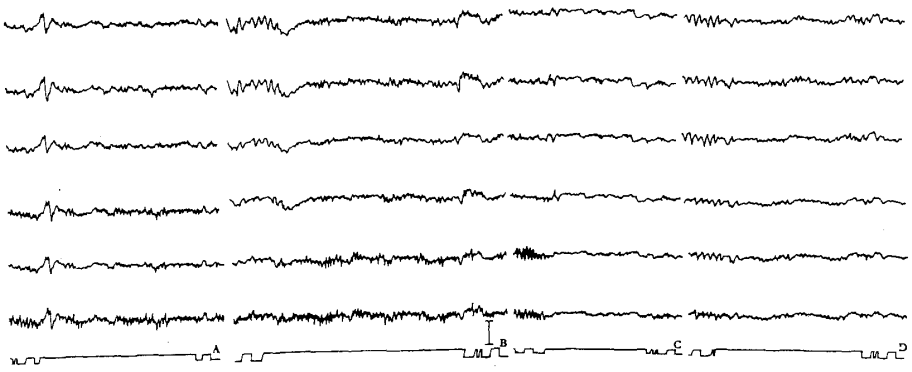

Fig. 3. The effects of the photic stimulation on EEG during saturation exposure to 36.5 m depth and 50 m depth
A. The EEG during photic stimulation compression. B. After 3 days of compression to the depth of 36.5 m. C. The controls made during photic stimulation before compression. D. During photic stimulation after 4 days stay at 50 m. Their recorded curves are parts of the EEG of right and left lobi frontalis, lobi temporalis and lobi occipitalis from top to bottom, and the time label (one second per measure). The sign stand for the photic stmulation when the label stopped. The adjusted voltage was 100 μv.

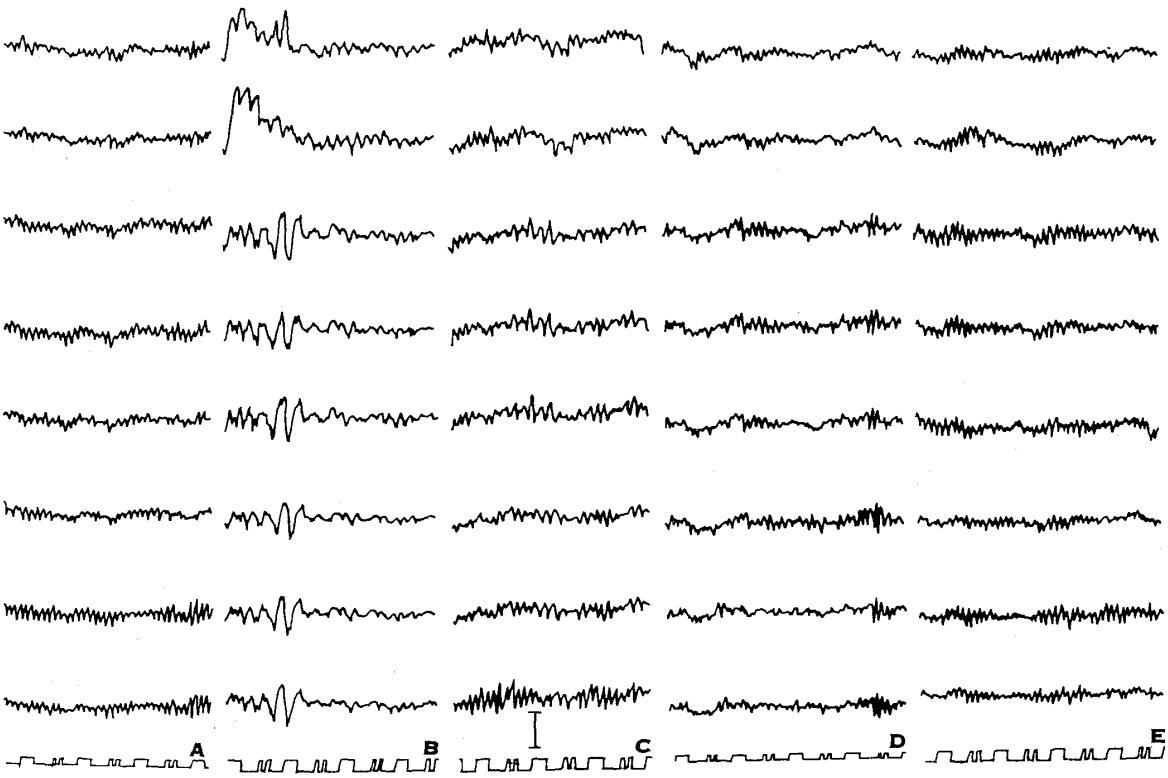

Fig. 4. The EEG under condition of saturations N_2-O_2 exposure to 50 m depth.
A. The controls of the EEG. B. On the 4th day after compressing to 50 m. C. After decompressing to 44 m. D. After decompressing to 19 m.E. On the 2nd day after decompression to atmospheric pressure.

at rest, conscious and eyes closed, sat in a small dark room. The photic stimulation was released to the subjects for 10 sec. Finally, in the second experiment the EEG were recorded during hyperventilation and rebreathing by CO_2 for three min and at every minute partial pressure of CO_2 of the end tidal gas was measured.

RESULTS

(I) The EEG changes during saturation exposure to high pressure condition (equivalent to 36.5m depth)

Under normal pressure, the contraction of cerebral blood vessels caused by hyperventilation resulted in the hypoxia of cerebral tissue, leading to the increase in the slow wave component on EEG. When the subjects were exposed to the high pressure saturation at the depth of 36.5m, in air excurison diving from this depth to 60, 70 or 75 m depth, hyperventilation would not cause the increase in the slow wave component. On the contrary, it would cause the decrease of the index in the slow waves, that is, hyperventilation under high pressure would cause the improvement of the EEGs. For instance, there were θ waves as prevailing frequancy on a diver's EEG at 36.5 m. However, as θ waves decreased, α waves increased. Much improvement had been made temporarily on his EEGs at the time of hyperventilation.

Under the atmospheric pressure, the photic stimulation always caused decrease in amplitude of α waves, or its rhythm disappeared and was replaced by β waves. When the subjects stayed at 36.5m depth for long time, the photic stimulation could

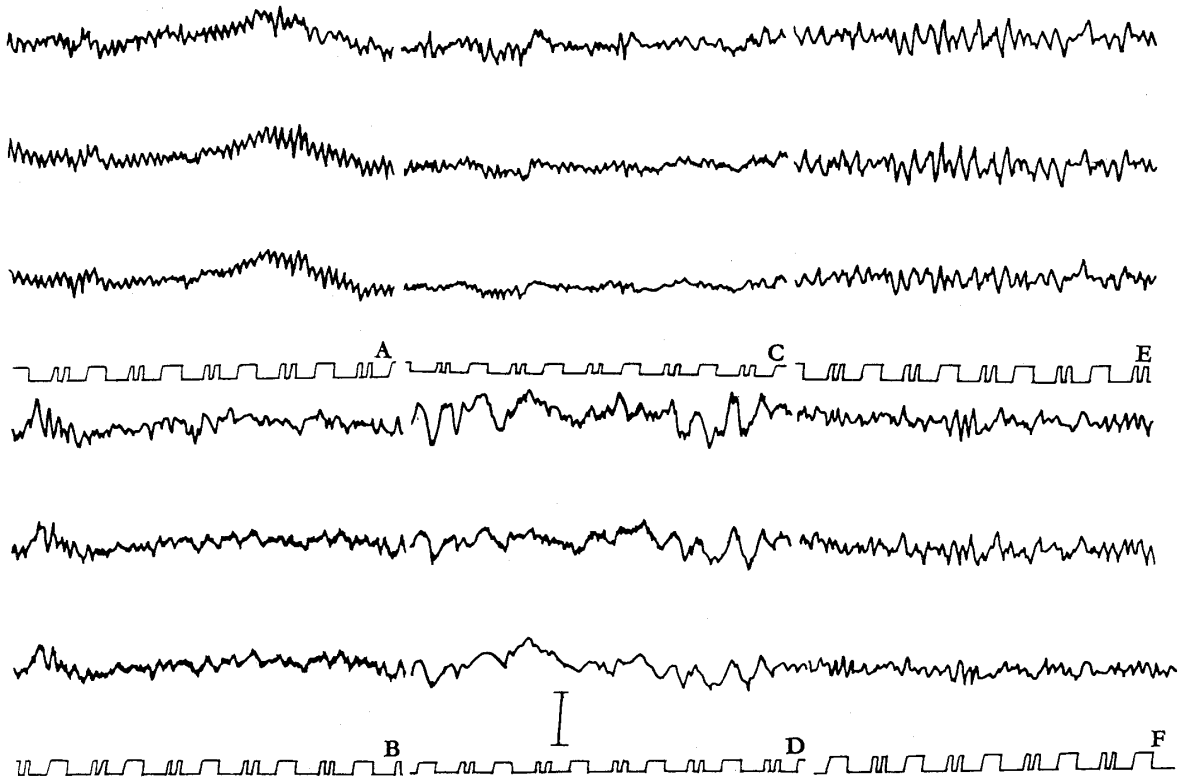

Fig. 5. The effect of high CO_2 on the EEG under normal and high pressure.
A. The control of the EEG under atmospheric pressure. B.At the end of 3rd min of CO_2 rebreathing under atmospheric pressure. C. On the 5th day after compression to 50 m. D. At the end of 3rd minute of CO_2 rebreathing at 50 m. E. On the 4th day of exposure at 50 m. F. Under the condition of low CO_2 at 50 m. The traces from top to bottom are the EEG of left lobi frontalis, left lobi parietalis, and lobi occipitalis. The time label indicated one second per measure. The adjusted voltage was 100 μv.

Table 1. Relation between changes of EEG and end tidal Pco_2 for 6 subjects

		prepressure				five days at 50 m			
breathing time with 0.069 ATA CO_2 mixture (min)		0	1	2	3	0	1	2	3
end tidal Pco_2 (mmHg)		39	49	57	62	53	55	59	67
EEG changes	α index (%)	66.2	55.1	43.6	37.9	52.8	44.3	32.1	28.3
	slow wave index (%)	15.3	29.1	33.2	48.7	23.7	28.8	47.3	53.0

cause another kind of response. When θ waves prevailed, the slow waves had temporary disappearance and α waves had short-lived restoration by the photic stimulation. After the stimulation, α waves disappeared and the slow waves reappeared (See Fig. 3).

(II) Changes of the EEG during nitrogen-oxygen saturation exposure to high pressure condition at 50 m depth

Changes of the EEG were more obvious than those of the first experiment because of the deeper condition and higher partial pressure of nitrogen. The main change was the lowering of α waves on each location of cerebrum. The amplitude of α waves on the first and fourth day at 50 m depth decreased significantly compared with control level ($P<0.01$). The θ waves slowed with their amplitude rising obviously and δ waves of more than 100 μv appeared on the EEG of some subjects. In decompression, the α waves increased gradually and it returned to control level after decompression to the normal pressure (See Fig. 4).

At 50 m depth photic stimulation might cause the disappearance of slow waves and the appearance of α waves.

During hypocapnia by hyperventilation, α waves increased but slow waves decreased. This shows that there might be some relationship between the appearance of abnormal rhythm of slow waves and the level of carbon dioxode in human under high pressure, because slow wave component decreased while hyperventilation resulted in the lowering of the level of carbon dioxide.

In this way, the CO_2 rebreathing was conducted at 50 m depth at which maximal Pco_2 was 0.069 ATA. At the end of the 3rd min rebreathing, the partial pressure of carbon dioxide of end tidal gas rose to 66.8 mmHg. The index of α waves was lower, but the index of slow waves increased with the rise in partial pressure of carbon dioxide, and the diffused θ waves appeared in great quantities during the rebreathing. The EEG changes were more obvious than those affected by unitary high-density carbon dioxide or high pressure alone (Fig. 5, Table 1).

(III) Changes of the EEG during helium-oxygen saturation diving at the depth of 302 m

When the subjects were exposed to the helium-oxygen condition at the depth of 302 m, every kind of parameter on the EEG changed with the rising of pressure. The most important changes were the lowering of α wave amplitude, the slowing of frequency and the decreased α wave index, the increaned θ and δ activities, the increase of θ and δ waves and the inhibition of the total EEG activities. For subject No. 1, at the beginning of his stay at 302 m depth, the percentage of integral value of α wave at lobi occipitalis was 47.8% of his cotrol at atmospheric pressure. It decreased significantly to 31.3, 33.7 and 28.8% during exposed time of half an hour, 18 hr and 42 hr, respectively at 302 m. As for subject No. 2, the percentage of integral values decreased to 34.7%, 35.1% and 37.5% from the control level 51. 0% in the same period at 302 m. When decompression began, the α wave component rose again gradually and restored to the normal level after decompressing to surface. Under high pressure, while α

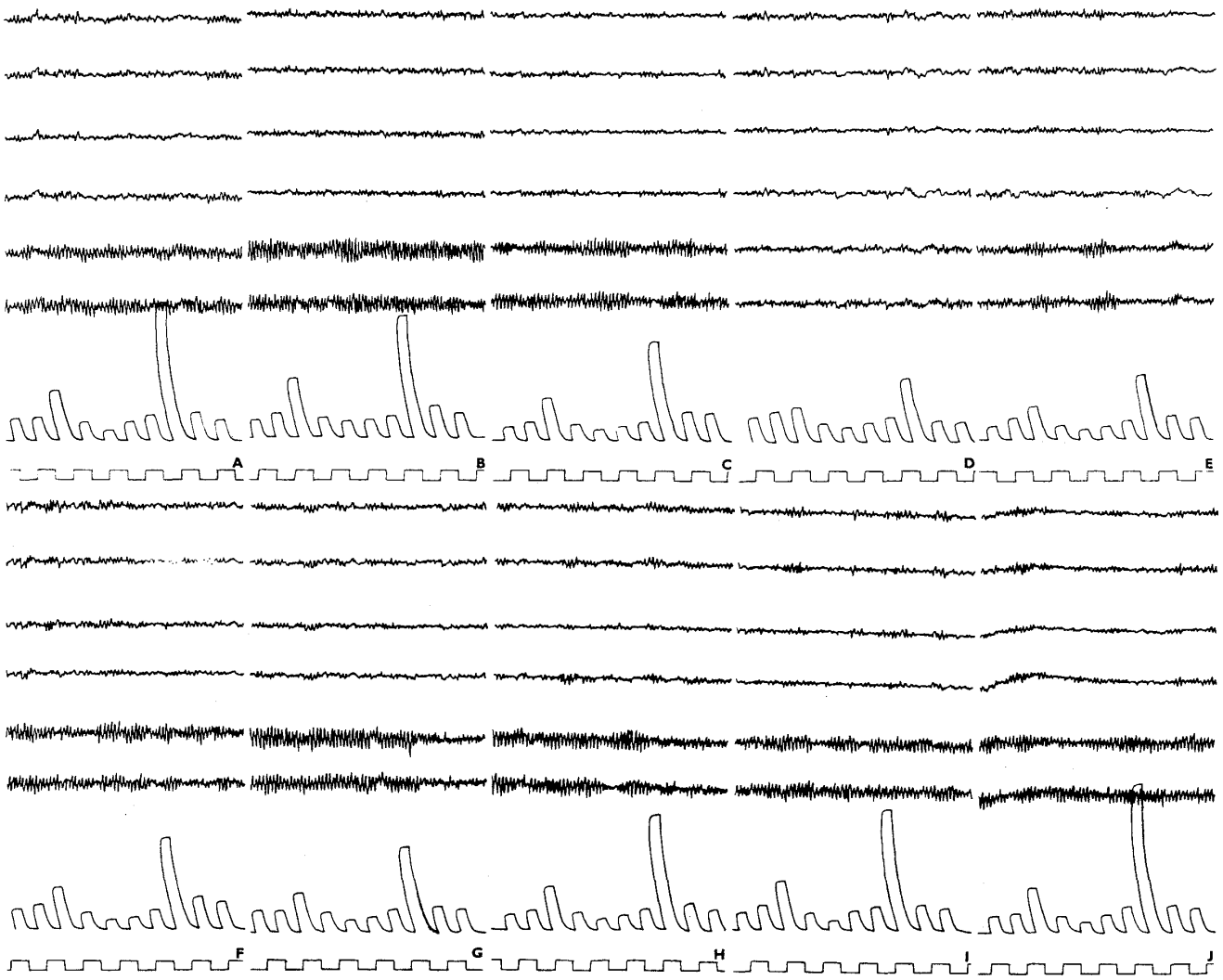

Fig. 6. The integral value of related component of the EEG in 10 seconds under helium-oxygen at various depths A. The controls at normal pressure. B. After compression to 200 m. C. At the end of compression to 302 m. D. After 18-hour stay at 302 m. E. After 42-hour stay at 302 m. F. At 250 m depth during decompression. G -I, At 200, 150, 100 m depth during decompression respectively. H. After decompression to 0 m. The recorded traces from top to bottom are parts of the EEG of right and left lobi centre, lobi frontalis and lobi occipitalis. Frequency analysis from left to right is the integral valus of δ, θ, α, β_1 and β_2 in 10 seconds in left lobi centre and left lobi occipitalis. The time label indicated one secod per squart.

activity decreased, its amplitude was lowered obviously, and it almost decreased by half at 302 m. The lowering of the amplitude at lobi occipitalis was the most obvious.

During exposure to 302 m depth, component of θ waves increased obviously at every lobe. The changes of EEG at the central lobi were the most obivous. These lobi were taken as an example. The percentage of the integral value of θ waves of subject No. 1 was 21.6% at atmospheric condition, as it increased respectively to 26.9% and 29.4% when the pressure was increased to 200 m and 250 m. At the beginning of 302 m depth, for less than half-an-hour, 18hr and 42hr, it increased respectively to 29.7, 36.2 and 29.6%. The δ wave also increased correspondingly. The change of θ component is similar to subject No. 2 and No. 3. During decompression their θ waves decreased gradually. After returning to "sea level", their θ waves were resumed to the normal level (Fig. 6).

Under atmospheric air condition, the α waves seen as a background of the EEG was inhibited by

photic stimulation, its amplitude was lowered or even replaced by β rhythm. However, at 302 m depth, slow waves were temporarily decreased and α waves were increased by photic stimulation, while slow waves were prevailing. As soon as photic stimulation was over, slow waves appeared diffused again. This proves that photic stimulation could result in temporary improvement at the great depth of 302 m, and the hindrance to the function of the cerebrum is reversible.

DISCUSSION

When human are exposed to hyperbaric environment for a long time, the different components of gas mixture which is breathed in, is necessary at different depths, and this affects the encephalic functions. In nitrogen-oxygen saturation diving at depths of 36.5 m and 50 m, the partial pressure of oxygen remained at the level of 0.30 ATA and had no obvious effects on EEG. Therefore, we can say that changes of EEG are due to the effect of high pressure of nitrogen. It is known that high pressure nitrogen has the narcotic effect as alcohol, but the degree of narcosis is directly proportional to partial pressure of nitrogen. The present experiments proved that the symptoms of nitrogen narcosis and the corresponding changes of EEG at the depth of 50 m were more obvious than those at depth of 36.5 m. This demonstrates that there might be some relation between the partial pressure of nitrogen and the changes of the EEG.[2,3]

For 26 days exposure to nitrogen-oxygen high pressure condition (the depth of 36.5 m), it was never seen in any report about research of the EEG. Our research shows that α waves decreased with the intial period of exposure and the above-mentioned codition (10-15 days). The results point out that the changes of the parameters of EEGs have a corresponding bearing on the adaptation to high pressure nitrogen ambience. In fact, six subjects had slight headache, excitement euphoria, bad appetite and lip numbness and sometimes were too excited to sleep, together with dull response, unsteady steps and the lowering of the work ability at the initial period of exposure. The subjecs adapted gradually to the new surrounding, and the symptoms which appeared at the initial period disappeared gradually with the prolongation of exposure. This means that there exists a process of adaptation to the high pressure nitrogen surroundings and also perform effective excursions at the depth of 75 m and complete underwater operation of medium strength. Experiment of Makay proved that the changes of the EEG together with other reflection could be improved with the prolonging of exposure duration[4].

Some scholars who research the nitrogen narcosis consider that there might be other causes affecting the changes on the nervous function when man is in the hyperbaric nitrogen and oxygen, besides nitrogen itself. Bean[5] once mentioned that carbon dioxide retention was the cause of nitrogen narcosis. The research made by Hesser[6] and Case [7] proved further that the increase of thepartial pressure of carbon dioxide in the human body at high pressure exposure could add effect of nitrogen narcosis and raise its degree. It was observed in our experiments that the partial pressure of carbon dioxide in the end tidal gas rose when the human body was exposed to 50 m depth. Hyperventilation resulted in the reduction of Pco_2 in human body and improved the EEG which was diffused by prevailing slow waves. This proved indirectly that the reduction of carbon dioxide caused by hyperventilation could relax the retention of carbon dioxide in body and thus result in the improvement of the EEG. We have drawn an inference from that there might be a relationship between the abnormality of the EEG and retention of the carbon dioxide in human.

Under nitrogen-oxygen saturation at 50 m the human encephalic function experiences double effect of high pressure nitrogen and carbon dioxide while people are breathing the high partial pressure of carbon dioxide repeatedly, and changes of electroencephalogic activities are much more obvi-

ous than that caused by any single factor. Under atmospheric pressure where carbon dioxide is maintained, a subject breathed repeatedly the mixture gas of high partial pressure of carbon dioxide for three minutes, and there was an increase of slow waves, but it took frequency as the dominant character. Under high pressure at 50 m depth, however, a great amount of δ waves appeared on EEG when subjects breathed mixture of carbon dioxide. This demostrates that nitrogen and high carbon dioxide at high pressure have a co-operative effect on the EEG. In fact, when the body in exposed to high pressure, the respiratory resistance is increased by high density ventilatory gas, and the insufficiency of ventilation and breath results in carbon dioxide retention in body, and the rising of carbon dioxide level in the tissues to changes of the EEG more obvious. This points out further that carbon dioxide retention under high pressure is the cause of nitrogen narcosis. Therefore, trying to increase carbon dioxide is an important measure to prevent nitrogen narcosis; it is of primary importance for divers to make an operation underwater safe.

At present, it is the most important method for man to dive into the deep sea to make scientific survey and resource exploration with a mixture of helium-oxygen or mixture of helium-nitrogen-oxygen in deep water diving activities. Under simulated deep sea cnditions, men have entered the depth of 686 m.[8] However, if the compression rate is too fast and the depth is too deep (more than 150 m), hindrances of the human action and physiological functions will be caused, which is mainly shown in myoclonus of hand, arm, leg, foot and face, convulsion, nausea, vomiting, dizziness, bad appetite, great quantity of θ waves on the EEG, together with fatigue and lowering of performance; all these are called "high pressure nervous syndrome", which is a limit factor of effective activity for man in the deep sea, Thus, the research on the prevention of HPNS or lessening of its degree is one of the main projects of underwater physiology and medical research.[9,10] The data accumulated by our predecessors have shown that a series of measures such as reducing compression rate, setting up stops during compression, which can help man to adapt himself to the high pressure condition, choosing reasonably and training them painstakingly, and choosing appropriate breathing gas mixture (for example, add nitrogen of 5-8% to mixture of helium-oxygen) could control HPNS effectively. In our experiments, we chose slow compression rate with stops (at 100 m and 200 m). 4% nitrogen was kept in breathing gas mixtures and the divers who had several years of diving experience were chosen. So until such a great depth as 302 m, the clinical symptoms of high pressure nervous syndrome did not appear.

The abnormal changes of the EEG under hyperbaric helium-oxygen conditions are the objective index of the symptom of the HPNS. For this reason during saturation diving at great depths, the human EEG are usually recorded in order to estimate the chosen schedules of compression and distribution ration of breathing the gas mixture and observe the health state of the divers and their adaptation ability to high pressure.[11] The results of our research show that during the exposure to more than 200 m and at 302 m depth, the most obvious changes of the EEG were a decrease in amplitude of α waves, an increase of θ and δ components and these changes of the EEGs were similar to those of sleep stage I. According to Bennett,[12] as regard to high pressure nervous syndrome, not only an increase of θ activity but also blocking of entire electroencephalographic activities was represented on the EEG. He also said that the former had something to do with the rate of compression but the latter had something to do with ambient absolute pressure during exposure. There has been no unified opinion about the problem as to why changes of the EEGs under high pressure appear. The most recent opinion is that the changes of the EEGs are caused by the helium gradient between the inside and outside of cell, because helium which entered the human body dissolves

quickly into the extracellular fluid but enters slowly into the intracellular fluid. Thus an obvious helium gradient between the inside and outside of the cells is formed at certain time which leads to the changes of the EEGs. When the time of the stay under high pressure is prolonged, the helium gradient between the inside and the outside of the cells becomes smaller and smaller, and the relative balance will be reached with the improvement of the EEG and symptoms are lightened. The human body gets new adaptation to the undersea surroundings.

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