EFFECT OF AN INCREASED ATMOSPHERIC PRESSURE ON NEUROMOTOR EXCITABILITY IN MAN

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In experiments on 10 subjects the reflex activity of spinal motoneurons and excitability of the neuromuscular system were investigated during exposure to a raised atmospheric pressure. An increase in pressure up to 6 atm caused a decrease in reflex excitability of the spinal motoneurons accompanied by increased activity of the neuromuscular system. Constant exposure for 18 min to an atmospheric pressure of 6 atm did not arrest the processes inducing changes in excitability of the spinal motoneurons or the neuromuscular system during brief exposure to the same pressure of 6 atm. A decrease in pressure to 2.2 atm during decompression caused no significant change in the excitability of the neuromotor system as recorded at the 18th min of the subject's stay in an atmosphere at a pressure of 6 atm.

Investigations have shown that during exposure to a hyperbaric atmosphere of air and of nitrogen and oxygen significant changes take place in human motor activity [3, 4, 8, 10, 14]. For instance, with elevation of the pressure the speed of typing is reduced [8, 10, 14, 16] and the latent period of the motor response to various afferent stimuli is increased [3, 4, 16, 19]. Disturbances of complex coordinated human actions are observed, causing changes in handwriting and in drawing [3, 6]. The severity of the changes in motor activity depends on the magnitude of the increased pressure and the duration of exposure to these conditions [2, 6]. Despite the considerable volume of facts indicating a disturbance of motor function under hyperbaric conditions, the neuronal mechanisms of these changes have been inadequately studied.

It was accordingly decided to investigate the reflex excitability of spinal motoneurons and the functional state of the neuromuscular system in man under hyperbaric conditions.

EXPERIMENTAL METHOD

The tests were carried out in a dry recompression chamber. Ten subjects (part-time divers) exposed regularly (once or twice a week) to an increased atmospheric pressure for a period of 1 year, took part in the investigation. During the experiment the subject lay on his back.

To assess reflex excitability of the spinal motoneurons, a method of monosynaptic testing (the H-reflex) was used [1, 5, 15]. The H-reflex was evoked by square electric pulses 1 msec in duration, applied to the tibial nerve in the popliteal fossa from a type ÉSU-1 high-frequency output transformer. The H-reflex was recorded by surface electrodes from the soleus muscle in one channel of a "Medicor" electromyograph. The peripheral motor response (M-response), obtained constantly from the medial head of the gastrocnemius muscle, was recorded on the other channel of the electromyograph.

The strength of nerve stimulation was chosen so that the amplitude of the control H-reflex was 40-50% of the maximal amplitude, whereas the amplitude of the control M-response with the same strength of stimulation was 20-25% of its maximal amplitude.

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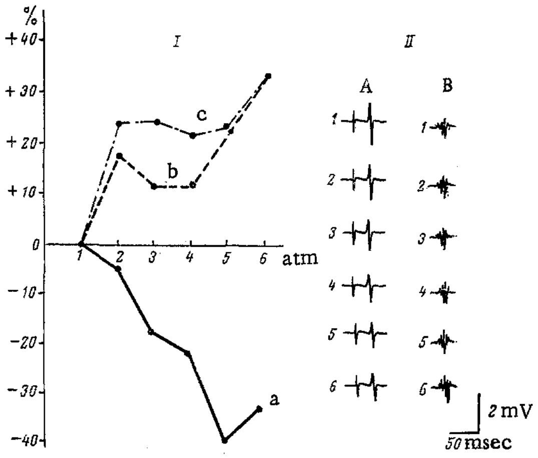

Fig. 1. Effect of elevation of atmospheric pressure on amplitude of evoked reflex and peripheral responses: I. Ordinate relative change in mean amplitude of control H-response (a), first components of M-response (b), and second components of M-response (c). Abscissa, pressure in atm. II. A) Reflex H- and B) peripheral M-responses during elevation of pressure to 6 atm. 1-6) Pressure in atm.

TABLE 1. Relative Change in Mean Amplitude (in percent) of Control H- and M-Responses during Elevation of the Atmospheric Pressure at the Rate of 1 atm in 3 min

Pressure (in atm)	H-response	M-response	
		I component	II component
		$\bar{x} \pm s$	
2	−4,7±3,3	+18,4±7,7	+24,1±10,1
3	−16,9±5,6	+12,0±4,0	+24,1±2,0
4	−21,8±6,9	+12,3±4,1	+22,1±7,3
5	−39,2±1,1	+23,4±7,8	+23,4±5,6
6	−32,1+9,0	+34,7±7,1	+34,4±7,2

The reflex H-response and peripheral M-response were evoked every 10 sec. At first, under ordinary atmospheric pressure, the control H- and M-responses were recorded for 5-10 min. After stabilization of the amplitude of the control H-response the pressure began to be raised at a relatively constant rate of 1 atm (excess atmosphere) in 3 min to 6 atm (absolute atmosphere). Throughout this time of elevation of pressure the evoked responses from the muscles were recorded. When the pressure reached 6 atm the compression was stopped and the subject was exposed to this pressure for 18 min. In this case, the H- and M-responses were recorded after exposure to this pressure for 5, 10, and 18 min. At the end of this period of exposure of the subject in the recompression chamber to a pressure of 6 atm, decompression was carried out. During decompression the amplitudes of the reflex and motor responses were recorded at the first stop at a pressure of 2.2 atm, 6 min after the beginning of decompression.

The relative change in the mean amplitude of the control H-response and peripheral M-response was determined from 6-20 measurements.

EXPERIMENTAL RESULTS AND DISCUSSION

Elevation of the atmospheric pressure causes a decrease in the reflex excitability of spinal motoneurons. It will be clear from the results given in Table 1 and Fig. 1 that with an increase in pressure there is a decrease in amplitude of the control H-response. The first significant decrease in the H-response was observed when the pressure was raised to 3 atm ($P < 0.05$). The greatest decrease in the H-response occurred at 5 atm. On a further increase in pressure to 6 atm no significant changes took place in the amplitude of the H-response.

The recorded amplitude of the 2-component M-response was increased during elevation of the pressure (Table 1; Fig. 2). A significant increase in amplitude of both components of the control M-response was observed as early as at 2 atm ($P < 0.05$). At 6 atm the amplitude of the M-response increased to its maximum. During elevation of the pressure from 2 to 5 atm the control M-response showed no significant change.

TABLE 2. Relative Change in Mean Amplitude of Control H- and M-Responses Depending on Time of Exposure to an Atmospheric Pressure of 6 atm

Pressure and time of exposure	H-response	M-response	
		I component	II component
		$\bar{x} \pm s$	
6 atm, 5th minute	—30,6±10,1	+44,6±11,3	+55,1±17,9
6 atm, 10th minute . . .	—29,6±9,5	+54,1±12,1	+68,9±17,0
6 atm, 18th minute . . .	—32,8±11,1	+56,0±14,1	+77,5±20,0
Decompression to 2.2 atm 6 min	—27,1±10,2	+56,8±20,4	+84,4±21,0

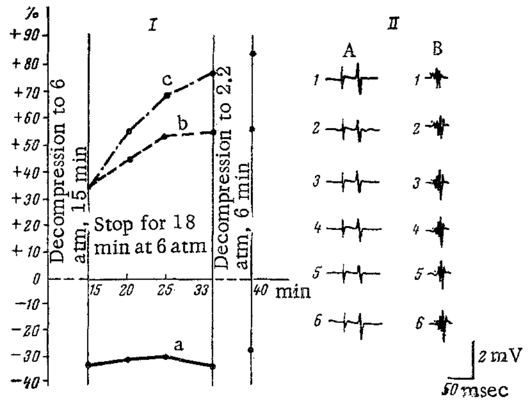

Fig. 2. Effect of exposure for 18 min to an increased atmospheric pressure (6 atm) on amplitude of evoked reflex and peripheral responses. I. Ordinate, legend as in Fig. 1; abscissa, time of exposure to increased pressure. II. Reflex H- and peripheral M-responses (legend as before): 1) control amplitude of evoked responses; 2-5) evoked responses at different times of action of increased pressure (6 atm); 6) evoked responses at first stop of decompression.

The mean results of the relative change in amplitude of the reflex and motor responses during constant exposure to a pressure of 6 atm are shown in Table 2 and Fig. 2. After elevation of the pressure, as already mentioned, the H-response was reduced and during 18 min of exposure to a pressure of 6 atm it showed no significant change.

The amplitude of the peripheral M-response was constantly increased during the 18 min of the subject's exposure to a pressure of 6 atm. For instance, the increase in amplitude of component II of the M-response was significantly increased by 23.0 ± 5.0% ($P < 0.05$) at the 18th min of the subject's exposure to this pressure by comparison with its increase at the 5th min of exposure. An increase in the growth of component I of the M-response also was observed. The increase in amplitude of component I of the M-response at the 18th min was 12.0 ± 3.0% greater than at the 5th min of the subject's exposure to a pressure of 6 atm.

The amplitudes of the H- and M-responses at the first stop during decompression at a pressure of 2.2 atm were not significantly different from their amplitudes at the 18th min of the subject's exposure to a pressure of 6 atm.

The decrease in amplitude of the H-response during elevation of the atmospheric pressure thus indicates a decrease in reflex excitability of the spinal motoneurons in man, in agreement with the results of experiments on decerebrate and spinal animals [10, 13], in which the reflex activity of the spinal cord was depressed during exposure to a hyperbaric atmosphere of nitrogen and oxygen.

Depression of reflex excitability of the spinal motoneurons was presumably due to increased activity of various inhibitory systems of the segmental apparatus [11, 12, 13, 18]. Suprasegmental regions of the brain and spinal cord may also, perhaps, participate in the change in functional state of the spinal motor centers, as shown by the results of Bennett's investigations, which demonstrated a decrease in amplitude of the evoked potentials of the reticular formation under hyperbaric conditions [9].

Simultaneously with the decrease in the control H-response during elevation of the pressure the amplitude of the peripheral M-response was increased, indicating activation of a larger number of muscle fibers [13, 17].

A change in the amplitude of the reflex and peripheral responses was observed from the moment that the pressure began to rise. During a halt in compression, at the 18th min of the subject's exposure to an atmospheric pressure of 6 atm, the amplitude of the M-response continued to rise while the amplitude of the H-response did not change significantly. This suggests that the processes of the change taking place at the spinal and peripheral levels of the neuromotor system do not cease during exposure to a constant pressure and, consequently, they are unconnected with the pressure drop effect. During decompression, at the first stop the amplitude of the H-response and M-responses did not differ significantly from the amplitudes of the reflex and motor responses at the 18th min of the subject's exposure to a pressure of 6 atm. Presumably, therefore, the excitability of the neuromotor system was still not restored when the pressure had fallen to 2.2 atm (the first stop during decompression).

The changes in excitability both of the motor centers of the spinal cord and of the neuromuscular apparatus recorded during these experiments may perhaps be linked ultimately with disturbances of human motor function.

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