PHYSIOLOGICAL HUMAN REACTIONS TO ACCELERATION ALONG THE VERTEBROSTERNAL AXIS.

CHANGES IN THE SYSTEM OF EXTERNAL RESPIRATION

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Several studies have been made of disturbances of the external respiratory function in persons exposed to the action of acceleration transversely to the body [1, 2]. In the present paper we describe the results of a study of the functional state of the human external respiratory system uring the action of accelerations directed along the vertebrosternal axis and a magnitude of 15 g. The basic experiments were carried out during the action of acceleration at an angle of 65° to the longitudinal axis of the human body. Altogether 128 experiments were conducted on 31 persons.

In most experiments, before the subjects were rotated on a centrifuge, they were instructed in the most rational mode of respiration to adopt, enabling them to reduce their respiration rate and increase the depth of respiration considerably. Again, in most experiments, the duration of exposure to acceleration was the value to be sought, and was determined by the subjects themselves in accordance with the critical state in which any further prolongation of their stay in the conditions of exposure to acceleration became impossible. As a rule, this point was also confirmed by a number of objective findings. The principal parameters studied in the experiments with accelerations of up to 12 g were analyzed statistically, and the significance of the results determined (Fig. 1).

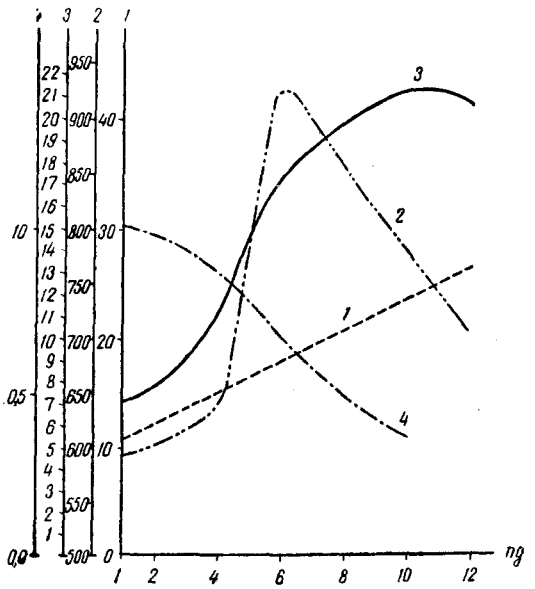

Fig. 1. Mean values of the principal indices of the external respiratory system during exposure to accelerations of a magnitude of up to 12 g. 1) Respiration rate for a standard deviation S=2.3; 2) respiratory volume (in ml) at S=147; 3) MIV (in liters) at S=1.35; 4) VC (VC during experiment/VC before experiment)

It was found that the respiration rate was higher the greater the acceleration, and its average value at 12 g was 29.4/min. The gradient of the increase in this index (grad. $= \frac{\Delta \text{ of respiration rate}}{\Delta g}$) in these conditions is constant and equals 2.8.

The depth of respiration (inspiratory volume) increased with accelerations of up to 6 g, and then fell progressively. For instance, immediately before exposure (in the cabin of the centrifuge with the harness fixed) the inspiratory volume had an average volume of 581 ml, and at 4 g it was 637 ml, at 6 g – 923 ml, at 8 g – 834 ml, at 10 g – 788 ml, and at 12 g – 704 ml. At 14-15 g the inspiratory volume was back within the initial range of values, and sometimes considerably less (in one or two cases it fell to 80 ml).

The minute inspiratory volume (MIV), which is essentially an integral characteristic of the system under study, changed with increasing acceleration up to 10 g in accordance with a complex law, giving an approximately S-shaped curve with a knee at a point corresponding to an acceleration of 5 g. Subsequently this function was observed to diminish. The same type of S-shaped curve,

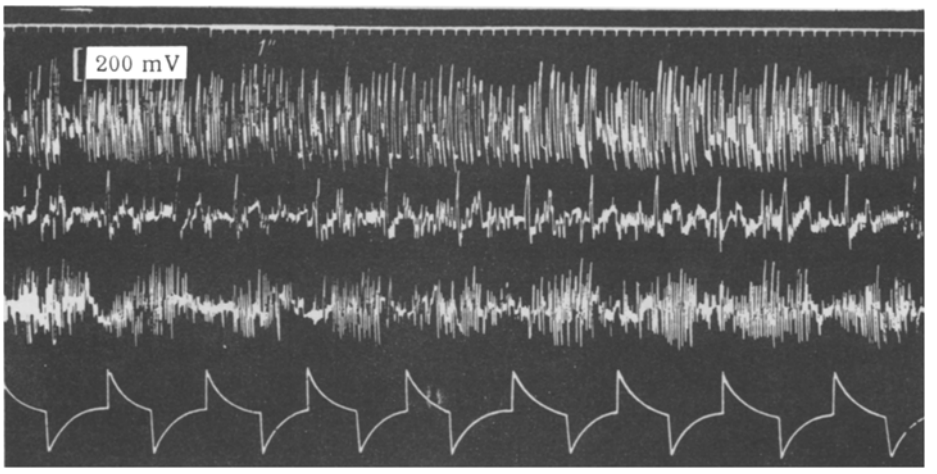

Fig. 2. EMG of various groups of skeletal muscles during exposure to acceleration of 8 g with marked increase in the activity of the EMG of the sterno-cleido-mastoid muscle in the inspiratory phase. Significance of the curves (from above down): time marker; EMG of posterior cervical group of muscles; EMG of quadriceps femoris muscle; EMG of sterno-cleido-mastoid muscle respiration rate (contact pick-up).

but as a mirror image of the MIV curve, described the changes in the vital capacity of the lungs (VC). The higher the acceleration the lower the VC, and the steepest fall in this index also corresponded to an acceleration of 5 g. To give a more reliable estimate of the degree of the change in the VC, not only the absolute value of this index, but also the ratio between the VC during exposure to acceleration of the particular magnitude and the VC before exposure in the same experiment was studied. It is this ratio which is represented in the graph.

During the period of action of acceleration on man, an oxygen debt develops. The size of this debt was determined indirectly from the results of integration of the MIV curve during the period before and immediately after exposure (over equal intervals of time). For instance, in one subject the MIV during the first 5 min after exposure (in an experiment with an acceleration of 12 g) was equal to 63,700 ml, whereas before exposure the value of this index was 30,000 ml.

Inhalation of pure oxygen at atmospheric or a higher pressure was accompanied by a definite increase in the resistance of the subject to acceleration.

The character of the changes in the main indices of external respiration at accelerations of different magnitudes indicates the severe embarrassment caused to this system. For instance, although during acceleration of "low" and "average" magnitudes, in response to the increasing oxygen demand of the body, a sharp increase in the MIV took place as a result of both a quickening and a deepening of the respiratory movements, the latter undoubtedly being more useful in this respect, during acceleration of "high" magnitudes the gradient of the increase in the MIV fell as a result of a decrease in the amplitude of the respiratory movements.

Attention must be directed particularly, in this respect, to the character of the change in the VC as the index of the potential capacity of the system under study. The progressive fall in the VC with the increasing magnitude of acceleration speaks for itself.

The main cause of the lowering of the functional level of the system of "external respiration at high acceleration values," in our opinion, is interference with the excursion of the thorax in connection with the increasing forces of inertia. This is especially noticeable in cases in which the direction of the force of acceleration comes close to the direction of the excursion of the chest. In the case under consideration, this difficulty arises on account of the phase of inspiration, a fact which is confirmed by the electromyographic findings. For instance, the EMG of the muscles concerned in the act of respiration showed well defined volleys of increased activity during the period of action of acceleration, coinciding with the phase of inspiration (Fig. 2). There is no doubt that the increased excitability of the respiratory center and the increased activity of the principal and accessory muscles of respiration, directed towards overcoming this resistance, must play an important role in this compensatory reaction.

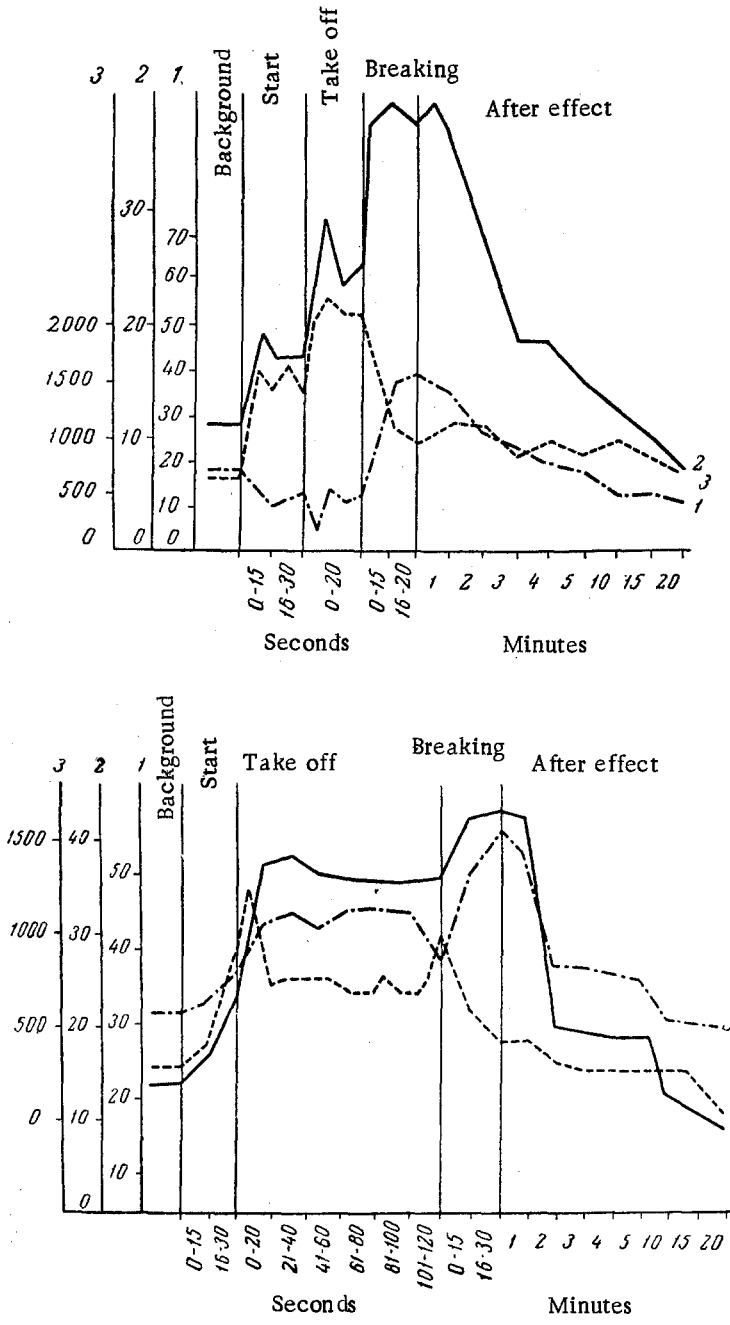

Fig. 3. Dynamics of the changes in the principal indices of the external respiratory system in subject P. in an experiment with acceleration of 8 g (a), and in subject E. in an experiment with acceleration of 14 g (b). 1) Inspiratory volume (in ml); 2) MIV (in liters); 3) respiration rate.

The character of the individual reactions of external respiration to accelerations of different magnitude also shows a definite pattern. With "average" acceleration (6-10 g), for instance, the development of the response reactions takes place in a series of stages. In relation to the function of external respiration, this was expressed as follows. The increase in acceleration (the "take-off" of the centrifuge) during the first period of the experiment was accompanied by a marked increase in the rate and depth of the respiratory movements and by disturbance of their rhythm. Subsequently, when in our opinion the body had reached a state of equilibrium with the external environment as a result of the development of adaptation reactions, the rate and depth of respiration remained adequately stabilized.

Near the time when the subject was given the signal to stop the centrifuge, another increase in the frequency of the respiratory movements could be observed, together with a decrease in the depth of respiration. The presence of three clearly demarcated stages in the development of the adaptation reactions apparently reflects the nature of the biological laws of adaptation of the organism to new external environmental conditions, and the last stage is indicative of the initial period of failure of the adaptive reactions as a result of overstrain. This view is confirmed by the reactions of the other functional systems which we investigated (see Report 1).

During acceleration of a "high" magnitude (12 g or more), when the duration of its action was limited to 10-40 sec, it was usually impossible to distinguish these stages in the development of the adaptive reactions. Only in individual cases, when the period of exposure was maximal (up to 60 sec), were these stages also apparent.

Examples illustrating the changes in the function of external respiration during accelerations of different magnitude are shown in Fig. 3. The action of forces of inertia, like that of other stressors, associated in turn with the specific and nonspecific activation of many other systems and organs (the central nervous system, the cardiovascular system, the external respiratory system, the skeletal muscles, the sympathetico-adrenal system, and so on), naturally increases the oxygen demand of the body. Meanwhile the specific effect of the acceleration severely impairs the function of the external respiratory system itself, primarily responsible for satisfying the oxygen requirements of the organism. It may be supposed that definite disturbances also take place at this time in the pulmonary circulation.

There can be no doubt that the combination of these various factors leads to extreme difficulty in the oxygen supplying system and to the development of an oxygen debt, which is not wiped out until early in the period of the after effect.

SUMMARY

An inquiry was made into the chief indices of external respiration in man during the action of accelerations (up to 15 g) directed along the dorso-thoracic axis at an angle of 65° to the back of the armchair. A definite regularity was established in the changes of the indices studied with various accelerations. Definite stages were noted in the development of individual reactions; this regularity reflected the essence of the general biological laws concerning body adaptation to new environmental conditions.

LITERATURE CITED

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2. T. A. Rogers and H. A. Smedal, Aerospace Med., 1961, Vol. 32, p. 737.