EXTERNAL RESPIRATION AND GAS EXCHANGE DURING ORTHOSTATIC SYNCOPE

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A comparative analysis was made of the response of the cardiorespiratory system to the orthostatic test in 80 subjects with good resistance and 19 who developed syncope while in the vertical position. Significant differences were found in the responses of the gas exchange, hemodynamics, and external respiration. Changes in the heart rate, pulmonary ventilation, and partial CO_2 pressure in the alveolar gas were most demonstrative. The results, especially the absence of the expected decrease in oxygen consumption in the period before collapse, considerably broaden our ideas of the pathogenesis of orthostatic collapse.

KEY WORDS: *orthostatic test; collapse, oxygen consumption.*

The main cause of orthostatic or gravitational collapse is considered by most workers to be hypoxia of the brain tissue resulting from insufficiency of its circulation which, in turn, is due to a decrease in the venous return to the heart and a decrease in the minute circulatory volume [2, 10, 11, 13]. It could be supposed that the gas exchange, dependent on the minute circulatory volume [6], would be substantially reduced a few minutes before the development of collapse and that its dynamics could be used to judge the possibility of development of this disturbance, presenting a threat to human life.

Investigations of certain parameters of external respiration, gas exchange, and hemodynamics in persons with normal and extremely low orthostatic resistance were undertaken, first, to clarify the pathogenesis of orthostatic syncope and, second, to determine the possibility of predicting the development of a precollapse state from changes in certain recorded parameters.

EXPERIMENTAL METHODS

An analysis was made of the results of orthostatic tests on 80 healthy male volunteers aged 19-42 years, with a height of 164-187 cm and a weight of 59-88 kg, distinguished by good orthostatic resistance (group 1), and also on 19 persons whose tests ended in syncope (group 2). These subjects were male volunteers aged 18-36 years, with a height of 163-186 cm and a weight of 57-82 kg, who were tested after the end of periods of 10-30 days of strict confinement to bed, under the influence of which, as is well known, human orthostatic resistance is considerably reduced [1, 3, 8].

Investigations on a revolving chair, fitted with a support for the lower limbs, were carried out during the morning after a light breakfast. The subjects started the test in the horizontal position directly from bed. The investigations were not preceded by any loading tests or by intravascular manipulation.

After a rest of 30 min with the subject lying in the supine position the revolving chair was tilted in the course of 5-7 sec to an angle of 75-80° with the head uppermost. During the next 7-10 min in the horizontal position (HP) and throughout the subject's stay in the vertical position (VP) the oxygen consumption and CO_2 excretion were recorded continuously by an automatic gas analyzer; the CO_2 concentration in the final portion of expired (alveolar) gas and the respiration rate were recorded on a low-inertia infrared gas analyzer; the volume of the pulmonary ventillation was calculated from the readings of dry gas meters of a pneumotachograph, and the heart rate was determined from the ECG. The "oxygen pulse" was calculated as the ratio between the oxygen consumption and the pulse rate, and the volume of the func-

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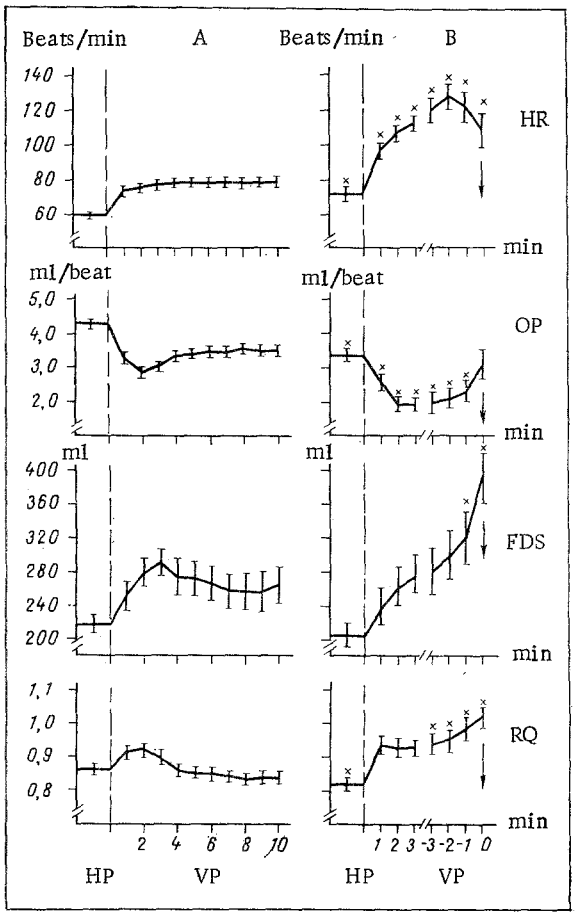

Fig. 1. Indices of hemodynamics, gas exchange, and external respiration recorded with subjects in horizontal (HP) and vertical (VP) positions of the body for groups 1 (A) and 2 (B) (mean value ±σ). HR) Heart rate, OP) oxygen pulse, FDS) function dead space at BTPS, RQ) respiratory quotient. Arrows indicate moment of collapse; crosses mark statistically significant ($P < 0.05$) difference between groups for comparison of mean values during HP, and also during each of the first and last minutes of the stay in VP.

tional dead space and the respiratory quotient were calculated by the usual formulas.

Since fainting responses developed in some subjects of group 2 at different periods of their stay in VP (6, 4, 5, and 4 cases after 6-10, 11-15, 16-20, and 26-39 min respectively), the indices specified above were analyzed in this investigation for a 3-min period at the beginning and a 4-min period at the end of VP, which immediately preceded the development of collapse. Indices for the same minutes at the beginning of the test (1st-3rd min) and also those recorded in a state of stable compensation during VP (7th-10th min) were analyzed for the subjects of group 1.

EXPERIMENTAL RESULTS AND DISCUSSION

The subjects of group 1 tolerated the orthostatic test well and had no complaints. The development of syncope, according to the reports of the subjects of group 2, was usually accompanied by weakness, dizziness, nausea, tinnitus, the appearance of a "veil" in front of the eyes, narrowing of the field of vision, and difficulty in breathing. Externally, the beginning of syncope was manifested as palor of the skin, acrocyanosis of the limbs, and perspiration from the palms, face, and neck.

Comparison of the data recorded in the subjects of both groups in the initial state (HP) showed a statistically significant difference ($P < 0.05$) only for the heart rate, the oxygen pulse, and the respiratory quotient (Fig. 1). During the first 3 min after the change in the position of the body a more rapid rise of the heart rate and pulmonary ventilation was observed (the latter only in the first minute in VO), together with a fall in the oxygen pulse and CO_2 partial pressure in the alveolar gas ($P < 0.05$) in the subjects of group 2 compared with group 1, whereas with respect to all other indices no statistically significant difference could be found (Figs. 1 and 2).

In the final minutes of the test, i.e., in the precollapse state, differences from normal were discovered for all indices recorded except the "oxygen pulse," which was virtually restored to normal in the last minute in VP (Fig. 1). This difference was manifested most clearly in relation to the heart rate, pulmonary ventilation, and the partial CO_2 pressure in

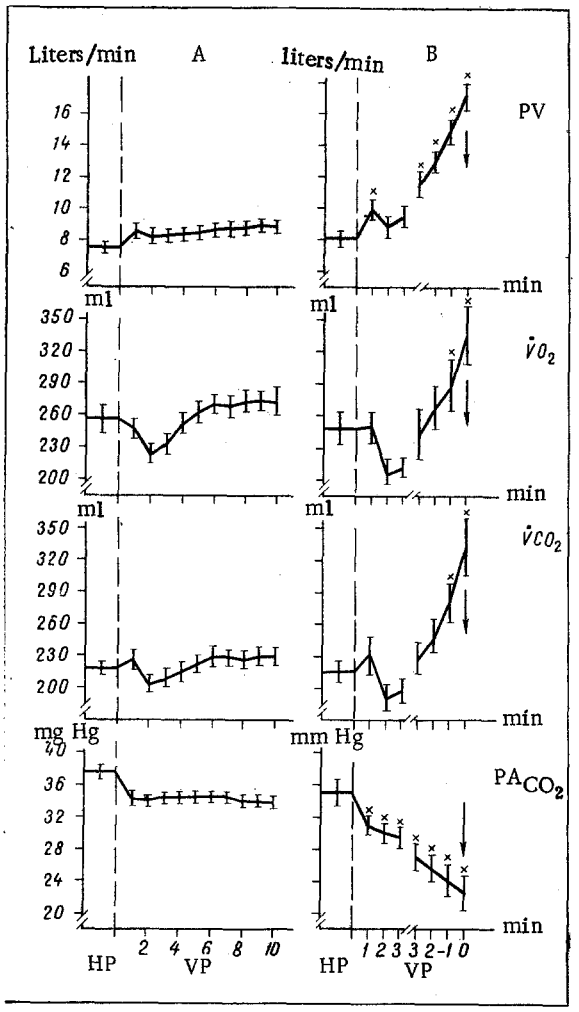

Fig. 2. Indices of gas exchange and external respiration recorded in subjects in horizontal (HP) and vertical (VP) positions of the body for groups 1 (A) and 2 (B) (mean data ±σ). PV) Pulmonary ventilation (BTPS), VO_2 and $\dot{V}CO_2$) oxygen consumption and CO_2 excretion (STPD), PA_{CO_2}) partial CO_2 pressure in alveolar gas. Remainder of legend as in Fig. 1.

the alveolar gas, which is dependent on it. The values of these indices and their dynamics (especially the latter) could clearly serve as reliable criteria of the development of the precollapse state.

For a few minutes before collapse the expected decrease in gas exchange could not be detected in any of the subjects of groups 2: The oxygen consumption and CO_2 excretion in this period were significantly greater than in HP and substantially greater than in subjects with good orthostatic resistance (Fig. 2).

Whereas the considerably greater increase in the volume of the pulmonary ventilation and in the heart rate in the orthostatically unstable subjects than normally can evidently be explained by stress of the protective mechanisms responsible for maintenance of an adequate circulation in VP, and the increase in the CO_2 excretion in the alveolar gas can be explained by hyperventilation, the increase in the oxygen consumption immediately before collapse is very difficult to interpret. Even a small increase in its reserves in the blood and lungs [12] could hardly cause such a sharp rise in the value of this index, especially when the minute circulatory volume is reduced. Whatever the case, the increase in the oxygen consumption during the development of collapse is a fact which deserves the closest study.

In the pathogenesis of orthostatic collapse clearly an important role must be played by the decrease in the partial CO_2 pressure in the alveoli and, hence, in the arterial blood. The reflex response to stretching of the lungs, the decrease in the venous return of blood and the minute circulatory volume, and the reflex response of the chemoreceptors to cerebral hypoxia after the change into the orthostatic position, due to the latter, are evidently the principal trigger mechanism of the substantial increase in pulmonary ventilation. Even in healthy subjects a decrease in the partial CO_2 pressure in the alveolar gas, i.e., "true" hyperventilation, was observed after the first minute of the stay in VP (Fig. 2). The value of this index in individual orthostatically unstable subjects fell in the last minute before

collapse to 20 mm Hg or below. A vicious circle was thus created, when hyperventilation, on the one hand, facilitated the return of venous blood and an increase in the minute circulatory volume as a result of an increase in the suction action of the thorax and the blood-expressing excursions of the abdominal wall, but on the other hand increased the hypoxia, for the consequent hypocapnia causes vasoconstriction in the brain and dilation of the peripheral vessels, thus interfering with the utilization of the blood oxygen by the tissues [5, 7, 9, 10].

The considerable increase in the dead space observed before collapse is evidently a defensive response of the body against hypocapnia. It can also be suggested that the breath holding periodically observed in the orthostatically unstable subjects was also a manifestation of defensive responses of the body against hypocapnia [4].

In conclusion, it must be emphasized once again that before the development of orthostatic collapse, instead of the expected decrease in the oxygen consumption a significant increase was observed. The decrease in the minute circulatory volume in the orthostatically unstable subjects, if indeed it took place at all, was compensated during this period evidently by the increasing arteriovenous oxygen difference [6, 12]. The onset of collapse is thus not preceded by the development of hypoxia in man, although it is probable that local circulatory hypoxia of the brain tissues is present.

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