RHEOGRAPHIC AND PLETHYSMOGRAPHIC INVESTIGATION OF VASOMOTOR CHANGES DURING BRIEF VOLUNTARY HYPERVENTILATION

V. L. Fantalova

UDC 612.216.2:612.1

Investigation of the effects of voluntary hyperventilation for 1 min by means of a composite amplitude characteristic, viz. the rheographic inflow index (RII) of the rheoencephalogram (REG) revealed an increase in the arterial inflow into the brain on account of an increase in the heart rate and dilatation of the arterial vessels. An increase in the cerebral circulation under the influence of hyperventilation was also observed by recording the rheoplethysmogram of the brain through a dc amplifier. The plethysmographic effect at the periphery could occur in the same direction, but other alternatives were possible. The direct response of the peripheral and cerebral vessels to hyperventilation was usually in different directions. Constriction of the peripheral arterial system with deepening alkalosis may be followed by dilatation. Variability of the effect of hyperventilation was observed not only in the peripheral but also in the cerebral circulation. The autonomous behavior of the cerebral and peripheral vessels during hyperventilation and immediately afterward may be followed by responses of uniform direction. This is demonstrated by the periodic respiration correlating with changes in the heart rate, the amplitude of the pulse volume fluctuations and the EEG picture. No direct correlation was found between the direct effects of hyperventilation on respiration and on the cerebral vessels.

Investigation of the vascular effect of hyperventilation is interesting both from the clinical aspect and also in connection with the theoretical problem of explaining the complex variable response of the body to hypocapnia [3].

Results described in the literature indicate dilatation of the limb vessels under the influence of hyperventilation, but responses of other types are also reported [2-4, 7-10]. With regard to the cerebral circulation, the view that the cerebral vessels are constricted during hypocapnia is widely held [11].

The object of this investigation was to compare vascular responses to voluntary hyperventilation by reference to indices of the cerebral and peripheral circulation, and with an analysis of the effect of hyperventilation on respiration, the ECG and the EEG.

EXPERIMENTAL METHOD

The effect of voluntary hyperventilation lasting about 1 min was investigated; the technique was described previously [3, 4].

The EEG, respiration, ECG, rheoencephalogram (REG), and the digital rheogram (RG_{dig}) were recorded on a multichannel electroencephalograph. The rheograph used had a working frequency of 150 kHz. The plethysmogram of the next finger and respiration were recorded simultaneously on a sensitive plethysmograph.

N. N. Burdenko Institute of Neurosurgery, Academy of Medical Sciences of the USSR, Moscow. (Presented by Academician V. N. Chernigovskii.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 75, No. 1, pp. 9-14, January, 1973. Original article submitted February 1, 1972.

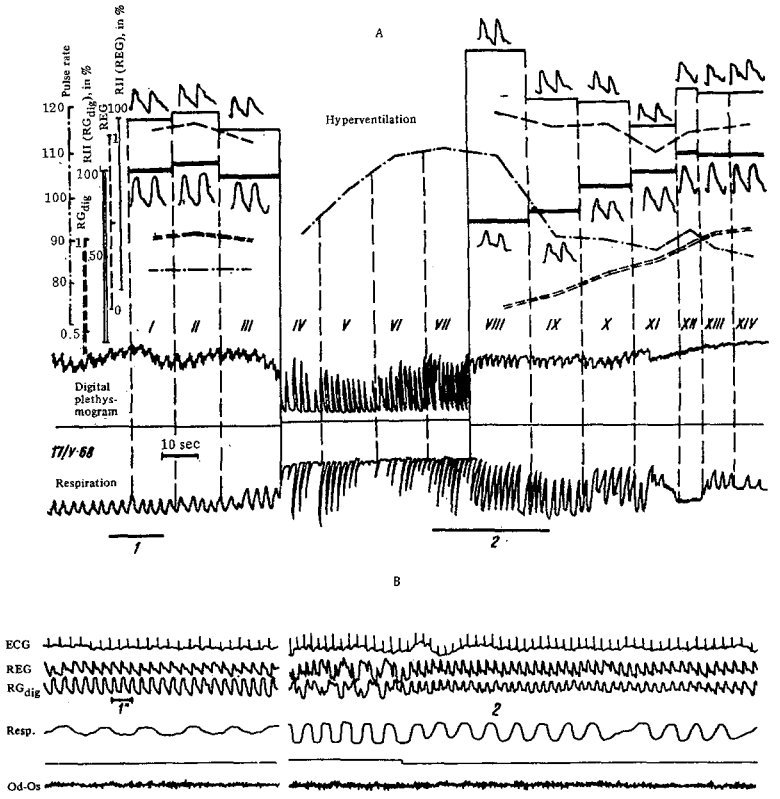

Fig. 1. One type of response to hyperventilation (healthy subject aged 25 years): A) plethysmographic record. From bottom to top: respiration, marker of hyperventilation, plethysmogram of left ring finger. The graph above the plethysmogram is plotted from the data of the digital rheogram and the REG; B) record on electroencephalograph. From top to bottom: ECG, REG (fronto-mastoid lead), plethysmogram of left index finger, respiration, marker of hyperventilation, EEG (transverse occipital lead). Cuts 1 and 2 of the record on the electroencephalograph correspond to the underlined cuts on the plethysmograph.

The data of the REG and RG_{dig} were analyzed by summation of their amplitudes over known time intervals and by calculation of the rheographic inflow index (RII) calculated for 1 min. The dynamics of the changes in RII in connection with hyperventilation were anlalyzed and compared with changes in the pulse rate and in the mean amplitude of the rheographic waves, and the changes in RII of the finger also were compared with the plethysmogram [5, 6]. To investigate fluctuations in the cerebral circulation in response to hyperventilation the technique of rheoplethysmography [1] was used.

Twelve healthy subjects aged from 17 to 35 years, 6 patients with peripheral nerve lesions (without a pain syndrome), and 35 patients with brain lesions, affecting mainly the brain stem and diencephalon, were investigated.

EXPERIMENTAL RESULTS AND DISCUSSION

The first characteristic response to hyperventilation was an increase in the pulse rate, commencing during the first 15-20 sec of hyperventilation (although immediately after the command the pulse rate often fell a little), followed by an even greater increase, and then by a gradual decrease after the end of the test (Figs. 1 and 2).

This increase in heart rate confirms results described in the literature.

The amplitude of the pulse waves on the REG usually increased somewhat after hyperventilation despite the increased pulse rate and the associated decrease in systolic output of the heart; this was evidence

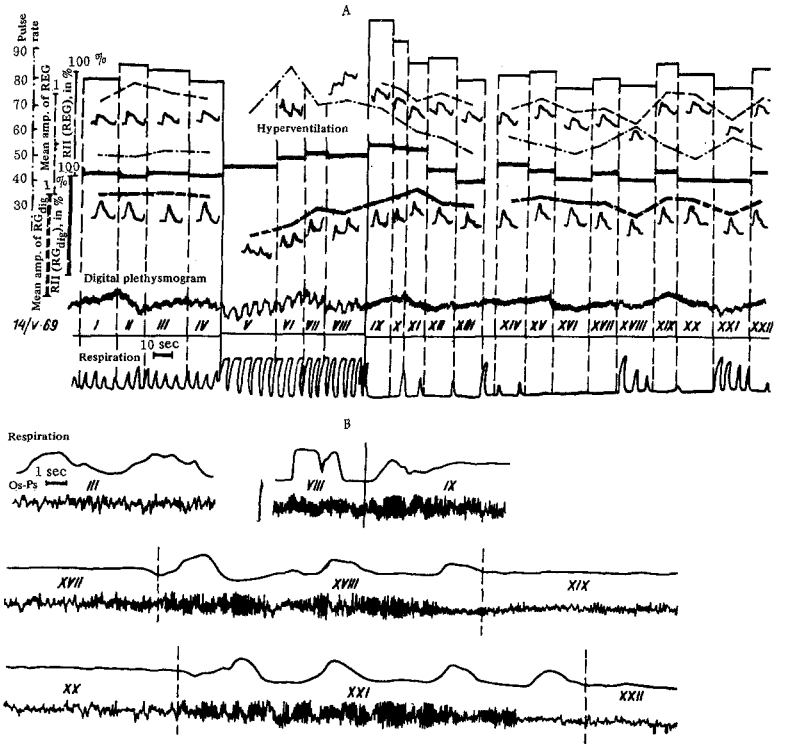

Fig. 2. Type of response to hyperventilation (healthy subject aged 32 years in slightly drowsy state): A) record on plethysmograph. From bottom to top: respiration, marker of hyperventilation, plethysmogram of left ring finger. Graphs above plethysmogram plotted from rheographic data; B) record on electroencephalograph. From top to bottom: respiration, EEG in left occipito-parietal lead. Continuous vertical line marks end of hyperventilation. Broken vertical lines correspond to equivalent lines on graph.

of dilatation of the brain arteries. The arterial inflow into the brain – RII(REG) – increased under these circumstances on account of two factors: the mean amplitude of the pulse waves and the pulse rate (see Figs. 1 and 2).

The dynamics of changes in the arterial digital inflow showed changes in various directions. The amplitude of the fluctuations in digital pulse volume usually fell first of all, evidently because of a fall in the systolic volume of the heart.

However, the decrease in amplitude could be on such a scale that, together with the increase in pulse rate, it led to a decrease in the peripheral arterial inflow – in RII (RG_{dig}) (Fig. 1). In that case it is logical to suggest constriction of the peripheral arteries. This was probably largely responsible for the decrease in the total blood volume in the organ, as in the observation recorded, for example, in Fig. 1. In other cases the peripheral pulse inflow rose under the influence of hyperventilation as the result of a marked increase in the heart rate and despite a decrease in the mean amplitude of the pulse waves (see Fig. 2, periods V, VI, VII, and VIII). This case demonstrates that the decrease was followed by a gradual increase in the mean amplitude of the pulse waves of digital volume, indicating some degree of dilatation of the peripheral arteries (Fig. 2, periods V, VI) under the influence of the increasing depths of alkalosis. This augments the increase in the peripheral arterial inflow (Fig. 2, periods V, VI, VII). The direction of the changes in the plethysmogram in this case corresponds once again to the direction of the fluctuations in RII (RG_{dig}). However, this was not always observed; in some cases, despite an increase in the arterial inflow, the digital plethysmogram fell evidently because of the increased venous outflow.

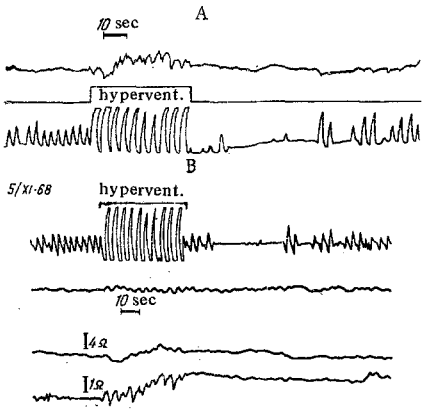

Fig. 3. Increase in blood filling of cerebral and digital vessels under the influence of hyperventilation (healthy subject aged 18 years): A) record on plethysmograph (shown as a control). From top to bottom: plethysmogram of left index finger, marker of hyperventilation, respiration; B) record on polygraph (PM-150, Japan). From top to bottom: marker of hyperventilation, respiration, photoplethysmogram of left ring finger, rheoplethysmogram of left middle finger. Rheoplethysmogram of brain (fronto-mastoid lead).

This conclusion regarding the increase in arterial inflow to the brain as the result of hyperventilation and the accompanying dilatation of the brain arteries is contrary to the well known view that the cerebral vessels are constricted under the influence of hypocapnia, and it was therefore checked by a series of observations using rheoplethysmography. The results of an investigation are shown in Fig. 3. Besides the digital mechanoplethysmogram and rheoplethysmogram, demonstrating an increase in the digital blood volume under the influence of hyperventilation, the rheoplethysmogram of the brain also was recorded. A marked increase in the cerebral blood volume was observed initially, gradually decreasing at the end of the test. This fact confirms the conclusion made on the basis of the amplitude characteristics of the REG that the cerebral arteries dilate under the influence of hyperventilation.

The increased inflow of blood to the brain was presumably the explanation of the sensation of a feeling of warmth arising in the head, which the subjects frequently described when questioned.

The possibility cannot be ruled out that the vasodilatation of the cerebral vessels observed in these experiments is only the first phase of the response to hypocapnia (in these experiments the hyperventilation was of short duration), but it may also be that arterial vasoconstriction in general is not the leading mechanism of this response.

It should also be noted that the type of response of the cerebral vessels described above was not the only type found during the investigation of healthy subjects. In one case in response to hyperventilation, in which the pulse rate remained unchanged, there was virtually no shift in the basic level of the cerebral rheoplethysmogram, whereas the peripheral blood filling decreased with a simultaneous fall in the amplitude of the pulse waves. The response of the cerebral vessels to hyperventilation, on the other hand, was hardly perceptible.

In another case there was a persistent decrease in the cerebral blood volume without any marked change in amplitude of the pulse waves of the REG, despite an increase in the pulse rate. This effect was evidently attributable to the sharply increased venous outflow from the cerebral vessels.

At the end of voluntary hyperventilation the values of RII (REG) and RII (RG_{dig}) returned gradually to their initial level which, in the case of healthy subjects, was reached at the end of the first or beginning of the second minute after the end of hyperventilation or even sooner. In some cases, if the dynamics of the digital arterial inflow was compared with the plethysmogram, an increase in the velocity of the peripheral blood flow could be detected after hyperventilation (Fig. 1); if the level of the plethysmogram was unchanged the inflow and, consequently, the outflow rose – i.e., the blood flow increased.

In the patients with brain lesions it often took much longer for the initial state of the vessels to be restored, and sometimes this happened predominantly or only in the peripheral circulation. In brain-stem pathology the response of the heart could be very prolonged.

Detailed results for the vascular responses to hyperventilation in the patients will be published later.

Changes in the shape of the pulse waves during and after hyperventilation call for special comment: tapering of the summit to a point, a steeper decline, downward displacement and sharpening of the dicrotic wave. They were largely similar on the cerebral and peripheral rheograms, although they were not always distinct on the REG, and in RG_{dig} they were sharper and more definite. These changes are clearly visible in Fig. 1 as regards both RG_{dig} and REG, but after hyperventilation they were well marked only in RG_{dig} (Fig. 2; in REG they are visible only during the test).

These changes in the shape of the pulse waves appeared independently of the direction of the changes in their amplitude and they were evidently connected with facilitation of the venous outflow during and immediately after hyperventilation.

In connection with the hypothesis of a possible link between the response of the respiratory system to hyperventilation and the character of the response of the cerebral vessels [4] it was interesting to compare them. The comparison, however, failed to demonstrate any definite direct correlation between them. For instance, in the two cases shown the respiratory effect immediately after hyperventilation was opposite (hyperpnea in Fig. 1, apnea in Fig. 2), yet in both cases an increase in the arterial inflow to the brain was observed as a result of an increase in the pulse rate and some degree of arterial vasodilation. Consequently, the absence of apnea cannot be explained by marked constriction of the cerebral arterial system, presenting hypocapnia, unless some specific manifestation of the vascular effect is assumed in the region of the structures controlling respiration.

The dynamics of the vascular changes in the period after hyperventilation was complex and varied, and was evidently influenced by several factors: the diminishing gas alkalosis, the irregularity of respiration, and the development of asphyxia – in the presence of long periods of breath-holding, and also the state of the brain systems the variations in whose level of activity may largely determine the course of this whole period, as reflected in the indices of physiological systems, including respiration and the EEG. This last effect can be demonstrated in the case of periodic respiration, arising at the end of the second minute after hyperventilation (Fig. 2). In this case the periods of hyperpnea are matched by bursts of high α-activity on the EEG (in patients with cerebral disorders bursts of Δ- and θ-waves were sometimes observed), an increase in the pulse rate, and a decrease in the mean amplitude of the pulse waves, leading to a decrease in RII (REG and RG_{dig}).

The changes during apnea are opposite in direction to this "activation complex" for they include suppression of α-activity on the EEG and the appearance of slower, low-voltage waves (the subject was in a drowsy state), slowing of the pulse, and an increase in the mean amplitude of the pulse waves in the cerebral and peripheral rheographic leads (Fig. 2, periods XVIII-XXII). This periodicity, of generalized character, undoubtedly reflects fluctuations in the level of excitation of the activating reticular formations of the brain stem, and this conclusion is also valid for the series of cases in which periodic respiration appeared spontaneously in patients with brain lesions.

These observations show that during and immediately after hyperventilation the responses of the cerebral vessels show some degree of autonomy by comparison with those of the peripheral vessels, while in the late period after hyperventilation, when respiration is ataxic or periodic, the vessels of the brain and limb may behave identically, although the scale of the fluctuations in amplitude of the pulse waves and, correspondingly of the arterial inflow (Figs. 1 and 2) may differ appreciably.

LITERATURE CITED

1. A. V. Anzimirov, B. G. Spirin, and V. L. Fantalova, Byull. Éksperim. Biol. i Med., No. 4, 116 (1970).
2. A. M. Bentelev, Fiziol. Zh. SSSR, 42, 363 (1956).
3. V. L. Fantalova and O. E. Turchaninova, Fiziol. Zh. SSSR, 56, 237 (1970).
4. V. L. Fantalova, Fiziol. Zh. SSSR, 56, 632 (1970).
5. V. L. Fantalova, Byull. Éksperim. Biol. i Med., No. 8, 3 (1967).
6. V. L. Fantalova, Byull. Éksperim. Biol. i Med., No. 12, 15 (1967).
7. D. J. Abramson and E. B. Ferris, Am. Heart J., 19, 541 (1940).
8. R. J. Clarke, J. Physiol. (London), 118, 537 (1952).
9. J. D. Colmann and P. Kelly, Am. J. Physiol., 211, 1255 (1966).
10. C. J. Roddie, J. F. Shepherd, and R. F. Whealen, J. Physiol. (London), 137, 80 (1957).
11. Z. A. Sapherstein, J. Clin. Invest., 41, 1429 (1962).