RESISTANCE IN LARGE AND SMALL ARTERIES OF THE BRAIN DURING PAROXYSMAL ACTIVITY

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The pressure in the aorta, the vessels of the circle of Willis, and the venous sinuses of the brain, the partial oxygen pressure in the brain tissue (pO_2), and cortical electrical activity were recorded in experiments on dogs. The resistance to the blood flow in the main arteries of the brain (R_m) and in the smaller arteries lying peripherally to the circle of Willis (R_p) was calculated mathematically. During paroxysmal activity induced by intra-arterial injection of strychnine, R_p was reduced, the cerebral blood flow was increased, and pO_2 was increased after a slight decrease. By contrast, R_m was increased, evidently as a result of compensatory vasoconstriction of the main arteries, aimed at reducing the excessive hyperemia of the brain.

In recent years differences have been observed in the functional behavior of different parts of the brain vascular system during paroxysmal activity. For instance, the main arteries of the brain do not react to local application of strychnine to the cerebral cortex, the pial arteries are regularly dilated (especially the small vessels), while the cortical arteries are progressively constricted [4]. The total resistance of the brain vascular system has been estimated from measurements of the intensity of the blood flow [1, 10, 11, 13], but its changes in different parts of the vascular system have not yet been studied.

The object of this investigation was to determine changes in the hydraulic resistance in the main arteries of the brain (R_m) and in the smaller arteries of the brain lying peripherally to the circle of Willis (R_p) during paroxysmal activity.

EXPERIMENTAL METHOD

The investigation was carried out with the aid of a recently introduced mathematical method consisting of the solution of a system of two equations: the first equation was obtained from experimental data and described the hemodynamic properties of the cerebral vascular system as an averaged curve (regression line), while the second equation expressed the principle of continuity of the blood flow [2, 6]. The equations were solved by computer, and the original information consisted of the pressure in the aorta (P_a), in the circle of Willis (P_{cw}), and in the venous sinuses of the brain (P_s), recorded experimentally. Changes in vascular resistance rather than its absolute values can be determined from these results.

Altogether 17 experiments were carried out on dogs of different breeds, anesthetized with pentobarbital (0.04 g/kg, intraperitoneally). The systemic arterial pressure and the pressure in the circle of Willis were recorded through catheters inserted into the right common carotid artery in the aortic and cranial directions after ligation of the corresponding branches; the venous pressure in the brain sinuses was recorded through a catheter introduced in the retrograde direction into the left external jugular vein after ligation of the corresponding branches. The surgical exposure of the vessels for insertion of the catheters was

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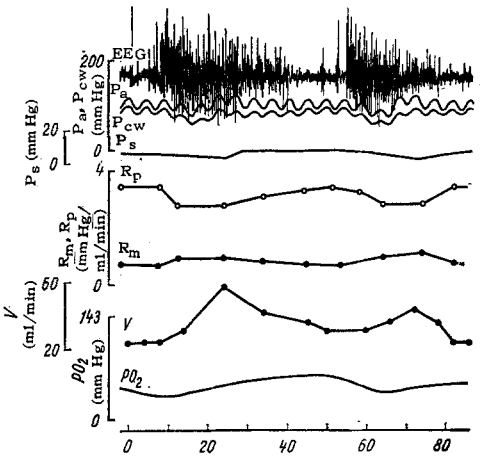

Fig. 1. Dynamics of indices of circulation and pO_2 in the brain during paroxysmal activity. EEG) Electroencephalograms; P_a) systemic arterial pressure; P_{cw}) pressure in circle of Willis; P_s) pressure in venous sinuses of the brain; R_p) hemodynamic resistance in vessels peripherally to circle of Willis; R_m) hemodynamic resistance in main arteries of the brain (internal carotid and vertebral); V) volume velocity of blood flow in brain; pO_2) partial pressure of oxygen in brain tissue. Abscissa, time (in sec).

described earlier [4]. The partial oxygen pressure (pO_2) was recorded quantitatively by means of a combined polarographic electrode as designed by Lübbers [9], placed on the brain surface (a burr-hole was drilled in the left parietal region). The EEG was recorded using electrodes screwed into the cranial bones at the margins of the burr-hole. The blood pressure levels (P_a, P_{cw}, P_s) were recorded by electromanometers simultaneously with pO_2 and the EEG on an eight-channel Mingograph-81 apparatus (Elema-Schenander, Sweden).

Paroxysmal activity in the brain was evoked by injection of 15 mg strychnine through a catheter inserted in a retrograde direction into the left anterior thyroid artery as far as the carotid artery, all branches of which except the internal carotid had been previously ligated. To abolish the convulsions, which prevented normal recording of the physiological parameters, the animal received an intravenous injection of the curariform agent diplacin (5 ml of the 2% solution), after which the lungs were artificially ventilated by the AM-1 apparatus.

EXPERIMENTAL RESULTS

After intra-arterial injection of strychnine paroxysmal discharges appeared on the EEG and could easily be distinguished against the background of the spontaneous activity (Fig. 1). Groups of spikes (over 200 μV in amplitude), 25-50 sec in duration, appeared periodically at intervals of about 40 sec. Under these circumstances P_a at first fell slightly and then usually rose, P_{cw} followed the changes in P_a more or less, while P_s usually rose slightly.

The changes in the vascular resistances calculated by the above method were as follows: during the periods of appearance of paroxysmal discharges R_p as a rule fell while R_m either remained unchanged or increased (Fig. 1). The blood flow in the brain (V), changes in which were obtained by the formula

$$V = \frac{P_{cw} - P_s}{R_p},$$

in the periods of paroxysmal activity were considerably increased, just as in previous investigations when measured by the clearance of radioactive krypton and other methods [1, 10, 12]. The increase in the cerebral blood flow undoubtedly depended on the decrease in R_p (more especially because in many experiments R_m was actually increased). The decrease in R_p depended in turn on dilatation of the pial arteries [3, 7], and this was not prevented by the constriction of the lumen of the cortical arteries which developed under these conditions [5].

During the period when spikes appeared on the EEG, pO_2 at first fell (evidently because of a marked decrease in the oxygen consumption of the brain tissue structures [8, 11, 13]), but then increased (Fig. 1). This increase could be explained only by an excessive increase in the blood flow and by the supplying of so much oxygen that it could not be absorbed by the cerebral cortex.

By contrast with local application of strychnine, when the main arteries of the brain did not respond [3], after intra-arterial injection of strychnine in these experiments R_m was frequently increased. This could not be explained by the direct vasoconstrictor action of strychnine on the main arteries of the brain, for the vasoconstriction did not take place during the period of strychnine administration, but it appeared periodically, simultaneously with the paroxysmal discharge on the EEG (Fig. 1). Consequently, constriction of the main arteries of the brain took place under the influence of impulses arising in the brain at the time of appearance of the paroxysmal discharges. Presumably this constriction of the main arteries of

the brain was reflex in origin as the result of excessive dilatation of the pial arteries and congestion of the brain with the blood, as has been observed in postischemic hyperemia and venous stasis in the brain [4].

During paroxysmal activity in the brain differences are thus found in the functional behavior of different parts of its vascular system, each of which has its own particular function to perform, despite the fact that this may exert a mutually opposite effect on the overall cerebrovascular resistance.

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