REFLEX CONTROL OF CEREBRAL CIRCULATION

COMMUNICATION III. THE ROLE OF THE CAROTID SINUS AND THE INTEROCEPTORS IN THE REGULATION OF CEREBRAL CIRCULATION

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In spite of many investigations, the problem of the effect of the sinocarotid reflexes on cerebral vascular tone remains unsolved. According to Bouckaert and Jourdan [4], A.A. Shlykov [3], K. Gollwitzeer-Meier [5], and N.M. Ryzhova [2] and others, stimulation or excision of the sinocarotid receptors causes expansion or contraction of the cerebral vessels independently of changes in the arterial pressure. However, H. Rein [10] M. Schneider and D. Schneider [11], C. Heymans [7] and others found that these cerebral blood vessel changes are accompanied by changes in the arterial pressure, and deny that impulses from the carotid sinus have any direct effect on the blood vessels.

In our laboratory N.M. Ryzhova has shown that these contradictory findings are due in many cases to faulty methods. She showed that under normal conditions, impulses from the baroceptors of the carotid sinus maintain the cerebral vessels in a somewhat dilated condition. Increase of impulses causes a further dilatation, while their elimination causes a marked contraction of these vessels. From this, one would expect that in pressor and depressor reflexes, a change in the number of impulses from the carotid sinus would affect cerebral vascular tone. However most authors maintain that this mechanism plays no part in the reflex control of cerebral circulation, but claim that the control is mediated through changes in arterial pressure (C. Heymans [7], B. N. Klosovsky [1] and others).

In studying changes in cerebral circulation due to stimulation of the intestinal receptors, we showed that the observed changes in the blood flow through the cerebral vessels are caused not only by changes in the arterial pressure, but also by changes in the size of the lumen of the vessels as they contract or expand. The rate at which the size of the lumen changes after stimulation indicates a direct nervous action. After considering the parallel changes in arterial pressure and flow rate during stimulation of the interoceptors in normal conditions and in hypercapnia we concluded: 1) there is a reflex control of vasoconstrictor and vasodilator nerves; 2) whichever of these two effects prevails, resulting in a contraction or a dilatation of the cerebral vessels, depends to a considerable extent on the initial tone of the cerebral vessels.

Because the impulses from the sinocarotid receptors exert a considerable control over the tone of the vessels in normal conditions, and are increased or decreased by changes in the arterial pressure, it is probable that the carotid sinus controls reflex changes in cerebral circulation.

EXPERIMENTAL METHODS

To confirm this hypothesis, first, we reduced the flow of impulses from the baroceptors of the carotid sinus by applying pressure to the carotid artery, and took measures to ensure that any increase in arterial pressure would not cause an increase in this flow, then, we found the effects of stimulating the (intestinal) interoceptors on the blood flow through the vessels of the cerebral meninges. An inelastic cuff of soft lead 4-7 mm thick was placed around the bifurcation of the carotid artery so that it enclosed this region without exerting pressure on it and without restricting the blood supply to the brain, but, so that it prevented any stretching of the vessel walls, occurring upon increase of arterial pressure. This method was based on that of C. Heymans [7], E. Koch [8], W.H. Hauss, H. Kreuziger and H. Asteroth [6], and S. Landgren [9], who showed that the stimulus to the baroceptors is the stretching of the walls of the sinus and not the pressure itself within the sinus. In a separate investigation it was shown that increase of pressure within the sinus causes no increase in the flow of impulses in the nerve from the sinus or any fall in arterial pressure.

The experiments were carried out on 25 dogs under morphine-urethane anesthesia. Simultaneous recordings were made on a photokymograph of the arterial pressure – using a membrane manometer in the femoral artery, of the respiration – using a pneumograph,of the blood flow in the cerebral meninges – using a thermoelectric method with a fine thermoelectrode (A.M. Blinova and N.M. Ryzhova). Stimulation of the interoceptors was effected by introducing air at a pressure of 80 – 90 mm into a section of small intestine.

EXPERIMENTAL RESULTS

As we showed previously, stimulation of the interoceptors of the intestine in anesthetized animals causes an increase of arterial pressure and an increase in blood flow through the vessels of the cerebral meninges, in most cases, though the effect varies somewhat from one animal to another. Compression of the carotid arteries causes an increase in arterial pressure and a reduced blood flow to the brain. In certain cases,after clamping the carotid arteries, stimulation of the interoceptors causes a poorly shown increase in blood flow to the brain, or else none at all. Fig. 1 shows the results of one of these experiments. Stimulation of the interoceptors caused first an insignificant fall and then a small rise in arterial pressure and a well-shown increase in cerebral blood flow, which at the end of the stimulation gradually returned to the original value (Fig. 1,a). When the carotid arteries were clamped and the same stimulus applied, there was a very much smaller and poorly-maintained increase of blood flow, despite the large increase of arterial pressure (Fig. 1,b). In most of these experiments with clamped carotid arteries stimulation of the interoceptors had an effect on cerebral blood flow which was the reverse of the normal reaction.

In the experiment shown in Fig. 2, in the first reaction there was a well-marked increase in arterial pressure and a small increase in cerebral blood flow (Fig. 2,a). When the carotid arteries were clamped, stimulation of the interoceptors, instead of causing an increase, caused a marked reduction in blood flow (Fig. 2,b). The increase in the arterial pressure was even greater than in the first reaction, so that the depression in the blood flow shows a considerable increase in tone, i.e. contraction of the cerebral vessels.

After placing the cuffs on the point of bifurcation of the carotid arteries, stimulation of the interoceptors did not cause a reversed reaction on the cerebral vessels. But in all these experiments we found that after stimulation there was a smaller increase in blood flow to the cerebral vessels than in the first reaction (prior to putting on the cuffs).

Figure 3 shows the results of one of these experiments. The increase in cerebral vessel blood flow which, is observed before placing the cuffs in position and after stimulation of the interoceptors (Fig. 3,a) no longer occurs when the stimulation is applied with the cuffs in position (Fig. 3,b). When the cuffs are removed the original reaction is again restored, and in some experiments the effect is even greater than after stimulation prior to putting on the cuffs (Fig. 3,c).

Thus, when increase in baroceptor impulse flow following increased intrasinus pressure is prevented, the reflex leads to a smaller dilatation of the cerebral vessels than in the case with normal carotid sinus function. After elimination, by clamping the carotids, of the impulses from the carotid sinus baroceptors which serve to dilate the cerebral vessels, the reflex action results in a contraction instead of the usual dilatation of the cerebral vessels, in most cases. These observations show that the more strongly the vasoconstrictor action at the

time the interoceptor stimulation occurs the fewer the vasodilator impulses from the carotid sinus receptors, i.e. the stronger the tone of the cerebral vessels.

The greater effects of interoceptor stimulation with clamped carotid arteries may follow from two causes. First, when the stimulus is applied, there is already an increased cerebral vessel tone, so that the same arterial pressure cannot cause a dilatation on account of the increased firmness of the walls (E. Wetterer and H. Piper [12]). Second, the dilator action of the impulses from the aortic region is not sufficient to compensate for the absence of the impulses from the carotid sinus, as the aortic mechanism has already been expended in compensating for the absent carotid sinus impulses caused by the reduced pressure following clamping the carotids. The greater increase of arterial pressure during stimulation of the interoceptors with clamped than with unclamped carotids, also points to the inadequacy of the aortic mechanism. At the same time, after the cuffs have been applied, the function of the aortic mechanism is still intact. Under these conditions there is no greater increase in arterial pressure than there was initially, as the absence of an increased flow of impulses from the carotid sinus baroceptors is completely compensated for by the increased flow from the baroceptors of the aortic region. However the smaller increase in cerebral blood flow during the interoceptor stimulation indicates the inadequacy of the compensation as affecting dilatation of the cerebral vessels.

The results we have obtained allow us to draw the following conclusions: the impulses from the baroceptors of the carotid sinus during pressor interoceptor reflex action reduce cerebral vessel tone and so increase the blood supply to the brain; reduction of the elasticity of the arterial wall of the carotid sinus may have the unfavorable effect of causing a reduction in cerebral blood flow following interoceptor stimulation.

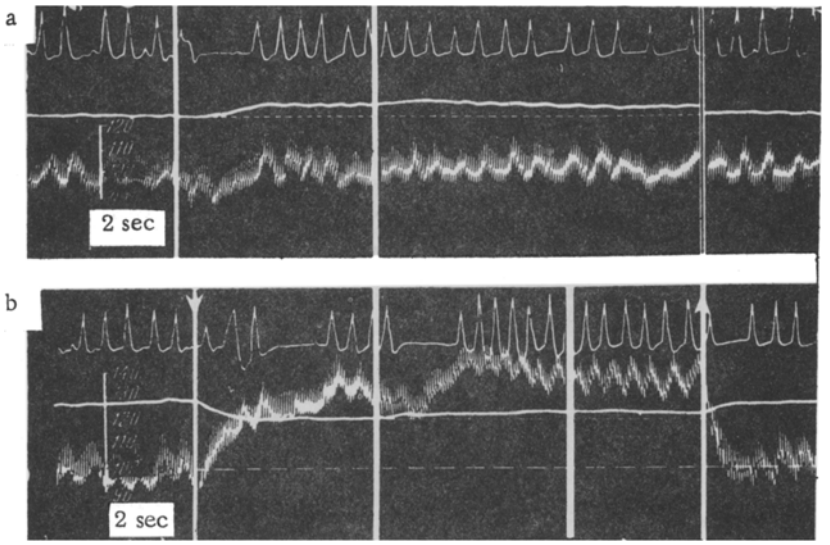

Fig. 1. Changes in cerebral blood supply after elimination of baroceptor impulses. Experiment of January 13, 1956. Curves, from above downward: Respiration; blood flow in cerebral meninges; arterial pressure. a) Initial reaction; b) reaction with clamped common carotid arteries. Vertical lines) beginning and end of stimulus; double vertical lines) interruption of record; vertical lines with an arrow) application and removal of clamps to common carotid arteries. Dotted line) values of the recorded functions prior to experiment. Traces read from right to left.

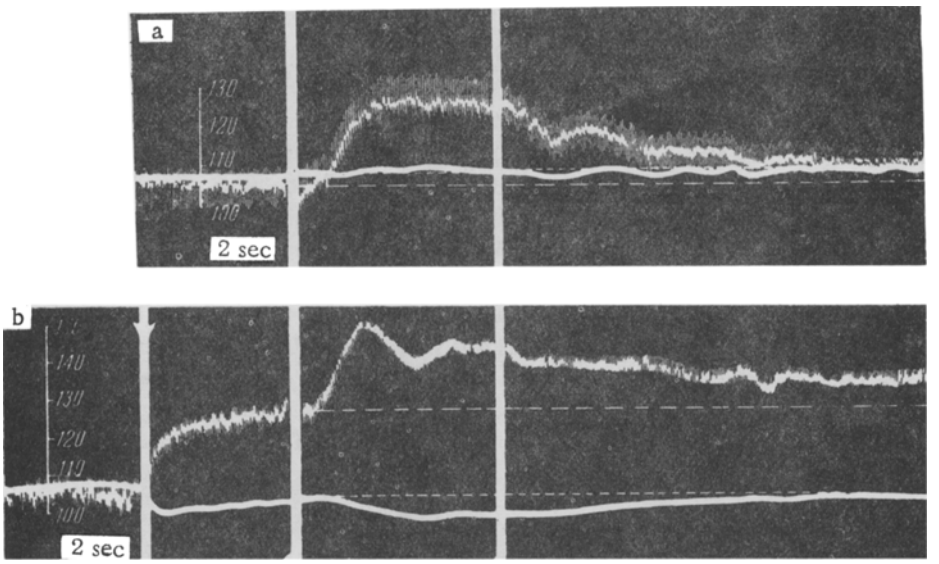

Fig. 2. Changes in cerebral blood flow. Experiment of June 6, 1956. Curves, from above downward: Blood flow in the cerebral meninges; arterial pressure.
a) Initial reaction; b) reaction after clamping common carotid arteries. Significance of vertical and dotted lines as in Fig. 1. Traces read from left to right.

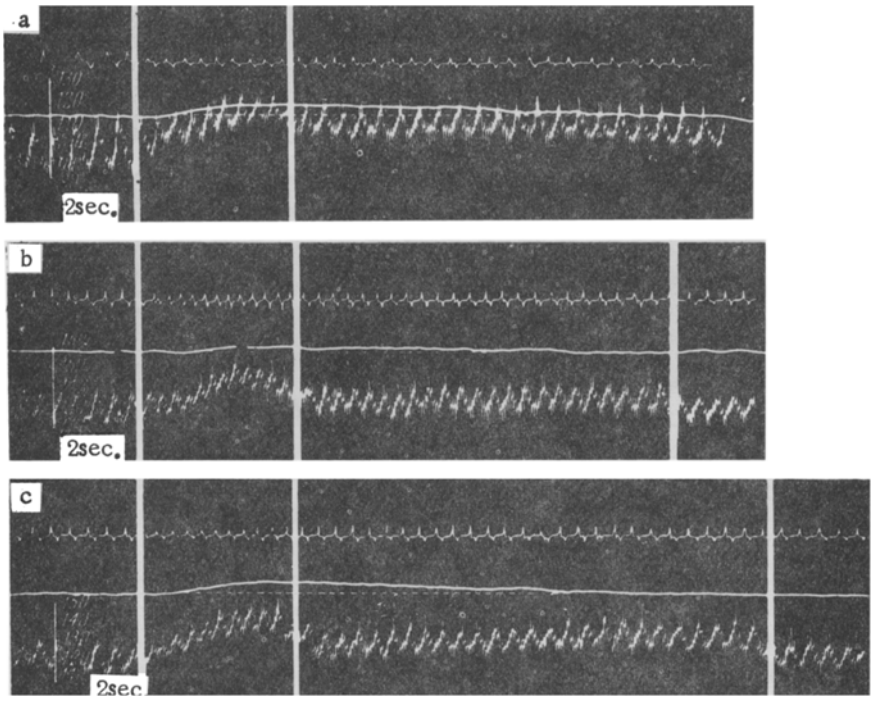

Fig. 3. Changes in cerebral Blood supply. Experiment of Feb. 28, 1956. Curves, from above downward: Respiration; blood flow to cerebral meninges; arterial pressure.
a) Initial reaction; b) reaction with cuffs on both carotid sinuses; c) reaction 15 minutes after removal of the cuffs. Significance of vertical and dotted lines as in Fig. 1. Traces read from left to right.

SUMMARY

The change of the volume velocity of the blood circulation was studied in the brain meninges during stimulation of intestinal interoceptors, which caused hypertension and increased the blood circulation of the brain. The thermoelectric method was employed. In stimulation of interoceptors with occluded carotid arteries there was a decrease in volume velocity of the blood circulation of the brain, instead of increase. When cuffs were placed on the area of carotid bifurcation the increase of the volume velocity was less marked than in the original reaction (before the application of the cuffs). It was concluded that the impulses from the baroreceptors of the sinocarotid zone during the pressor interoceptive reaction cause decrease of the tonus of the brain vessels and provide increased blood supply to the brain.

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* In Russian.

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