# BLOODFLOW MEASUREMENTS OF THE NEUROMUSCULAR APPARATUS OF THE INTERNAL CAROTID ARTERIES MADE BY A PHOTOMETRIC METHOD. COMMUNICATION II. CONTROL OF THE SYMPATHETIC NERVOUS SYSTEM

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It is known that the carotid nerve runs from the superior cervical sympathetic ganglia to the internal carotid artery and that its fibers spread out along the cerebral arteries as far as their finest divisions [3]. However, in physiological experiments in which the ganglia are stimulated it is difficult to obtain any definite contraction of the pial arteries: the effect is variable, extremely weak [3, 9], and sometimes absent altogether [10]. Only when the total bloodflow to the brain is recorded by photoelectric counting of drops from the sagittal sinus [11] or determined by resistance recordings [1] has any reduction of cerebral circulation been observed; however, it is still not clear which cerebral vessels are affected by the sympathetic system.

Because of the discovery made in the last few years of the important part played by the internal carotid and vertebral arteries in the regulation of the cerebral circulation [5] we decided to study the extent of the sympathetic control over them.

## EXPERIMENTAL METHOD

Because each of the four regional arteries receives its sympathetic innervation from different sources (the carotids from the superior cervical, and the vertebral from the stellate ganglia), we have studied the influence of one of the superior cervical ganglia on the internal carotid artery of the same side.

Some of the experiments were carried out on 12 adult rabbits under 1 g/kg urethane anesthesia, but most were performed on 21 dogs anesthetized with 0.1 g/kg chloralose.

On both sides all branches of the carotid arteries except the internal carotids were ligated (in some experiments we left only the external carotid arteries free). Cannulae from a hemotachometer for recording the bloodflow (and resistance) in the internal carotid arteries were tied into the common carotids*.

After the superior cervical sympathetic ganglia had been carefully dissected out, electrical stimuli from 2-6 v at 50 impulses per second were applied.

In order to eliminate possible changes of general arterial pressure which might influence bloodflow in the internal carotids, in some experiments we used the normal type of compensator whose cannula was inserted into the femoral artery.

## EXPERIMENTAL RESULTS

Influence of the superior cervical ganglion. Electrical stimulation at 2-6 v caused a reduction of bloodflow through the ipsilateral internal carotid, while as a rule there was no change on the opposite side. This result indicates that the nervous elements contained in the superior cervical ganglion exert a direct influence on the carotid

*See Byull. éksper. biol., 1963, No. 1, p. 6.

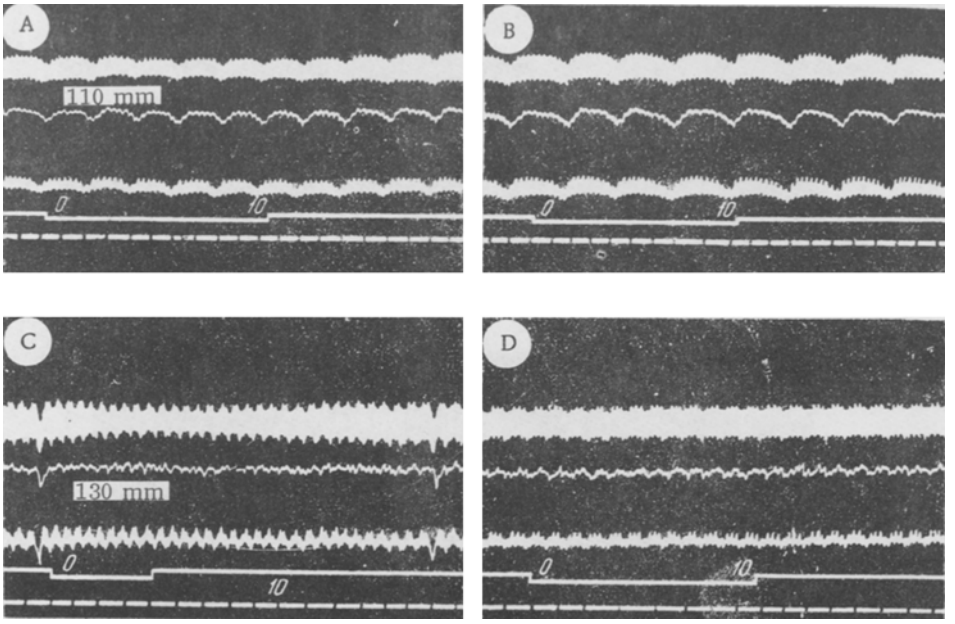


Fig. 1. Constriction of ipsilateral internal carotid artery on stimulation of the superior cervical sympathetic ganglion with impulses of 2 v (A, C). The absence of any effect on stimulation with 3 v after intravenous injection of 0.1 mg/kg ergotamine is shown in B and D. A and B) Dog weighing 16.5 kg. Curves, from above downwards: bloodflow in left internal carotid artery (on stimulated side); arterial pressure in femoral artery (pressure compensator used); bloodflow in right (control) internal carotid artery; stimulus marker; time marker (1 sec). Bloodflow in left internal carotid artery for A, as follows: 0 sec - 1.27 ml/sec, 10 sec - 0.95 ml/sec, for B: for the whole record 1.34 ml/sec. C, D) Dog weighing 12 kg. Curves, from above downwards: current flow in right internal carotid artery (on stimulated side); arterial pressure; bloodflow in left internal carotid artery; stimulus marker; time marker (1 sec). Bloodflow in right internal carotid artery, for C, as follows: 0 sec - 1.12 ml/sec, 5 sec - 0.88 ml/sec, for D: for the whole trace 0.98 ml/sec.

artery of the same side. The extent of its contraction (as judged from the reduction of bloodflow) for stimuli of 2 v varied according to the experiment from 17 to 72% of the original blood pressure (Fig. 1); with stimuli of 6 v as a rule bloodflow ceased, indicating complete closure of the lumen of the internal carotid artery*.

The constrictor effects obtained in the case of the internal and external arteries could be compared. The bloodflow through the external carotid was measured when the internal carotid was ligated. Stimuli were applied to the superior cervical ganglion. It was found that the threshold of excitability was approximately the same in the two cases: both the internal and external carotid arteries constricted when the stimulus intensity was 2 v. This result indicated that the sensitivity to nervous impulses of the neuromuscular apparatus of the internal carotid artery regulating cerebral circulation is approximately the same as that of the supply to the extracerebral arteries†.

The strictly ipsilateral contraction of the internal carotid arteries indicated that sympathetic impulses from the superior cervical ganglia did not extend their influence to the opposite internal carotid. At the same time the absence of any effect on the contralateral internal carotid showed that neither the vessels of the circle of Willis nor the

* At the present time it is difficult to determine whether the artery was closed along the whole of its length or only in certain places, as occurred in our previous experiments[4].

† Some authors [3, 9] have found that in cats only when vigorous stimulation is applied to the superior cervical sympathetic ganglion is there any constriction of the pial arteries, and that it does not exceed 8% of the diameter, whereas the extracranial arteries close completely.

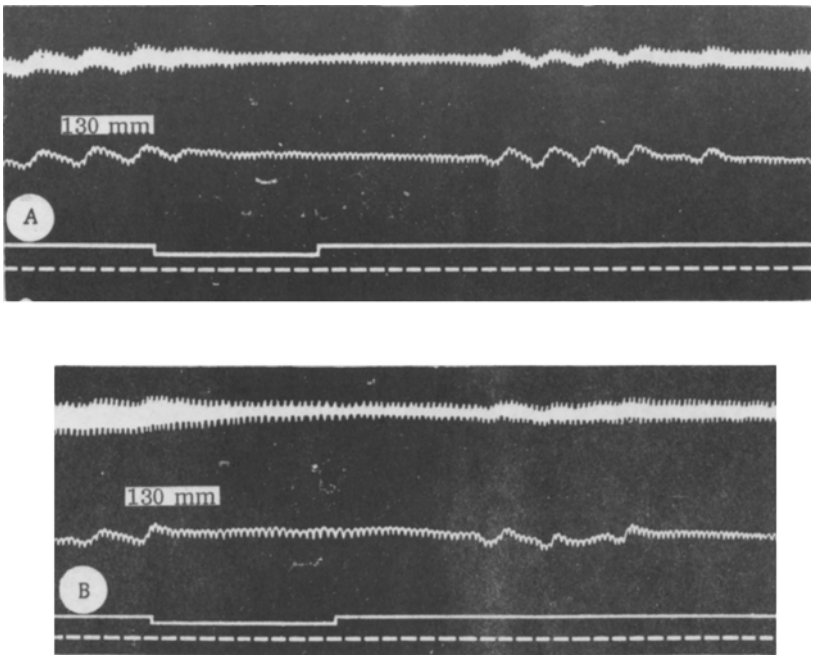


Fig. 2. Reduction of bloodflow in right internal carotid artery of a dog on electrical stimulation of the superior cervical sympathetic ganglion of the same side (A) before and (B) after enucleation of the ipsilateral eye. Curves, from above downwards: bloodflow in artery; arterial pressure in femoral artery; stimulus marker (2 v), time marker (1 sec). Bloodflow for A: before stimulation - 0.87 ml/sec, after stimulation - 0.54 ml/sec; for B: before stimulation - 1.07 ml/sec, after stimulation - 0.54 ml/sec.

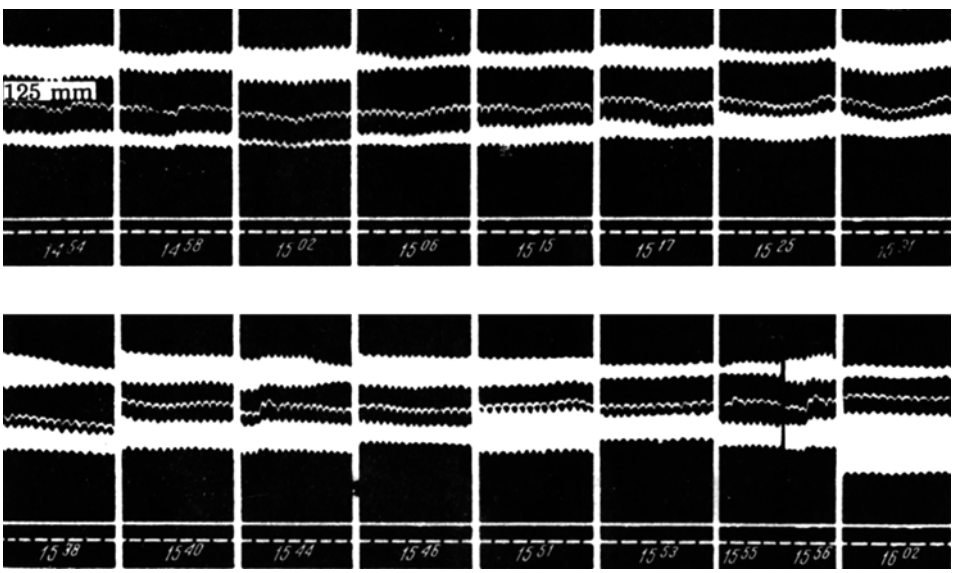


Fig. 3. Periodic fluctuations of the diameter of the internal carotid arteries of a dog weighing 9 kg. Curves, from above downwards: bloodflow in left internal carotid artery; arterial pressure in femoral artery; bloodflow in right carotid artery; time marker (1 sec). Figures below – time of recording (in hours and minutes). Minute volume of bloodflow and arterial pressure, see table. At 15 hr 40 min there was no increase in the general arterial pressure, but the pointer was raised.

cerebral vessels peripheral to it changed their diameter as a result of stimulation of the superior cervical sympathetic ganglia.

The same thing was shown also by other experiments in which serial micrographs of the pial arteries were taken during stimulation of the superior cervical ganglia. Subsequent measurement of their diameter revealed no consistent changes in the diameter of the pial arteries (even in unanesthetized animals), although any constrictor vasomotor influence on these vessels would undoubtedly have been shown up in this way.

In order to be sure that the constrictor effects on the internal carotid arteries were really a manifestation of sympathetic control, in several experiments injections of 0.1 mg/kg ergotamine were made into the internal carotid. As a result the constrictor effect on the internal carotids due to stimulation of the superior cervical ganglion was eliminated (see Fig. 1). The blocking action of ergotamine lasted for 15-30 min, after which the internal carotid arteries could once more be made to contract.

Because the reduction of bloodflow in the internal carotid artery occurring on stimulation of the superior cervical sympathetic ganglion might depend in some measure on constriction of the vessels of the eye*, we carried out control experiments in which the eye on the stimulated side was enucleated. However, under these conditions a reduction of bloodflow still occurred (Fig. 2). We observed the same result when an arterial anastomosis was made between the internal carotid and the internal maxillary artery of dogs.

By use of a modification of a Rein's timer, Shimizu [14] also obtained a constriction of the internal carotid arteries on stimulation of the superior sympathetic ganglia; the vertebral arteries also constricted on stimulation of the stellate ganglia, though the method he used did not enable quantitative results to be obtained nor the degree of sympathetic control over the regional arteries to be measured.

Therefore nervous impulses spreading out from the superior cervical sympathetic ganglia may cause a considerable constriction or even complete spasm of the internal carotid arteries on the same side, and the sensitivity of the neuromuscular apparatus of these vessels to sympathetic influences is approximately the same as that of the extracranial arteries (branches of the external carotid artery).

Periodic fluctuations of the lumen of the internal carotid arteries. In the last few years one of us (G. I. Mchedlishvili) has shown that the pulse pressure in the circle of Willis is extremely small, and that the pressure variations are damped out in the regional arteries; while the general blood pressure remains constant, the arterial pressure in the circle of Willis remains unchanged under normal conditions.

Our researches have revealed new facts concerning the function of the neuromuscular apparatus of the regional cerebral arteries. Simultaneous recording with a photohemotachometer of the blood flow in both internal carotid arteries has shown that there is a periodic fluctuation in their diameter: while one artery dilates the other contracts. This fluctuation of the lumen is shown in Fig. 3, and the values of the bloodflow at different times is given in the table.

Periodic fluctuations and the width of arteries have been described in various organs, and it has been shown that they are associated with the activity of vasoconstrictor nerves [2, 7, 8, 14, 16]. The periodic fluctuations of the lumen of the internal carotid arteries reported here may also result from vasomotor control. Evidence is provided by the fact that the effect does not occur in deep anesthesia, and that it disappears after intravenous injection of 0.1 mg/kg ergotamine.

The effect of the periodic fluctuations of the regional cerebral arteries is to control the rate of bloodflow and the blood pressure in the circle of Willis; because these changes are "reciprocal" in nature (see Fig. 3 and table) the net result may be that these quantities remain unchanged and furnish an uninterrupted blood supply to the brain.

The results we have reported indicate that the internal carotid and vertebral arteries which play such an important part in regulating the cerebral blood supply are under the continuous control of the nervous system, which exerts its effect through the cervical sympathetic ganglia.

However when an investigation was made of the reflex influence from the baroreceptors of the various sinuses

*Long ago Jensen [12] used a primitive stromuhr, and found that after stimulation of the cervical sympathetic region bloodflow in the internal carotid arteries was reduced; he thought the result was caused by constriction of the cerebral arterioles and capillaries. However Tigerstedt [15] doubted whether the effect was due to the cerebral vessels because it might be brought about merely by constriction of the ocular vessels.

Minute Volume of Blood in the Left and Right Internal Carotid Arteries of a Dog

| Time of day | Minute vol. of blood (in $mm^3/sec$) in artery | | Arterial pressure (in mm) |
|---|---|---|---|
| | left | right | |
| 14 hr 54 min. . . . . | 0,93 | 0,55 | 120 |
| 14 " 58 " . . . . . | 0,64 | 0,55 | 125 |
| 15 " 2 " . . . . . | 0,93 | 0,00 | 117 |
| 15 " 6 " . . . . . | 0,64—0,45 | 0,39 | 118 |
| 15 " 15 " . . . . . | 0,55 | 0,64 | 120 |
| 15 " 17 " . . . . . | 0,71 | 0,64 | 120 |
| 15 " 25 " . . . . . | 0,55 | 0,74 | 120 |
| 15 " 31 " . . . . . | 0,87 | 0,64 | 120 |
| 15 " 38 " . . . . . | 0,84—0,55 | 0,71 | 120 |
| 15 " 40 " . . . . . | 0,84 | 0,81 | 120 |
| 15 " 44 " . . . . . | 0,87—0,64 | 0,81 | 120 |
| 15 " 46 " . . . . . | 0,87 | 0,59 | 120 |
| 15 " 51 " . . . . . | 0,78 | 1,01 | 120 |
| 15 " 53 " . . . . . | 0,50 | 0,71 | 120 |
| 15 " 55 " . . . . . | 0,50 | 0,71 | 120 |
| 15 " 56 " . . . . . | 0,78 | 0,84 | 120 |
| 16 " 2 " . . . . . | 0,55 | 1,25 | 120 |

of the brain acting on the regional arteries [6] it was found that the effect was maintained even after complete cervical sympathectomy (from the superior cervical to the stellate ganglia, inclusive). The conclusion is that there are also constrictor reflexes acting on the internal carotid and vertebral arteries, and that they are mediated by the sympathetic cervical trunk (in any case this will apply to those reflexes which originate in receptors within the skull). The fact that there are several sources of innervation of the regional arteries is important physiologically because in the case of damage to some of the pathways, the regulators of cerebral blood pressure are not denervated or deprived of control from the central nervous system.


## SUMMARY

Experiments were made on the internal carotid arteries of dogs and rabbits using Cybulski and Klisiecki's method of photohemotachometry. A considerable constriction of the ipsilateral internal carotid artery followed electrical stimulation (2 v) of the superior cervical sympathetic ganglion; a stronger stimulation (6 v) caused complete closure of the arterial lumen. Ergotamine blocked this effect. Simultaneous recordings of the bloodflow in both internal carotid arteries revealed periodical vasomotor fluctuations of the lumen. The effect appeared to be exerted by sympathetic control over these arteries.


## LITERATURE CITED


1. A. M. Blinova and N. M. Ryzhova, Vestn. AMN SSSR, 1961, No. 5, p. 56.
2. I. M. Zaalishvili, In Book: Collected Works to Mark the 80th Birthday of V. V. Voronin, Tbilisi, 1952, p. 103.
3. B. N. Klosovskii, Cerebral Blood Circulation [in Russian] Moscow, 1951.
4. G. I. Mchedlishvili, Dokl. AN SSSR, 1959, Vol. 124, No. 6, p. 1371.
5. G. I. Mchedlishvili, Transactions of the Institute of Physiology, AN Georgian SSR, 1961, Vol. 12, p. 121.
6. G. I. Mchedlishvili and L. G. Ormotsadze, Byull. éksper. biol., 1962, No. 2, p. 9.
7. M. A. Nutsubidze, Transations of the AN Georgian SSR, 1955, Vol. 16, No. 4, p. 319.
8. R. Chambers. and B. W. Zweifach, Physiol. Rev., 1947, v. 27, p. 436.
9. H. S. Forbes and S. S. Cobb, Brain, 1938, v. 61, p. 221.
10. E. S. Gurdjian, J. E. Webster, F. A. Martin et al., Arch. Neurol. Psychiat., 1958, v. 80, p. 418.
11. Holmqvist, D. H. Ingvar, and B. Siesjö, Acta physiol. scand., 1957, v. 40, p. 146.
12. P. Jensen, Pflüg. Arch. ges. Physiol., 1904, Bd. 103, S. 196.
13. Schiff, Arch. physiol. Heilk., 1854, Bd. 13, S. 523.
14. T. Shimizu, J. Kyoto Pref. med. Univ., 1958, v. 63, p. 603.
15. R. Tigerstedt, Die physiologie des Kreislaufes, Berlin, 1923, Bd. 4.
16. H. C. Wilson, Am. J. Physiol., 1953, v. 174, p. 162.