Carotid flow, intrasinusal pressure, and collateral flow during carotid occlusion

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As a maintenance dose 1.5–3 mg/kg were added at approximately 1-hr intervals. Experiments were interrupted for 10 min after each addition of the anesthetic.

The common carotid artery was exposed at the neck for about 5 cm on both sides. A 2.5 or 3 mm Statham electromagnetic flow probe was placed around each artery. A polyethylene tube was placed in the brachial artery and connected to a strain gage pressure transducer to record systemic arterial pressure. Signals were smoothed out to observe mean flow rate and pressure. For intrasinusal pressure, another polyethylene tube was placed in the lingual artery. No intentional smoothing was done for the intrasinusal pressure. All flows and pressures were recorded with a four-channel pen-writing oscillograph.

To stimulate the cervical sympathetic nerve, the cervical vagosympathetic trunk was isolated from the surrounding tissues and cut. The central end of the trunk was then desheathed. Two bundles were usually found in one sheath and they could be separated from each other by gently pulling each end with forceps. Sympathetic fibers were contained in the thinner bundle: its stimulation on a pair of silver-silver chloride electrodes induced a decrease in carotid flow and dilation of the pupil. Stimulating currents were provided by an electronic pulse generator. Square pulses of 10 v, 5 msec, 1–5/sec were routinely used.

RESULTS

Flow and pressure during carotid occlusion. Effect of unilateral carotid occlusion on contralateral carotid flow and arterial pressure was studied on 16 sides of 8 dogs. In 6 of the 8 dogs intrasinusal pressure was also recorded. One example of the experiment is presented in Fig. 1. During unilateral carotid occlusion, contralateral carotid flow increased by $32.4 \pm 7.9\%$ ($N = 16$), significant at $P < 0.001$. Systemic arterial pressure also increased, but its rate was only $9.8 \pm 4.6\%$ ($N = 16$, significantly smaller than that of carotid flow ($P < 0.001$). After severance of the carotid sinus nerve on one side, occlusion of the ipsilateral common carotid artery induced no appreciable change in arterial pressure ($2.1 \pm 2.34\%$, $N = 8$), but contralateral carotid flow still increased significantly ($26.6 \pm 10.3\%$, $N = 8$, $P < 0.001$) (Fig. 2). Therefore, the main cause of the increase in contralateral carotid flow during unilateral carotid occlusion was due to collateral flow, through the circle of Willis and other communicating vessels to the area originally supplied by the occluded carotid artery.

When the common carotid was occluded, confirming pre-
vious authors' results (1, 9), intrasinusal pressure first dropped precipitously and then recovered partially to reach a plateau (Fig. 1, left). After severance of the carotid sinus nerve on the side of the occlusion, the recovery of intrasinusal pressure during the occlusion was less marked (Fig. 2, right). As seen in Figs. 1 and 2, attenuation of the intrasinusal pulse pressure was much greater than that of the mean pressure during carotid occlusion. Pulse pressure was attenuated by a factor of 0.26 ± SD 0.09 (plateau value against control, mean for six dogs after severance of the ipsilateral sinus nerve), while mean pressure was attenuated only by a factor of 0.74 ± SD 0.02. The plateau value of mean intrasinusal pressure during carotid occlusion was 96.3 ± SD 12.0 mm Hg, above threshold for the baroreceptors.

During occlusion of one common carotid artery, if the contralateral carotid artery was also occluded, intrasinusal pressure dropped further temporarily but soon started rising again and almost recovered to the level of ipsilateral occlusion (Fig. 3). The reflex rise in systemic arterial pressure is more marked during bilateral carotid occlusion than unilateral occlusion (4). The rise in systemic arterial pressure contributed greatly to the recovery of intrasinusal pressure. The recovery of intrasinusal pressure was small after severance of the sinus nerves bilaterally.

Estimation of collateral flow and resistance. During carotid occlusion, the vascular area originally supplied by the carotid artery is drained by collateral flow from other arteries. This situation is schematized in Fig. 4, where several communicating vessels between the bilateral carotid arteries are lumped as one channel with flow resistance $r_c$. The amount of the collateral flow was estimated on the basis of this scheme, assuming Poiseuille's law relating flow, pressure, and resistance. Since the model was crude, this analysis was an approximate one.

In the experiment shown in Fig. 2, since control intrasinusal pressure and left carotid flow were 115 mm Hg and

![FIG. 1. Simultaneous recording of right and left common carotid flow and left brachial artery and left intrasinusal pressure (from top downward). Effects of common carotid occlusion on flow and pressure. First left and then right common carotid was occluded. (10-kg dog.)](image)

![FIG. 2. After severance of left carotid sinus nerve. Otherwise same as in Fig. 1.](image)

![FIG. 3. Effect of bilateral carotid occlusion. Same dog as in Figs. 1 and 2. Bilateral sinus nerves were still intact. Note sizable intrasinusal pressure even during bilateral carotid occlusion.](image)

![FIG. 4. A scheme of bilateral carotid arteries (LC and RC) with their branches and communications. Shaded area is supplied by left common carotid artery during right carotid occlusion. P = systemic arterial pressure; $P_v$ = venous pressure, assumed 0 for simplicity; $P_i$ = right intrasinusal pressure; $r_s$ and $i_s$ = flow resistance and flow in the communications between carotid arteries; $r_x$ and $i_x$ = flow resistance and flow in the communications between the right carotid artery and adjacent arteries other than the left carotid; $R$ = synthetic flow resistance between the carotid sinus area to the central veins.](image)
75 ml/min, respectively, peripheral resistance of the area supplied by the left carotid artery (R in Fig. 4) was 115/75 ≃ 1.5 mm Hg/ml per min. During left carotid occlusion, left intrasinusal pressure fell to 80 mm Hg. Since the left carotid sinus nerve had been severed and the change in vasomotor nerve activity seemed minimal, the systemic arterial pressure remained almost unchanged. If we can assume that R did not change appreciably during this intervention, flow through the vascular area originally supplied by the left carotid artery (i_e + i_R, Fig. 4) may be estimated as 80/1.5 ≃ 53 ml/min. This was the collateral flow from the adjacent arteries. In this instance the percentage of collateral flow to the original carotid flow was 53/75 ≃ 70%. Mean of this percentage in five dogs was 66 ± SD 7%.

In Fig. 2 the increase in right carotid flow during left carotid occlusion was 30 ml/min. This extra amount of flow was a part of the collateral flow to the area originally supplied by the occluded carotid artery (i_e, Fig. 4). Its percent contribution to the total collateral flow was 30/53 ≃ 57%. However, this percentage was variable from animal to animal. The mean with SD for five dogs was 41 ± 15%, range 24-64%.

In the experiment of left carotid occlusion shown in Fig. 2, systemic arterial pressure was 120 mm Hg, while left intrasinusal pressure was 80 mm Hg. Therefore, pressure drop in r_c, resistance in the communicating vessels between the bilateral carotid arteries (Fig. 4), was 40 mm Hg. Since the flow through r_c was i_e = 30 ml/min, r_c = 40/30 ≃ 1.3 mm Hg/ml/min. Mean of r_c for six dogs was 1.5 ± SD 0.8 mm Hg/ml/min.

Effect of sympathetic nerve stimulation. In the experiment reproduced in Fig. 5, effect of stimulation of the left cervical sympathetic nerve on bilateral common carotid flow, systemic arterial pressure, and intrasinusal pressure was observed. The left cervical sympathetic nerve was cut and prepared for stimulation. The left carotid sinus nerve had been severed and the right sinus nerve as well as the right cervical sympathetic nerve was intact. By stimulating the left cervical sympathetic nerve from P to Q in the figure with pulses of 10 v, 5 msec, and 1/sec, the ipsilateral, left carotid flow was reduced to about one-third of the pre-stimulation flow rate. However, the stimulation was without appreciable effect on the contralateral, right carotid flow. Systemic arterial pressure was not affected appreciably, but the left intrasinusal pressure was slightly elevated.

When the stimulation was repeated during occlusion of the left carotid artery (R to S, Fig. 5), right carotid flow, which had been increased about 20% by left carotid occlusion, was now decreased considerably by stimulation of the left cervical sympathetic nerve. It is noteworthy that the intrasinusal pressure, which had dropped from 130 to 85 mm Hg by carotid occlusion, recovered to 120 mm Hg during stimulation of the cervical sympathetic nerve. Intrasinusal pulse pressure was also increased by the stimulation.

The experiment of sympathetic nerve stimulation was done in five dogs. In one exceptional dog, contralateral carotid flow did not decrease appreciably even during occlusion of the common carotid artery of the side of the stimulation. However, the marked rise in intrasinusal pressure on stimulation during ipsilateral carotid occlusion was observed in all five dogs.

DISCUSSION

When the unilateral common carotid artery was occluded, contralateral carotid flow increased about 30%. The major part of the increased flow was estimated to pass to the occluded side through the communicating vessels, as schematically shown in Fig. 4. The communications between the tributaries of the bilateral common carotid arteries through the circle of Willis and other places are known from pressure recordings as well as by dissections of injected specimens (1, 6, 9). Presumably these communications are always open but the system is so balanced that there is no net flow through them when the bilateral carotid arteries are open.
Because of the flow through these and other communications, intrasinusal pressure stayed at a fairly high level during common carotid occlusion. However, attenuation of pulse pressure was much greater than that of mean pressure (ca. 3:1). It is known that decrease in intrasinusal pulse pressure, mean sinus pressure remaining constant, results in a decreased activity of the carotid sinus baroreceptors and, in turn, in a reflex elevation of systemic arterial pressure (2, 7). It is possible that, during carotid occlusion, the decrease in intrasinusal pulse pressure plays a larger part in inducing the pressor reflex than the decrease in mean pressure.

The contralateral common carotid artery is not the sole source of collateral flow; it is obvious from the fact that the intrasinusal pressure was still sizable even during bilateral carotid occlusion (Fig. 3). Systemic arterial pressure rose considerably in the reflex due to decrease in the baroreceptor activity. The elevated systemic pressure was transmitted to the sinus area through the vascular communications.

When an artery draining x% of cardiac output is occluded, systemic arterial pressure will tend to increase about x%; if the artery is a true end artery without any collaterals, cardiac output remains unchanged and a proportionality holds between flow and pressure (5). After severance of the carotid sinus nerve, carotid occlusion does not induce any appreciable increase in systemic arterial pressure because of abundant collaterals in the carotid flow region. The lack in reactive hyperemia after releasing unilateral carotid occlusion is another sign for the abundant collateral flow. Assuming that Poiseuille's law holds good, collateral flow to the occluded carotid area is estimated as about 70% of the original flow.

Electrical stimulation of the cervical sympathetic nerve induced a decrease in ipsilateral carotid flow only: contralateral carotid flow remained almost unchanged (Fig. 5). This is consistent with the general belief that the sympathetic distribution is strictly unilateral, stopping abruptly at the midline (3, 8). However, contralateral carotid flow was decreased by the same stimulation when the ipsilateral carotid artery was occluded. This was due to the presence of collateral flow from the contralateral carotid artery to the side of nerve stimulation during carotid occlusion. The major site of the vasoconstriction on cervical sympathetic stimulation seemed peripheral to the outlets of the collateral paths. The explanation of this situation may be facilitated by a simulated electric circuit, in which blood flow is analogous to electric current, blood pressure to electric potential, and flow resistance to ohmic resistance (Fig. 6). Similar to Fig. 4, rC represents flow resistance in the collateral path between the bilateral carotid areas, P systemic arterial pressure, and Pp intrasinusal pressure. For simplicity venous pressure (Pv) is assumed 0. The flow resistance through collaterals from arteries other than the contralateral carotid artery, such as the vertebral and spinal arteries, is lumped together as rX.

Normally, or without common carotid occlusion, switch S is closed and intrasinusal pressure Pp is almost equal to systemic pressure P. There is then almost no flow through resistances rC and rX. During ipsilateral carotid occlusion, switch S is open and flow passes through rC and rX to reach the sinus and adjacent areas. If the analogy of electricity is valid, intrasinusal pressure may be determined as FR/(r + R), where r is the synthetic resistance of rC and rX in parallel combination. During common carotid occlusion, stimulation of the cervical sympathetic nerve increased intrasinusal pressure considerably (Fig. 5). This fact indicated that the stimulation increased R much more than r. This situation is symbolized in the scheme of Fig. 6 by using the sign of a variable resistor for R. In other words, the sympathetic vasoconstrictors were without appreciable effect on the communicating vessels and the influence of the constrictors was mainly in the blood vessels peripheral to the communicating vessels.

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