Analysis of the Effect of Bilateral Sympathetic Stimulation on Cerebral and Cephalic Blood Flow in the Dog

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SUMMARY Unilateral stimulation of the cervical sympathetic in dogs had no effect on cerebral blood flow (CBF) measured by the venous outflow technique. Since this technique measured CBF from both cerebral hemispheres, small changes induced by unilateral stimulation could have been masked by a large constant CBF measured from the contralateral hemisphere. To test this possibility the effect of simultaneous bilateral sympathetic stimulation was studied when the dog was breathing either normal air or a gas mixture of 10% CO₂. During normocapnia, no changes in CBF occurred; during hypercapnia CBF increased 19% following passively the increase in blood pressure. These data indicate that bilateral stimulation of extracranial sympathetic nerves does not exert a significant effect on CBF. We show mathematically and experimentally that unoccluded anastomoses will cause CBF to appear to decrease in response to sympathetic stimulation. This may explain why others have observed changes in CBF during sympathetic stimulation.

THE ROLE of the sympathetic innervation on the control of the cerebral-vasculature still is a controversial subject. Several investigators have reported an effect of sympathetic stimulation on the cerebral vasculature. Results from this laboratory suggest that stimulation of the stellate ganglion in the dog has no significant effect on cerebral blood flow. Since this preparation measured blood flow from both hemispheres, small blood flow changes during unilateral stimulation could have been masked by constant blood flow from the contralateral hemisphere. Furthermore, the major sympathetic innervation affects the cephalic extracerebral arteries leading to the brain and these vessels have extensive interconnecting anastomoses. The extent of this collateral circulation is indicated by studies showing that complete occlusion of one carotid artery produces only a small decrease in pressure in the homolateral pial arteries. Thus, unilateral vasoconstriction of extracerebral arteries might not cause a reduction in cerebral blood flow due to an increased flow through anastomoses from arteries in the non-innervated hemisphere.

These reservations can be resolved in large part by a study of the effect of bilateral stimulation on cerebral blood flow. However, the effect of neurogenic vasoconstriction of cephalic extracerebral arteries on cerebral blood flow could be greatly reduced by autoregulatory vasodilation in the noninnervated portion of the cerebral arteries. Therefore, in the present study the effect of bilateral sympathetic stimulation on cerebral blood flow was tested during normocapnia and also under conditions of hypercapnia sufficient to impair autoregulation.

The effects of sympathetic stimulation on CBF found by other investigators may be due to extracerebral contamination in the flow measurement path brought about by anastomotic communications between the intracranial and extracranial beds. Therefore, the effect of stimulation was studied both before and after occlusion of the intervenous communications between intra- and extracranial circulation. Finally, an analysis is lacking of the interactions between flow through a cognate autoregulated bed and flows through communicating nonautoregulated beds. We will show analytically that if anastomoses are present then CBF may appear to vary with changes in perfusion pressure or extracranial bed flow resistance, even when CBF is perfectly autoregulated.

Methods

Mongrel dogs weighing 16–24 kg were anesthetized with either sodium pentobarbital (30 mg/kg) or chloralose (80 mg/kg) and xylazine (0.5 mg/kg). Sodium heparin (500 units/kg) was administered as anticoagulant. The animals were intubated and mechanically ventilated with a Harvard respirator. End-tidal CO₂ was continuously monitored with a Beckman LB-1 gas analyzer and was maintained at 4–5%. In 10 experiments hypercapnia was induced by increasing end-tidal CO₂ to 10%. Arterial PCO₂ and pH were measured with a Radiometer blood gas analyzer. Body temperature was maintained constant with a heating pad.

The right and left stellate ganglia were approached through a single midternal incision. Two Teflon insulated stainless steel wires were tied around each ganglion and connected to a Grass SD9 stimulator. The bare end of each wire acted as a stimulating electrode. Stimuli were routinely applied at 5 volts, 15 Hz, 5 msec pulse duration. Marked pupillary dilation and a bilateral decrease in common carotid flow were taken as evidence of effective bilateral stellate stimulation.

Cerebral blood flow was measured by cannulating the confluens sinum and diverting cerebral venous outflow through an electromagnetic flowmeter to a femoral vein, as previously described. Communications between the intracranial and extracranial venous circulations were essentially eliminated by occlusion of the transverse sinuses with bone wax. Right and left common carotid artery flows were measured with noncannulating flow probes. Systemic blood pressure was measured with a Statham P-23 transducer via a catheter placed in the femoral artery.

The pressor effect of stellate stimulation was eliminated by controlled arterial hemorrhage in some experiments.
Hemorrhage from one femoral artery was adequate to prevent an increase in blood pressure during stimulation.

The percent change between stimulation (S) and control (C) values was calculated as 100 × (S - C)/C. The five animal average percent change was calculated, and a Student’s t-test was performed on the null hypothesis that a change had not occurred.

Results

Throughout all experiments following ganglionic stimulation a highly significant \( p < 0.01 \) decrease averaging at least 50% was observed in the left and right carotid blood flows (tables 1, 2) as well as marked pupillary dilation indicating adequate bilateral sympathetic stimulation. Mean arterial pressure was significantly increased due to stimulation of the stellate cardiac nerves. Responses to stellate stimulation were the same during either pentobarbital or chloralose and sernyan anesthesia.

Bilateral Stimulation During Normocapnia

The effects of bilateral stellate stimulation during normocapnia are shown in table 1. Bilateral stimulation caused an 18%, \( SE = 5.8, p < 0.05 \) increase in blood pressure and a 13%, \( SE = 3.0, p < 0.01 \) decrease in cerebral conductance. Responses to stellate stimulation were the same during either pentobarbital or chloralose and sernyan anesthesia.

During constant arterial blood pressure no significant change was observed in cerebral blood flow (—1.7%, \( SE = 1.7 \)) between control and stimulation.

Bilateral Stimulation During Impaired Autoregulation

During hypercapnia (table 2) bilateral stimulation caused a 10.6%, \( SE = 2.9, p < 0.05 \) increase in cerebral blood flow and a 20.5%, \( SE = 4.7, p < 0.01 \) increase in arterial blood pressure. A significant \( p < 0.05 \) decrease was found in cerebral conductance (—7.7%, \( SE = 3.4 \)). However, during constant systemic blood pressure and hypercapnia, no significant change occurred in either cerebral blood flow or conductance (+2.3%, \( SE = 2.7 \)).

Stellate Stimulation Before and After Occlusion of the Cerebral Extracranial Venous Communications

The effect of communications between intra- and extracranial venous circulations was tested by stimulation before and after occlusion of the two transverse sinuses. Before transverse sinus occlusion, unilateral stimulation produced a decrease in venous outflow (fig. 1A). This effect was markedly enhanced by bilateral stellate stimulation (fig. 1B). However, when the transverse sinuses were occluded, no reduction of venous outflow occurred during bilateral stimulation (fig. 1C).

Discussion

It has been shown that fluorescence in the vertebrobasilar region disappears after sectioning of the stellate ganglia, and that of the carotid arterial tree after sectioning the superior cervical ganglia. Bilateral stimulation of both stellate ganglia should therefore encompass all of the cerebral}

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\begin{array}{|c|c|c|c|c|c|}
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\text{DOG} & \text{FLOW (ml/min)} & \text{CEREBRAL} & \text{BLOOD} & \text{PRESSURE} \\
& \text{CAROTID} & \text{C} & \text{S} & \text{C} & \text{S} & \text{C} & \text{S} \\
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\text{C} & \text{S} & \text{C} & \text{S} & \text{C} & \text{S} & \text{C} & \text{S} \\
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34 & 40 & 10 & 50 & 25 & 23 & 23 & 120 & 135 \\
37 & 31 & 6 & 29 & 9 & 15 & 15 & 95 & 100 \\
45 & 44 & 14 & 42 & 17 & 26 & 26 & 100 & 110 \\
47 & 20 & 12 & 23 & 10 & 27 & 29 & 80 & 110 \\
52 & 20 & 4 & 18 & 5 & 17 & 17 & 80 & 100 \\
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\text{During constant systemic blood pressure} & \text{C} & \text{S} & \text{C} & \text{S} & \text{C} & \text{S} & \text{C} & \text{S} \\
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44 & 47 & 8 & 32 & 7 & 17 & 17 & 95 & 95 \\
50 & 52 & 10 & 50 & 10 & 23 & 23 & 75 & 75 \\
52 & 23 & 3 & 24 & 3 & 24 & 24 & 90 & 90 \\
58 & 60 & 10 & 55 & 10 & 23 & 21 & 105 & 105 \\
59 & 38 & 10 & 30 & 10 & 19 & 19 & 95 & 95 \\
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vascular sympathetic nerves. The results of the present study show that cerebral blood flow is not affected when these sympathetics are stimulated under normocapnic conditions. Furthermore, when autoregulation in the cerebral circulation was impaired by inducing hypercapnia cerebral blood flow was still unaffected by sympathetic stimulation.

The apparent sympathetic induced increase in cerebral blood flow observed during hypercapnia was secondary to an

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**TABLE 2**

Responses to bilateral stellate stimulation during hypercapnia

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<thead>
<tr>
<th>DOG PQ</th>
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increase in arterial pressure, since there was no change in flow when arterial pressure was held constant during stimulation. The barely significant decrease in cerebral conductance when blood pressure was not held constant indicates that autoregulation was impaired but not abolished. During normocapnia the pressor effects of stellate stimulation did not result in an increase in cerebral blood flow, owing to an autoregulatory decrease in cerebral conductance. The decrease in conductance was an autoregulatory response to the pressor effect since neither conductance nor cerebral blood flow changed when systemic blood pressure was held constant.

The present study demonstrates a problem which must be considered when attempting to measure blood flow through an autoregulated bed (cognate) in the presence of a collateral bed. The results of stellate stimulation before and after occlusion of extracranial communication with the cerebral venous outflow shows that a large error can exist when there is failure to isolate the venous systems of the cerebral from the collateral extracranial bed. Before occlusion of the communication, sympathetic induced constriction of the extracranial circulation produced a large reduction in confluence sinum outflow (figs. 1A, 1B). This large reduction was probably due not only to reduction of extracranial blood flow which drained via the confluens, but also to diversion of some cerebral venous flow from the confluens outlet to the transverse sinus outlets, owing to the reduction of extracranial venous pressure induced by extracranial arterial constriction.

A simplified schematic of blood flow in the head relative to venous outflow measurements is shown in figure 2. This approximation is, however, sufficient to illustrate the pitfalls in interpretation of cerebral blood flow measurements that are created by the presence of anastomoses between intra- and extracranial circulation. In particular, an answer is sought to the following question: If one assumes that autoregulation of cerebral blood is perfect and that this blood flow is constant, then under what conditions is one likely to conclude that a change in cerebral blood flow has occurred when, in fact, cerebral blood flow has not changed?

In the venous outflow measurement technique measured flow, $Q_v$, will always be equal to cerebral blood flow, $Q_b$, if venous-venous anastomotic resistance, $R_{vv}$, is infinite (zero conductance). Then the only possible path for $Q_b$ is through $R_m$, the flow measuring circuit resistance.

When anastomoses are present the accuracy of $Q_v$ as an estimate of $Q_b$ is affected by the values of $R_{vv}$, $R_{eb}$, $R_e$, and $R_m$. Application of mass conservation (Kirchoff's law) to this flow circuit yields:

$$Q_v = mP + bQ_b$$

where

$$P = \text{perfusion pressure}$$

$$m = \frac{R_{ev}/[(R_{ev} + R_{eb})(R_{vv} + R_m) + R_{eb}Rev]}{R_{ev} + R_{eb}}$$

the slope of a straight line with intercept $bQ_b$ where

$$b = \frac{R_{ev}(R_{eb} + R_m) + R_{eb}Reb}{(R_{ev} + R_{eb})(R_{vv} + R_m) + R_{eb}Rev}$$

This shows that the apparent measured cerebral blood flow, $Q_v$, is dependent on pressure $P_v$, and extracranial bed resistance, $R_{eb}$, as well as $R_m$, $R_{ev}$, and $R_{vv}$. These effects are shown in figure 3. Note that even though $Q_b$ is completely pressure independent, $Q_v$ exhibits a strong perfusion pressure dependence over a wide range of values for $R_{eb}$. This dependence decreases with increasing $R_{eb}$. At a perfusion pressure of 100 mmHg, increasing $R_{eb}$ from 1.0 to 4.0 PRU causes $Q_v$ to decrease from 50 ml/min to 27 ml/min. Therefore, sympathetically induced vasoconstriction altering $R_{eb}$ will cause changes in $Q_v$, suggesting falsely that $Q_b$ has changed.

Figures 1A and 1B show that if the anastomoses are not occluded then $Q_v$ decreases greatly during sympathetic stimulation. The large decrease in common carotid flows indicates that $R_{eb}$ has increased. Occluding the anastomoses increases $R_{ev}$ and causes $b \approx 1$ and $m \approx 0$ in equation 1. In this case $Q_v = Q_b$ and $Q_v$ is unaffected by changes in $P$, $R_{eb}$, $R_m$, or $R_{vv}$. Figure 1C shows that sympathetically induced vasoconstriction does not affect $Q_v$ after the anastomoses are occluded.

![Figure 2. Simplified schematic of cerebral venous outflow measurement. Flowmeter location is indicated by F. $P_a$ = systemic arterial pressure. $P_v$ = systemic venous pressure. $R_{eb}$ = extracranial bed resistance. $R_{ev}$ = extracranial venous outflow resistance. $R_{vv}$ = resistance of venous-venous anastomosis. $R_m$ = intracranial venous outflow resistance. $Q_v$ = flow through $R_m$. $Q_b$ = cerebral blood flow.](image)

![Figure 3. Measured blood flow as a function of perfusion pressure for various values of extracranial bed resistance and $R_{vv} = R_{ev} = R_m = 0.1$PRU.](image)
On the basis of evidence suggesting the existence of a dense sympathetic innervation to the cerebral extraparenchymal arteries along with an almost complete lack of innervation to the intraparenchymal arteries, Harper proposed that the cerebral circulation might be under both neural and metabolic control. According to this hypothesis metabolic autoregulation in the intraparenchymal vessels would normally compensate for neurogenic vasoconstriction of the extraparenchymal arteries, resulting in no change in total vascular resistance of the brain. The lack of effect of sympathetic stimulation on cerebral blood flow during normocapnia obtained in the present study could conceivably be explained in terms of this model. During hypercapnia cerebral blood flow was dependent on perfusion pressure since cerebral blood flow increased with the sympathetic induced increase in systemic arterial blood pressure (table 2). But when arterial pressure was held constant during hypercapnia, stellate ganglia stimulation should have produced a decrease in cerebral blood flow due to a neurogenically induced decrease in extraparenchymal resistance. The findings of no change in cerebral blood flow with stellate stimulation during hypercapnia when arterial pressure was held constant shows that there was little, if any, neurogenic constriction of the extraparenchymal arteries. The possibility that hypercapnia itself might in some way prevent the sympathetic vasoconstriction seems unlikely.

The negative results obtained when using the venous outflow technique have been criticized on the grounds that surgery altered the sensitivity of the cerebral vasculature to sympathetic stimulation. However, the preparation is highly responsive to increases in PaCO₂ (compare tables 1 and 2) and to changes in perfusion pressure, two accepted tests of integrity of cerebrovascular responses. Some autoregulatory capability was maintained even during hypercapnia. The claim that surgery altered sensitivity has been based, in part, on the argument that the blood flow values obtained with our preparation are low compared with total CBF obtained by other methods. Since the venous outflow technique only measures the CBF draining through the dorsal and straight sinuses we do not find our low values surprising. When these values are corrected for the fraction of the brain served by the dorsal and straight sinuses the total calculated CBF is within the range of values found by other investigators. Furthermore, it would appear that extensive blood trauma increases rather than decreases cerebral blood flow. Preliminary results from other experiments using this preparation show that vasoconstriction in the extraparenchymal cerebral vasculature could be obtained by intrarterial injection of serotonin, which produced a reduction of blood flow during hypercapnia although not during normocapnia. This suggests that sympathetic stimulation was not an adequate stimulus.

Studies on isolated vessels suggest that at least part of the explanation for the lack of effects of the sympathetics on cerebral hemodynamics may be vascular insensitivity to the norepinephrine neurotransmitter. Both found that the internal carotid arteries were much less sensitive to norepinephrine than mesenteric arteries of similar size, and that many cerebral vessels were completely insensitive to norepinephrine. Similar results have been obtained with an isolated middle cerebral artery, and with topical application of norepinephrine to pial arteries. In addition to decreased responsiveness of cerebral arteries to norepinephrine, Toda and Fujita found that these vessels did not have a characteristic contractile response to transmural electrical stimulation as did vessels from the other regions. Although electrical stimulations of long pulse duration did elicit a contractile effect, this effect was not mediated by neurotransmitter release since it could not be blocked with tetrodotoxin. These findings lead Toda and Fujita to conclude that the sympathetic nerves do not play a significant role in the regulation of resistance in the cerebral extraparenchymal arteries.

In conclusion, it has been shown that bilateral sympathetic stimulation does not significantly affect cerebral conductance during either normocapnia or hypercapnia. An analysis of the effects of venous-venous communications on the accuracy of direct flow measurement techniques shows that these communications may cause autoregulatory capability to be underestimated, extracebral flow resistance changes to appear to have an effect on CBF, and CBF to be either under or overestimated. The effects of sympathetic stimulation on CBF found by other investigators may be due in some cases to the presence of unoccluded communications.

Acknowledgment

The authors gratefully acknowledge the excellent technical assistance of Mr. William Brooks.

References

Thromboendarterectomy for Total Occlusion of the Internal Carotid Artery: A Reappraisal of Risks, Success Rate and Potential Benefits

T. KUSUNOKI, M.D., D. W. ROWED, M.D., F.R.C.S.(C), C. H. TATOR, M.D., PH.D., F.R.C.S.(C), AND W. M. LOUGHEED, M.D., F.R.C.S.(C)

SUMMARY Forty consecutive patients undergoing thromboendarterectomy for total internal carotid artery occlusion were studied in an attempt to determine a) whether careful case selection could be expected to reduce future postoperative mortality and morbidity, b) whether the achieved patency rate justified early operation and c) whether patients in whom patency was restored and maintained had a better long-term prognosis.

CAROTID ENDARTERECTOMY effectively eliminates a source of emboli or improves blood flow in atherosclerotic stenoses of the internal carotid artery. Thromboendarterectomy for total occlusion of the internal carotid artery, on the other hand, remains a controversial subject.

Although the earlier literature was less encouraging, more recent studies have shown that patency of the internal carotid artery can frequently be restored by thromboendarterectomy. Most authors report a success rate of approximately 40%. Restoration of patency has been reported in as few as 20%, and as many as 64% of operated patients. The earlier the operation is performed after total occlusion is believed to have occurred, the higher is the rate of postoperative patency. For example, 100% of occluded internal carotid arteries have been successfully reopened within 6 hours of presumed occlusion, 90% within 48 hours, and 70% within 72 hours.

Postoperative mortality and morbidity have been low. It is well recognized that mortality resulting from carotid endarterectomy is much higher in patients with recent cerebral infarction, who are neurologically unstable. This has been true for patients with complete occlusion, as well as stenosis, with virtually all operative deaths occurring in drowsy patients with recent cerebral infarction. In series where case selection has excluded such ill patients, the mortality has been low, and in some series there has been no postoperative mortality.

The results show that a group of patients can be selected that will have low postoperative mortality and morbidity. The success rate for restoration of blood flow is high, particularly if the operation is performed soon after occlusion. The long-term prognosis in patients in whom patency of the internal carotid artery is restored and maintained appears to be better than in those with persistent occlusion of the carotid artery.

Long-term follow up of patients operated on successfully for total internal carotid artery occlusion indicates that the incidence of recurrent neurological symptoms is low. This data must be evaluated by comparison with the natural history of internal carotid artery occlusion in untreated survivors. Immediate mortality and morbidity from cerebral infarction following internal carotid artery occlusion are high. Survivors, however, have been shown, by some authors, to have relatively low late mortality and morbidity rates from cerebrovascular causes, even when compared with a matched group of patients with carotid stenosis. Patients who reocclude the internal carotid artery after successful thromboendarterectomy have also been shown to have a low rate (4%) of recurrent neurological symptoms. There is dissent regarding the natural history, however, and other authors have found that total occlusion is less benign, with 27% of patients dying from repeated cerebral infarction.

A further problem relating to assessment of the efficacy of thromboendarterectomy for total occlusion of the internal carotid artery is the likelihood of reocclusion after successful operation. Most authors (see above) have not shown results of late follow up angiography. It has been shown that almost 50% of reopened vessels reocclude.

With the above problems in mind, we re-examined the results of thromboendarterectomy for total occlusion of the internal carotid artery in our own institution. Applying more recently evolved criteria for operative risk, we wished to determine if better case selection would result in decreased operative mortality and morbidity. We wished to see whether the long-term success rate of maintaining patency,
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