Effects of Decreasing Arterial Blood Pressure on Cerebral Blood Flow in the Baboon

INFLUENCE OF THE SYMPATHETIC NERVOUS SYSTEM

By William Fitch, Eric T. MacKenzie, and A. Murray Harper

ABSTRACT

The influence of the sympathetic nervous system on the cerebral circulatory response to graded reductions in mean arterial blood pressure was studied in anesthetized baboons. Cerebral blood flow was measured by the $^{133}$Xe clearance method, and arterial blood pressure was decreased by controlled hemorrhage. In normal baboons, the constancy of cerebral blood flow was maintained until mean arterial blood pressure was approximately 65% of the base-line value; thereafter, cerebral blood flow decreased when arterial blood pressure was reduced. Superior cervical sympathectomy of 2-3 weeks duration did not affect the normal response. In contrast, both acute surgical sympathectomy (cervical trunk division) and $\alpha$-receptor blockade (1.5 mg/kg of phenoxybenzamine) enhanced the maintenance of cerebral blood flow in the face of hemorrhagic hypotension in that cerebral blood flow did not decrease until mean arterial blood pressure was approximately 35% of the base-line value. The results indicate that the sympathetic nervous system is not involved in the maintenance of cerebral blood flow in the face of a fall in arterial blood pressure. Indeed, the implication is that the sympathicoadrenal discharge accompanying hemorrhagic hypotension is detrimental to, rather than responsible for, cerebral autoregulation.

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Constancy of cerebral blood flow is maintained in the face of moderate variations in systemic arterial blood pressure (1-4), but the nature of this phenomenon is a matter of some debate. One view is that this mechanism is intrinsic. According to this view, cerebral blood flow is regulated by tissue metabolites (5, 6) or by a myogenic, Bayliss reflex (7-10). If either of these hypotheses is correct, then the blood pressure-flow relationship is a result of autoregulation. The other major view is that the characteristic blood pressure-flow relationship of the cerebral circulation is controlled or modified by the extrinsic innervation of the cerebral vasculature—the neurogenic hypothesis (4, 11, 12).

Some support for the extrinsic mechanism has been taken from morphological studies. Both fluorescent and electron microscopy have confirmed a dual adrenergic and cholinergic innervation of the cerebral circulation (13-15). Sympathetic nerves are the more abundant. The large arteries at the base of the brain receive the densest innervation and the pial vessels are moderately innervated, whereas autonomic nerves have rarely been noted in proximity to the intracerebral arterioles. However, under conditions of normocapnia and normotension, the largest fraction of the total vascular resistance in the brain is located in the parenchymal arteriolar vessels (16, 17).

The present study was undertaken to investigate the effects of surgical (acute and chronic) cervical sympathectomy and of $\alpha$-receptor blockade (phenoxybenzamine) on the cerebral pressure-flow relationship existing during hypotension induced by hemorrhage.

Methods

This study was carried out on 31 young baboons (Papio cynocephalus or Papio anubis) weighing 8-12 kg. The baboons were anesthetized with phencyclidine (12 mg, im) and sodium thiopental (7.5 mg/kg, iv). They were then intubated and connected to an intermittent positive-pressure respiratory pump (Starling) which delivered a mixture of 75% N$_2$O-25% O$_2$ in open circuit. Phencyclidine (2 mg, im) and suxamethonium chloride (100 mg, im) were administered at 30-minute intervals. Body temperature was controlled (36-38°C) by a heating lamp.

The scalp and the temporal muscle were removed from the right side of the cranium to ensure that no radioactivity was detected from extracranial tissues. The right common carotid artery and its branches were then exposed. All of the branches of the right external carotid artery were ligated except the linguofacial trunk, which was cannulated with a fine polyethylene catheter.

Cerebral blood flow was measured by the intracarotid $^{133}$Xe injection method and calculated by the height/area equation (18). A heavily collimated 2.5-cm scintillation crystal was placed directly over the exposed skull and angled in such a way that there was no possibility o
counting radioactivity from the surrounding tissues of the face and the neck. The scintillation crystal was connected to a ratemeter, a scaler, and a direct-writing recorder. The pulse-height analyzer was set at 81 kev with a gate of ±10%, i.e., with a lower limit of 73 kev. This setting effectively prevented any recording of Compton scatter. For each measurement of cerebral blood flow, 0.4-0.8 mc of \(^{133}\)Xe, dissolved in approximately 0.5 ml of saline, was injected into the internal carotid artery via the catheter in the linguofacial trunk. The recording was followed for 10 minutes on each occasion.

The abdominal aorta was catheterized via the left femoral artery, and arterial blood pressure was monitored with a Statham strain-gauge transducer. During the operative procedure only, a slow intravenous infusion of normal saline was administered via a cannula in the right femoral vein. Arterial carbon dioxide tension (Pco\(_2\)) was measured frequently, using a direct-reading electrode system (Radiometer or Corning EEL). In each investigation, ventilation was controlled, and the minute volume was adjusted to maintain an arterial Pco\(_2\) of approximately 10 mm Hg. This procedure took approximately 10-15 minutes to complete. Once the desired arterial blood pressure had been obtained, the pressure was held constant for 15-20 minutes. Cerebral blood flow (0.4-0.8 mc of \(^{133}\)Xe, dissolved in approximately 40 mm Hg. Arterial blood pressure was lowered by bleeding the baboons into a reservoir, which was heparinized and kept at 37°C, from a wide-bore catheter inserted in the right femoral artery. The reservoir was connected to a sphygmomanometer and could be held at any desired pressure. The baboons were heparinized during the experiments. Five groups of baboons were studied.

GROUP 1: HEMORRHAGIC HYPOTENSION ALONE

In ten baboons, stepwise reductions in mean arterial blood pressure were obtained by the intermittent withdrawal of blood. Sufficient blood was removed on each occasion to lower mean arterial blood pressure by approximately 10 mm Hg. This procedure took approximately 10-15 minutes to complete. Once the desired arterial blood pressure had been obtained, the pressure was held constant for 15-20 minutes. Cerebral blood flow was determined after each step reduction in pressure. The same protocol was followed in the other four groups.

GROUP 2: HEMORRHAGIC HYPOTENSION PLUS CHRONIC CERVICAL SYMPATHECTOMY

In five baboons, the right cervical sympathetic trunk was divided in the neck 1 cm below the superior cervical ganglion. After 2-3 weeks, the baboons were subjected to a stepwise hemorrhage as was done in group 1.

GROUP 3: HEMORRHAGIC HYPOTENSION PLUS ACUTE CERVICAL SYMPATHECTOMY

In six baboons, hemorrhagic hypotension was induced within 60 minutes of the division of the cervical sympathetic trunk on the right side.

GROUP 4: HEMORRHAGIC HYPOTENSION PLUS PHENOXYBENZAMINE

In five baboons, the \(\alpha\)-receptor blocking agent phenoxybenzamine was given intravenously at a dose of 1.5 mg/kg. The baboons were then subjected to stepwise hemorrhage.

GROUP 5: HEMORRHAGIC HYPOTENSION PLUS RETRANSFUSION

In five baboons, an attempt was made to reproduce the protocol used by James et al. (4) by bleeding the baboons (in four stages) until arterial blood pressures between 30 and 40 mm Hg were established, retransfusing the withdrawn blood, and then repeating the hemorrhage. However, in contradistinction to the study by James et al. (4), the cervical sympathetic trunk was not divided after the retransfusion. In each baboon, the period of time at the lowest arterial blood pressure was limited to 20 minutes.

TREATMENT OF RESULTS

Each baboon’s resting cerebral blood flow and resting mean arterial blood pressure were used as its own control values and expressed as 100%. Changes in arterial blood pressure and cerebral blood flow in each baboon were expressed as a percent of its own control values. For ease of analysis, the blood flow results in each group of baboons were meaned at 10% intervals of the resting arterial blood pressure (for instance, 79-70 and 69-60% of base-line arterial blood pressure). Student’s \(t\)-test was used for the statistical comparisons. The only exception to this procedure was in group 5 for which absolute values were used, since two separate hypotensive episodes were carried out in each baboon.

Results

Table 1 shows the base-line values obtained in each of the first four groups studied. The groups’ mean control values for cerebral blood flow ranged from 44 to 54 ml/100 g min\(^{-1}\) (average 49 ml/100 g min\(^{-1}\)). These values are comparable to those from other studies using the same method of measuring cerebral blood flow and similar conditions of anesthesia (19, 20). The groups’ mean arterial blood pressure (MABP) and cerebral blood flow (CBF), arterial carbon dioxide tension (Pco\(_2\)), and pH in Groups 1-4

<table>
<thead>
<tr>
<th>Group 1</th>
<th>Group 2</th>
<th>Group 3</th>
<th>Group 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>MABP (mm Hg)</td>
<td>92 ± 7</td>
<td>97 ± 5</td>
<td>104 ± 14</td>
</tr>
<tr>
<td>CBF (ml/100 g min(^{-1}))</td>
<td>52 ± 11</td>
<td>54 ± 11</td>
<td>45 ± 6</td>
</tr>
<tr>
<td>Pco(_2) (mm Hg)</td>
<td>39.8 ± 1.8</td>
<td>40.3 ± 1.8</td>
<td>40.6 ± 1.2</td>
</tr>
<tr>
<td>pH</td>
<td>7.41 ± 0.03</td>
<td>7.41 ± 0.05</td>
<td>7.40 ± 0.03</td>
</tr>
</tbody>
</table>

All values are means ± SD.

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pressures ranged from 91 to 104 mm Hg (average 96 mm Hg). Arterial PCO₂ was consistently held at 40 mm Hg with a maximum standard deviation of 1.8 mm Hg. In each group, there was a decrease in arterial pH on bleeding. The decrease in pH ranged from 0.19 units in group 1 to 0.16 units in group 4. There were no significant differences in the degrees of acidosis which developed in any of the groups studied.

GROUP 1: HEMORRHAGIC HYPOTENSION ALONE

Prior to the induction of hypotension, base-line values for mean arterial blood pressure ranged from 77 to 103 mm Hg (mean ± SD 92 ± 7 mm Hg) in the individual baboons. At normal levels of PCO₂, mean cerebral blood flow ranged from 40 to 70 ml/100 g min⁻¹ (mean ± SD 52 ± 11 ml/100 g min⁻¹). Following the induction of hypotension, mean arterial blood pressure could be reduced to approximately 65-70% of the initial value before there was any decrease in cerebral blood flow, but at mean arterial blood pressures below this value cerebral blood flow was pressure dependent (Fig. 1).

GROUP 2: HEMORRHAGIC HYPOTENSION PLUS CHRONIC CERVICAL SYMPATHECTOMY

The results obtained in this group as mean arterial blood pressure was decreased progressively were not significantly different from those observed in the baboons subjected to hemorrhagic hypotension alone (group 1) (Fig. 1).

GROUP 3: HEMORRHAGIC HYPOTENSION PLUS ACUTE CERVICAL SYMPATHECTOMY

In this group of baboons, the pressure-flow plateau persisted to lower levels of mean arterial blood pressure (Fig. 2). Cerebral blood flow remained relatively constant until a mean arterial blood pressure of approximately 40% of the initial value had been reached. At mean arterial blood pressures of 55% and 45% of the initial value, cerebral blood flow was significantly greater (P < 0.05 and P < 0.001, respectively) in the baboons subjected to hemorrhage plus acute surgical sympathectomy than it was in the baboons subjected to hemorrhage alone.

GROUP 4: HEMORRHAGIC HYPOTENSION PLUS PHENOXYBENZAMINE

The administration of phenoxybenzamine (1.5 mg/kg) reduced mean arterial blood pressure, in its own right, from 91 ± 15 (SD) mm Hg to 68 ± 11 mm Hg. Thereafter, the five baboons were subjected to a stepwise hemorrhage.

As did the baboons subjected to an acute surgical sympathectomy, this group of baboons showed a pressure-flow plateau that persisted to levels of mean arterial blood pressure lower than was the case in the baboons subjected to hemorrhagic hypotension alone (Fig. 3) or in those subjected to chronic cervical sympathectomy. Cerebral blood flow remained relatively steady despite the progressive changes in arterial blood pressure until a mean arterial blood pressure of 35-40% of the initial value had been reached. In fact, cerebral blood flow was significantly greater in this group...
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CBF (H/A) % CONTROL

120-1
100-
80-
60-
40-
20-
0-

FIGURE 3

Effect of decreasing mean arterial blood pressure on mean cerebral blood flow (CBF) in the baboons subjected to hemorrhagic hypotension alone (solid circles) and those subjected to hemorrhagic hypotension following the administration of phenoxybenzamine (1.5 mg/kg) (open squares). Cerebral blood flow was significantly greater in the phenoxybenzamine-treated baboons at all mean arterial blood pressures less than 55% of the baseline value (one asterisk = \( P < 0.05 \), and two asterisks = \( P < 0.01 \)). Values are means ± SE.

than it was in those baboons subjected to hemorrhage alone at mean arterial blood pressures of less than 55% of the base-line value (\( P < 0.05 \)).

Figure 4 summarizes the findings in these four groups of experiments and shows the difference found between the baboons subjected to hemorrhagic hypotension alone and hemorrhage plus chronic sympathectomy on the one hand and the baboons subjected to hemorrhage plus either acute surgical or chemical sympathectomy on the other.

GROUP 5: HEMORRHAGIC HYPOTENSION PLUS RETRANSFUSION

First Hemorrhage.—As mean arterial blood pressure was reduced in steps from 86 ± 9 (sd) mm Hg to an average of 31 ± 1 mm Hg (Fig. 5), mean cerebral blood flow changed in a manner similar to that observed in the baboons previously subjected to hemorrhagic hypotension alone. Although there were only four steps in the arterial blood pressure decrease in this particular group of baboons, the pressure-flow plateau appeared to persist to levels of mean arterial blood pressure similar to those observed in the other baboons subjected to hemorrhage alone, namely to 65–70% of the initial value.

Second Hemorrhage.—The mean arterial blood pressure was held at the level gained following retransfusion for 20 minutes prior to the measurement of cerebral blood flow. Following the retransfusion of the blood removed during the first period of blood loss, mean cerebral blood flow was significantly greater than the value obtained at the start of the first period of hypotension (Fig. 5). The base-line arterial PCO₂ was 40.1 ± 2.3 (sd) mm Hg before retransfusion and 41.4 ± 1.3 mm Hg thereafter. This small and insignificant (\( P > 0.20 \)) rise in PCO₂ is not great enough to explain the very considerable rise in cerebral blood flow following retransfusion.

CBF (H/A) ml/100g/min

ARTERIAL PRESSURE (MEAN) mmHg

FIGURE 5

Comparison of the effect of decreasing mean arterial blood pressure on mean cerebral blood flow (CBF) during the first period of hemorrhage (solid circles) and the second period of hemorrhage, i.e., following retransfusion (solid diamonds). Cerebral blood flow was significantly higher at each level of mean arterial blood pressure except the lowest level after retransfusion (one asterisk = \( P < 0.05 \), two asterisks = \( P < 0.01 \), and three asterisks = \( P < 0.001 \)). Values are means ± SE.
As the mean arterial blood pressure was decreased in steps for the second time, at arterial blood pressure values insignificantly different (P > 0.30) from those pertaining to the first hemorrhage and at normal levels of Pco₂, the mean cerebral blood flow was significantly greater at each pressure step than it had been during the first period of hemorrhage with the exception of the lowest arterial blood pressure level (Fig. 5). In addition, as mean arterial blood pressure was reduced progressively, mean cerebral blood flow decreased concomitantly: in other words, following retransfusion there was no pressure-flow plateau (Fig. 5). The linear correlation coefficient for mean cerebral blood flow against mean arterial blood pressure during this second hemorrhage was r = 0.79 (P < 0.001), indicating that flow was pressure dependent during the second hemorrhage.

Discussion

The purpose of the present study was to determine what influence, if any, the sympathetic nervous system had over the response of the cerebral circulation to induced hemorrhagic hypotension.

GROUP 1: HEMORRHAGIC HYPOTENSION ALONE

The lower limit of autoregulation found in the hemorrhagic hypotension group was in close agreement with that found in other studies in which hypotension has been induced by bleeding both in dogs (2, 3) and in primates (4, 21).

GROUP 2: HEMORRHAGIC HYPOTENSION PLUS CHRONIC CERVICAL SYMPATHECTOMY

In this group, the lower limit of autoregulation was again approximately 65 mm Hg. This value is consistent with that from the experiments of Waltz et al. (22) who studied cortical blood flow and pial vessel diameter in cats with chronic unilateral denervation of the cerebral vasculature. No side-to-side differences were noted in their study. When Eklöf et al. (21) used the ¹³³Xe clearance techniques in rhesus monkeys, they failed to identify any difference in the response to induced hemorrhagic hypotension between normal monkeys and monkeys with prior excision of the superior cervical ganglia.

GROUP 3: HEMORRHAGIC HYPOTENSION PLUS ACUTE CERVICAL SYMPATHECTOMY

Various anatomical studies have shown that the superior cervical ganglia are the trophic centers for the adrenergic fibers that innervate the cerebral vessels (23–26). The fibers from one ganglion are distributed to the vessels of the ipsilateral hemisphere, and there is no overlapping. Thus, it would seem that either superior cervical ganglionectomy or cervical trunk division should effectively ablate the adrenergic pathways to the majority of vessels in the cerebral hemisphere from which blood flow is measured.

To our knowledge, there has only been one previous study of the effects of acute sympathectomy on cerebral autoregulation: that of James and his co-workers (4). Their findings differ from those of the present report on the following points. In the first instance, James et al. (4) found that cerebral blood flow was significantly elevated at normotension following acute sympathectomy, whereas we found no such difference. Second, James et al. (4) stated: “After sympathectomy . . . blood flow in gray matter fell steadily as mean arterial pressure was reduced in steps from 130 down to 70 mm Hg.” Possible reasons for the discrepancies between the studies of James and his colleagues (4) and the present investigation will be discussed later.

The concentration of norepinephrine in pial vessels decreases to almost zero within 2 weeks following either cervical sympathetic trunk division or superior cervical ganglionectomy; this change correlates with the destruction of the postganglionic adrenergic neurons (27). A plausible explanation for the difference between the effects of acute and chronic sympathectomy on cerebral autoregulation could be the phenomenon of denervation hypersensitivity. Denervation hypersensitivity reaches a maximum about 2 weeks after sympathectomy (28). In conditions of stress, such as extreme hemorrhagic shock, there is a considerable discharge of catecholamines from the suprarenal medullae. These circulating amines could then interact with the sensitized α-receptors, resulting in a pressure-flow pattern in the chronically sympathectomized animal similar to that in the normal animal.

GROUP 4: HEMORRHAGIC HYPOTENSION PLUS PHENOXYBENZAMINE

Alpha-receptor blockade has been studied previously in humans (29, 30). The first of these studies showed that phentolamine does not affect the normal cerebral response to arterial blood pressure manipulations, although, on the average, arterial blood pressure was lowered by only 17%. Therefore, no statement could be made about the effects of α-receptor blockade on the lower limit of autoregulation. In the investigation by Meyer’s group (30), the effects of intracarotid administration of phenoxycbenzamine on the relationship of arterial blood pressure and cerebral blood flow were studied in a
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series of patients with impaired autoregulation. This impairment was associated with various degrees of cerebral ischemia and infarction. Their findings indicated an improved ability of the cerebral vessels to dilate during induced hypotension following α-receptor blockade.

GROUP 5: HEMORRHAGIC HYPOTENSION PLUS RETRANSFUSION

The severe, although not extreme, hypotension induced on the first hemorrhage was followed by a progressive reduction in cerebral blood flow when arterial blood pressure was decreased for the second time. The probable reason for the observed increase in blood flow at normal blood pressures following hypotension and retransfusion is the cerebral hypoxia accompanying the first arterial blood pressure reduction. This posthypotensive loss of the usual pressure-flow relationship (and cerebral hyperemia at normotension) has been noted in both dogs (31) and primates (32), although in the primate study the duration of the hypotensive episode was more extreme. Other studies have confirmed this posthypoxic loss of the pressure-flow relationship, which may persist for several hours following the insult in both stagnant and hypoxic hypoxia (33, 34). Lassen (35) was the first to hypothesize that this phenomenon, which he termed “luxury perfusion,” is due to the acute cerebral metabolic acidosis that accompanies hypoxia. Later studies have confirmed this concept: the reactive hyperemia after cerebral hypoxia is related to a tissue lactic acidosis (36, 37).

In all of the groups, there was a decrease in arterial pH, which was approximately the same in each group. However, it has been shown that systemic metabolic acidosis, even of a greater degree, will not affect cerebral blood flow as long as the arterial PCO₂ remains constant (38).

A relatively constant cerebral blood flow was preserved to a lower absolute level of mean arterial blood pressure following both acute surgical sympathectomy and pharmacological adrenoreceptor blockade in these studies. Accordingly, we can offer little support for the neurogenic hypothesis of the nature of autoregulation (4, 12). Indeed, the implication of this study is that the sympathicoadrenal discharge that accompanies hemorrhagic hypotension is detrimental to, rather than responsible for, cerebral autoregulation.

Similar situations occur in other organs, but to a greater extent. Using the kidney as an example, blood pressure reduction by either ganglionic blockade or graded occlusion of the abdominal aorta proximal to the renal arteries results in a relative constancy of renal blood flow over a wide range of arterial blood pressures, the lower limit being approximately 65 mm Hg in dogs. However, hemorrhagic hypotension alone results in a linear pressure-flow relationship. The conclusion is that sympathetic activity plays a major role in the vasocclusion of the renal cortex which follows hemorrhage. This hypothesis has been confirmed in the kidney, since maintenance of a constant flow following bleeding is restored when a ganglionic blocking agent is given (39). It is also pertinent to note that the pressure-flow relationship in the kidney and in other tissues is now recognized as an intrinsic mechanism. This intrinsic mechanism has been termed “autoregulation” which is defined as the inherent ability of an organ or tissue to maintain a relatively constant blood flow in the face of moderate changes in perfusion pressure. There is now good evidence indicating that the normal response of the cerebral circulation to changes in arterial blood pressure can be properly termed autoregulation. (1) Acute sympathectomy does not impair autoregulation; in fact, in the present study, it improved it. Neither in this study nor in others does chronic sympathectomy affect autoregulation. (2) Autoregulation has been noted following the administration of ganglionic blocking agents, such as trimetaphan, which interrupt all autonomic pathways both in man (40) and in baboons (41). (3) Autoregulation is still present when cerebral perfusion pressure is lowered by increasing cerebrospinal fluid pressure rather than by lowering arterial blood pressure (6, 42). (4) Symon’s group (10) has shown that an increase in pressure in an artery on the surface of the cortex results in a rapid autoregulatory change as seen by constant venous drainage from the area of cortex supplied by that artery.

The observation in the present study that α-receptor blockade or surgical sympathectomy actually improved autoregulation can best be explained by the “dual control” hypothesis advanced by Harper and his colleagues (19). They observed that, whereas cervical sympathetic stimulation and norepinephrine have minimum effects on cerebral blood flow at normal resting conditions, there is a pronounced fall in cerebral blood flow when the cerebral vessels are already dilated during hypercapnia. They suggested that the cerebral circulation can be described as two resistances in series: the extraparenchymal vessels are influenced by the autonomic nervous system, but the intraparenchymal vessels are regulated by intrinsic metabolic or myogenic mechanisms. This theory has been sup-
ported by a number of other more recent investigations (43-47). The intraparenchymal resistance vessels are the site of the principal autoregulatory adaptation to variations in arterial blood pressure (10, 17, 48). Before these vessels are near maximum dilation, that is, at the lower limit of autoregulation, it might be expected that any adrenergic vasoconstriction of the large arteries at the base of the brain would be met by a compensatory vasodilation of the intraparenchymal arterioles. When the lower limit of autoregulation is exceeded and the intraparenchymal vessels are already dilated, then any adrenergic influence on the larger, extraparenchymal arteries would tend to compromise cerebral blood flow. Evidence for this hypothesis can be adduced from angiographic studies in baboons which demonstrate that in hemorrhagic shock there is a constriction (not autoregulatory dilation) of the arteries at the base of the brain proportional to the degree of hypotension (49). Further evidence obtained from in vitro studies indicates that there is a shift of the flow-limiting resistance from the small arterioles to the larger arteries at the lower limit of autoregulation (J. K. Farrar, personal communication).

The current investigation gives no indication as to the functional significance of the sympathetic nerves that innervate the cerebral vessels. It could be that they are involved in the regulation of cerebral blood volume (50) or in the cerebrovascular mediation of the Cushing response (51). Thus, there exists the possibility that adrenergic nerves are important in the cerebral responses to changes in intracranial pressure.

Therefore, we believe that our results demonstrate that cerebral autoregulation is not controlled by the sympathetic nervous system. However, under conditions of hemorrhagic hypotension alone, sympathetic constriction of the extraparenchymal cerebral vessels decreases the possible range of autoregulation in the anesthetized baboon. A clinical implication of this work is that the cerebral circulation is at greater risk during a state of hemorrhagic or cardiogenic shock than it is if the hypotension is induced by autonomic blocking agents.

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