Reaction of Pial Arteries and Veins to Sympathetic Stimulation in the Cat

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SUMMARY  The diameters of pial arteries and veins were continuously monitored by a multichannel video-angiometer through a closed cranial window in 13 cats. Seventy-two arterial portions (diameter 30–283 μm) and 103 venous segments (diameter 32–486 μm) were studied under resting conditions and during stimulation of the cervical sympathetic chain. Arteries with a diameter of ≤ 150 μm constricted 7.3 ± 0.8%; those > 150 μm 13.1 ± 1.4% (p < 0.0005 for both groups). Veins constricted significantly more than arteries of corresponding size (p < 0.005). Veins ≤ 150 μm constricted 11.5 ± 0.9% and those ≥ 150 μm constricted 19.9 ± 1.9% (p < 0.0005 for both groups compared to resting levels). Since the venous compartment contains about 70% of the regional blood volume the tone of the veins is of importance for intracranial pressure. Further studies on the role of sympathetic nervous activity in the regulation of cerebral venous tone under physiological and pathological conditions seem essential.

Stroke, Vol 12, No 4, 1981

SINCE THE EARLY STUDIES on the functional importance of the adrenergic innervation of cerebral vessels initiated by Forbes & Wolff the role of the sympathetic nervous system in regulation of cerebral vessel tone has been debated. Many in vivo investigations on the reaction of pial vessels have been performed with an open cranial window technique. Investigations have generally concentrated on pial arteries and, surprisingly, pial veins have been neglected. Since sympathetic activity is a main regulator of the tone in veins in other vascular beds and since the filling of the veins regulates blood volume, the present study was performed to compare the reaction of pial arteries and veins during sympathetic stimulation. A multichannel videoangiometer for continuous monitoring of vessel diameter variations through a closed cranial window was used. A preliminary report on the venous reaction has been published elsewhere.

Material and Methods

Thirteen cats with a body weight of 1.5–3.0 kg were anesthetized with 30 mg·kg⁻¹ pentobarbital sodium, immobilized with 0.006 mg·kg⁻¹ pancuroniumbromide, intubated endotracheally and respirated with a 3:1 mixture of N₂O:O₂. Body temperature was continuously controlled with a Philips rectal-thermosensor unit and maintained at 37°C–38°C with a heating pad. Mean arterial pressure (MAP) was continuously monitored via a PVC-catheter into the descending aorta through the left femoral artery. The right femoral artery was cannulated for administration of bicarbonate solution if required to maintain acid-base balance. The right cerebral sympathetic chain was isolated and sectioned below the superior sympathetic ganglion and the cranial end mounted on bipolar silver electrodes fixed in a plastic tube. The animals were then placed in a prone position, and the head was fixed into a stereotaxic head-holder. A parietal cranial window with a diameter of 10 mm ipsilateral to the side of the sympathetic preparation was made, closed with a glass shield, and sealed with acrylic. Pial arterial and venous segments were investigated through a closed cranial window in 13 cats. Seventy-two arterial portions (diameter 30–283 μm) and 103 venous segments (diameter 32–486 μm) were studied under resting conditions and during stimulation of the cervical sympathetic chain. Arteries with a diameter of ≤ 150 μm constricted 7.3 ± 0.8%; those > 150 μm 13.1 ± 1.4% (p < 0.0005 for both groups). Veins constricted significantly more than arteries of corresponding size (p < 0.005). Veins ≤ 150 μm constricted 11.5 ± 0.9% and those ≥ 150 μm constricted 19.9 ± 1.9% (p < 0.0005 for both groups compared to resting levels). Since the venous compartment contains about 70% of the regional blood volume the tone of the veins is of importance for intracranial pressure. Further studies on the role of sympathetic nervous activity in the regulation of cerebral venous tone under physiological and pathological conditions seem essential.

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SYMPATHETIC STIMULATION OF CAT PIAL ARTERIES, VEINS/Auer et al.

Figure 1. High speed paper record of 3 arteries between 120 µm and 320 µm resting diameter: the largest artery is the first to start constricting, the smallest artery has the longest latency period.

Figure 2. Resting arterial diameters in µm plotted against percent diameter changes (ΔpA). Large arteries constrict more than small arteries.

Figure 3. Five-channel recording of arterial reaction to stimulation. The resting diameters are between 190 µm and 25 µm. On several vessel portions, the phase of constriction is preceded by a short episode of dilatation.

Figure 4. Averaging diameters between 32 and 486 µm, mean vessel caliber 141 ± 9.4 µm.

The mean overall latency period after onset of stimulation was 9.1 ± 1.2 seconds. Comparison of veins with diameter < 150 µm (mean value 101.8 ± 4.2 µm) and > 150 µm (mean 279 ± 22 µm) resting diameter revealed no significant difference in the speed of reaction although the mean interval for veins < 150 µm was 10.8 ± 1.6 seconds, for veins > 150 µm 7.2 ± 0.8 seconds. Veins with a resting
diameter Table

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FIGURE 4. Venous resting diameters (φV) plotted against the degree of constriction during stimulation in Δ% of φV.

FIGURE 5. Example of venous reactions to stimulation in a single experiment: Maximal constriction is reached at the end of stimulation. The return to the initial diameters is much slower than the change from normal to maximal constriction. The time interval from resting diameter to maximal constriction is markedly shorter than the period between the end of stimulation and return to initial diameters.

diameter > 150 μm constricted significantly more than smaller veins. The percent of constriction is plotted against resting diameter in figure 4. Mean constriction of veins ≤ 150 μm resting diameter was 11.5 ± 0.9% and of veins > 150 μm 19.9 ± 1.9% (p < 0.0005 for both groups compared to resting levels). Individual examples of diameter changes during sympathetic stimulation are shown in figure 5.

Veins with resting diameter ≤ 150 μm as well as > 150 μm constricted significantly more than arteries of corresponding size (p < 0.005). The differences in reaction time lacked statistical significance.

Discussion

The influence of the sympathetic nervous system on cerebral blood flow and on pial vessel reactivity under resting conditions is still a controversial subject which recently has been extensively reviewed. Earlier reports on the reaction of pial arteries to sympathetic stimulation have dealt with small numbers of arteries. The presently used method to study pial vessel diameter has the important advantage that a great number of vessels can be analyzed simultaneously. Our data show that both arteries and veins respond to sympathetic stimulation and that larger vessels (> 150 μm in diameter) generally respond more than smaller ones. Moreover, veins constricted significantly more than arteries of corresponding size. Our finding that larger arteries react more than smaller ones is in agreement with some earlier reports. All sizes of pial arteries constricted (see fig. 2) and we could thus not confirm the suggestion by Wei et al. of a threshold resting diameter below which there is a complete unresponsiveness to sympathetic stimulation.

Little attention has so far been given to the regulation of cerebral veins. To our knowledge there has been no systematic study on the innervation of cerebral or pial veins but it has been briefly reported that pial veins have a more scarce adrenergic innervation than pial arteries. The only earlier information about the reaction of pial veins to sympathetic stimulation is a figure legend from Forbes and Wolff stating that "the vein did not show any measurable change." Our results indicate that sympathetic activity can modify the diameter of pial veins which is in agreement with the role of sympathetic nerves in other vascular beds. The significantly higher degree of constriction in veins than in arteries suggests that the changes in the veins are not only passive responses to arterial diameter variations. The present observations allow us to postulate an active regulation of the veins and consequent decrease of blood volume in the brain during sympathetic stimulation. Considering the importance of blood volume for intracranial pressure and the fact that the venous compartment contains about 70% of regional blood volume, further studies on the regulation of the veins seem essential. It has been shown that sympathetic nerve stimulation can reduce the cerebral blood volume in mice. It is known that intracranial pressure can be modified by sympathetic stimulation; this could be a dual effect on blood volume and alteration of the secretion of cerebrospinal fluid.

While the present technique with a closed cranial window eliminates some of the risks for unphysiological reactions of pial vessels, the question of the relevance of data from electrical sympathetic stimulation as compared to physiological stimulation by reflex activation of neuronal pathways remains to be elucidated.

Acknowledgment

This study was supported in part by the Swedish Medical Research Council (project 4968). We are thankful for the skilful technical assistance of Silvia Schreiner and Bertil Svensson.

References

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L M Auer, B B Johansson and S Lund

Stroke. 1981;12:528-531
doi: 10.1161/01.STR.12.4.528

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Print ISSN: 0039-2499. Online ISSN: 1524-4628

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