Cerebral Venous Pressure During Actively Induced Hypertension and Hypercapnia in Cats

L. Auer, M.D., B. Johansson, M.D., and E. T. MacKenzie, M.D., Ph.D.

SUMMARY Superior sagittal sinus pressure, intracranial pressure and arterial pressure were recorded in an experimental series on 10 cats. During drug-induced, severe, acute arterial hypertension and parallel hypercapnia, venous pressure could exceed intracranial pressure in both the supra- and infratentorial compartment. From these data it is concluded that cerebral venous pressure during acute arterial hypertension may contribute to protein extravasation at the postcapillary-venular level.

Methods

The experiments were performed on 2 groups of cats totalling 10 animals. They were intubated endotracheally and respirated with a 3:1 mixture of N₂O:O₂. Body temperature was maintained between 37–38°C using a heating pad controlled thermostatically or by aid of a Philips rectal thermocontrol unit. Descending aorta and vena cava inferior were cannulated via a femoral artery and vein.

In group 1, 4 cats were anesthetized with 60 mg/kg alphachloralose and immobilized with 30 mg gallamine (Flaxedil®). Acute hypertension was induced by injection of 200 µg/kg metaraminol (Aramine®) first at normocapnic and then hypercapnic levels. To induce hypercapnia, 10–20% CO₂ was added to the respired gas mixture for about 2 min, starting just before induction of hypertension.

A Portex No. 2 catheter was placed into the superior sagittal sinus (SSS) at 5–10 mm rostral to the torcular and the bone defect was covered with acrylic. The cisterna magna was punctured percutaneously for registration of CSF pressure changes.

In group 2, 6 animals were anesthetized with 30 mg/kg pentobarbital (Nembutal®). In addition to the procedures already described for group 1, a second Portex No. 2 catheter was placed into the subarachnoid space of the supratentorial compartment at about 10 mm frontotemporal to the position of the catheter in the SSS. Acute arterial hypertension was induced by intravenous injection of 5 µg/kg norepinephrine. All animals were made hypercapnic before every episode of repeated hypertensive flush with the same procedure as described for group 1. Thus, the behavior of infra- and supratentorial pressure during acute hypertension could be observed in comparison with SSS-pressure.

Before every experiment, all low pressure channels were calibrated together to minimize deviations of single pressures caused only by technical insufficiency. Statistical calculations were performed by comparing the difference of maximal pressure values in every single experiment (parallel random).

Results

In group 1, Paco₂ reached maximal values between 55 mm Hg and 106 mm Hg, or 85 mm Hg on the average. Resting values ranged between 30.3 mm Hg and 44 mm Hg (mean 34.2 mm Hg). During hypertension, mean arterial blood pressure (MAP) rose to a mean value of 206 mm Hg; for single values and resting values see table 1. During this period of hypercapnic hypertension, the mean increase of SSSP was from 2.8 mm Hg to 35.1 mm Hg, that of cisterna...
magna pressure (CMP) from 2.1 mm Hg to 31.8 mm Hg. The difference is statistically significant ($p < 0.01$). The mean time for values to arrive at a maximum was 26.6 sec for MAP, 86.4 sec for CMP and 103.6 sec for SSSP. An example of a single experiment is shown in figure 1.

In group 2, PaCO$_2$ maximum during hypercapnia was 110 mm Hg, rising from a mean resting value of 30.5 mm Hg. Within 81 sec after intravenous injection of norepinephrine, MAP rose from 158 mm Hg to 242 mm Hg on the average (for single values see table 2). SSSP again rose to a higher value (mean 47 mm Hg) than CMP (38 mm Hg), the difference being statistically significant ($p < 0.001$). From 7 experiments, SSSP was higher than CMP in 6; in only 1 experiment did CMP exceed SSSP by 3 mm Hg. Comparing CMP with the subdural pressure (SDP) in the supratentorial compartment, the latter rose significantly higher than CMP, 43 mm Hg average ($p < 0.001$). From 7 experiments, SDP was higher than SSSP in only 1 (see table 2). In the remaining 6 experiments, SSSP became higher with a difference of mean values of 4 mm Hg, a statistically not significant result. SSSP reached its maximum within 108 sec, CMP within 91 sec and SDP within 88 sec. These mean values, as well as those from group 1, indicate the characteristic behavior pattern of pressure curves from single experiments, where CMP/SDP were seen in most cases to increase more quickly than SSSP, but also to decrease within a shorter period of time. SSSP remained on a higher level than SDP/CMP for a couple of minutes after the peak and the curves thus frequently formed a spindle (fig. 2).

**Discussion**

Both cerebral venous pressure$^{21-24}$ and intracranial pressure$^{18, 21, 22}$ have been shown to increase during acute arterial hypertension. The results indicate that SSSP does not always equal CMP/SDP, which means that under certain circumstances it is not possible to draw conclusions on changes of the one pressure by

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**TABLE 1** Mean Values and Standard Deviation from Animals in Group 1

<table>
<thead>
<tr>
<th></th>
<th>SSSP</th>
<th>CMP</th>
<th>MAP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Resting values (mm Hg)</td>
<td>2.80</td>
<td>2.10</td>
<td>112.50</td>
</tr>
<tr>
<td>±1.77</td>
<td>±2.29</td>
<td>±20.60</td>
<td></td>
</tr>
<tr>
<td>Peak values (mm Hg)</td>
<td>35.30</td>
<td>31.80</td>
<td>206.00</td>
</tr>
<tr>
<td>±6.87</td>
<td>±7.06</td>
<td>±42.36</td>
<td></td>
</tr>
<tr>
<td>Peak within sec.</td>
<td>103.60</td>
<td>86.40</td>
<td>26.60</td>
</tr>
<tr>
<td>±47.53</td>
<td>±41.86</td>
<td>±12.68</td>
<td></td>
</tr>
</tbody>
</table>

PaCO$_2$ PaO$_2$ PaCO$_2$ Peak

Gas-- Check  

SSSP = superior sagittal sinus pressure, CMP = cisterna magna pressure, MAP = mean arterial blood pressure.

**TABLE 2** Mean Values and Standard Deviation from Animals in Group 2

<table>
<thead>
<tr>
<th></th>
<th>SSSP</th>
<th>CMP</th>
<th>SDP</th>
<th>MAP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Resting values (mm Hg)</td>
<td>6.05</td>
<td>6.00</td>
<td>6.63</td>
<td>158.25</td>
</tr>
<tr>
<td>±4.92</td>
<td>±2.65</td>
<td>±3.97</td>
<td>±34.98</td>
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</tr>
<tr>
<td>Peak values (mm Hg)</td>
<td>47.14</td>
<td>38.49</td>
<td>43.09</td>
<td>241.90</td>
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<tr>
<td>±16.92</td>
<td>±15.00</td>
<td>±16.01</td>
<td>±32.30</td>
<td></td>
</tr>
<tr>
<td>Peak within sec.</td>
<td>106.50</td>
<td>91.00</td>
<td>88.50</td>
<td>81.00</td>
</tr>
<tr>
<td>±19.11</td>
<td>±23.86</td>
<td>±33.37</td>
<td>±44.43</td>
<td></td>
</tr>
</tbody>
</table>

PaCO$_2$ PaO$_2$ PaCO$_2$ Peak

Gas-- Check  

Symbols as in table 1. SDP = subdural pressure in the supratentorial compartment.

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**Figure 1.** Repeated hypercapnia and hypertension in an animal of group 1. Arrows indicate moment of intravenous injection of 200 μg per kg metaraminol. By adding 10% CO$_2$ to the respirator gas mixture during the first experiment, PaCO$_2$ reached a maximum of 64.5 mm Hg. During a 20% inhalation, a 106 mm Hg PaCO$_2$ maximum was found (second experiment). SSSP can be seen to rise significantly higher during the second experiment than ICP (cisterna magna pressure). MAP = mean arterial blood pressure. Marks below ICP-curve indicate intervals of 1 min.
measuring the other. When intracerebral vasodilatation becomes maximal owing to hypercapnia, and CBF is additionally increased by inducing acute arterial hypertension, general overloading of cerebral venous outflow capacity may result. This conclusion may be made from the explanation of the venous volume-pressure relationship (see Folkow and Neil 28). The volume of the venous vessels depends — up to a certain point — on venous pressure. With increasing pressure the lumen changes from a flat structure to an elliptical one, and the maximal volume is reached when the diameter becomes circular. Further increase in intraluminal pressure can increase venous vessel diameter only a small extent, which is limited by the amount of collagen fibers in the venous vessel wall. Venous vessel wall resistance increases with venous pressure, leading to flattening of the venous volume — pressure curve up to a point where vessels do not further dilate with rising intravascular pressure. The next vessel reaction would be rupture at the moment when intraluminal pressure exceeded wall resistance. At levels of venous pressure beyond the level of venous distensibility, intracranial pressure is not further increased since there is almost no more pressure transfer elsewhere.1) However, our data show that venous pressure can reach very high levels and may be of importance in protein extravasation, as an earlier in vivo microscopic study indicated.21 Pressure upstream in cerebral venules is higher than the pressure measured downstream in the SSS.17,18

During normocapnic severe hypertension, venous overloading, demonstrated here as a generalized reaction, could take place within circumscribed areas. Such areas are known as small foci of extremely high flow.6,20 and are identical with the spotty pattern of protein extravasation.1,18 The assumption of focal venular overloading additionally becomes likely from intravital microscope observations21 where pial blood flow increase during acute hypertension (vasodilatation, venular reddening, venular flow speed increase) was seen somewhat earlier in one small region when compared to an adjacent region. Thus, the capacity of a larger draining vein could have been exhausted by early filling from one small region, and the increased venous blood volume entering somewhat later from the adjacent area, was blocked, leading to congestion.

In conclusion, our data seem consistent with the hypothesis that cerebral venous pressure can be a contributory factor leading to protein extravasation at the postcapillary venular level during acute arterial hypertension.

**Acknowledgment**

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**References**


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