Hypertension Does Not Cause Spontaneous Hemorrhage of Intracranial Arteriovenous Malformations

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The authors measured blood pressure changes non-invasively in 56 conscious, unpremedicated patients with cerebral arteriovenous malformations (AVMs) during preparation for proton beam therapy. The procedure requires six injections of local anesthetic and application of a stereotactic frame by fixation into the outer table of the skull, and has been used during the past 20 yr to treat over 1,000 patients with cerebral AVMs. No effort was made to control blood pressure. Blood pressure increased during administration of the local anesthetic and application of the frame. Maximum systolic and mean arterial pressures averaged 160 ± 17 and 118 ± 7 mmHg (mean ± SD), respectively. This represented an average increase of 44 mmHg (38%) in systolic pressure and 32 mmHg (37%) in mean blood pressure at some point during the procedure (P < 0.01 compared with pretreatment control pressures). Systolic pressure increased more than 60 mmHg in 21% of patients. Nevertheless, none of these 56 patients nor any of the more than 1,000 patients treated in similar fashion suffered a clinically evident AVM hemorrhage during the procedure. Since the treatment protocol has not changed materially during the past 20 yr, the authors assume that most patients treated in this fashion developed a similar degree of hypertension and conclude from this large clinical experience that moderate arterial hypertension does not precipitate spontaneous hemorrhage of intracranial AVMs. (Key words: Blood pressure: Hypertension. Brain: arteriovenous malformation; hemorrhage. Complications, arteriovenous malformation: Intracranial hemorrhage.)

IT IS WELL ESTABLISHED that arterial hypertension can cause rupture of an intracranial aneurysm, but whether hypertension contributes to spontaneous rupture of intracranial arteriovenous malformations (AVMs) is less clear. Although evidence from human or animal studies is lacking, clinicians often presume that arterial hypertension can precipitate spontaneous AVM hemorrhage and, consequently, prevention of hypertension is advocated in the anesthetic management of such patients.1,2 We have unique, extensive experience with a nonoperative treatment of patients with AVMs wherein the usual principles of blood pressure control appear to be violated. Yet, after 20 yr and more than 1,000 cases, no AVM has ruptured during the procedure. This suggests that arterial hypertension does not lead to spontaneous hemorrhage of these lesions, and it was this clinical impression that forms the basis of this report.

Methods

Over the course of 1 yr, we measured arterial blood pressure systematically in 56 randomly selected adult patients with intracranial AVMs undergoing Bragg-peak proton beam therapy.3 Proton beam therapy3 is a non-invasive stereotactic surgical procedure that capitalizes on the physical properties of protons such that small areas of tissue can be intensely irradiated while surrounding normal tissue is spared. This procedure is an alternative to surgery when the size or location of the AVM make surgical excision particularly risky or impractical.

The treatments took place at the Cyclotron Laboratory of Harvard University, located approximately 4 miles from the Hospital. Because hospitalized patients are typically transported to and from the Cyclotron by taxi and many individuals are treated as outpatients, we followed a protocol that has been used for 20 yr in patients undergoing this treatment. Specifically, the procedure was performed entirely under local anesthesia. Seizure medication was continued but no premedication was given and patients received no intravenous analgesic or sedative drugs. With the patient in a 45° head-up position, approximately 12 ml of 2% lidocaine with epinephrine 1:200,000 was administered in four separate injections into the periosteum and subcutaneous tissues in the area of the maxilla and occiput. An additional 1–2 ml of 2% lidocaine (with or without epinephrine) was administered subcutaneously into the outer rim of each external auditory canal. Soon thereafter, the stereotactic frame was attached to the patient’s head by skeletal fixation. This 10–20-min process involves inserting metal rods into the external auditory canals for temporary support and subsequently stabilizing the frame using drill rods inserted into the outer table of the calvarium. The remainder of the procedure, including treatment, lasts 1–2 h and is painless. It is important to emphasize that a single neurosurgeon (RKK) has treated over 1,000 patients with AVMs in this fashion over the past 20 yr with few modifications (except with respect to the stereotactic apparatus) in the treatment protocol.4

In the 56 study patients, blood pressure was measured noninvasively from an arm at three time points. Control

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血流压力是在3天的不断测量中得出的，其方式为在患者住院期间，通过收缩压+脉压的平均值来计算。连续的测量被用于在治疗前和治疗后进行分析。这种方法允许我们通过减少合并症的变异来减少测量的次数。在所有的测量中，数据被平均，然后进行变异分析，以确定其差异。

讨论

动脉静脉畸形的脑部通常不常见，因其吸收的血液较少而被识别。为了获得结果，我们采用了一种直接向大动脉输送药物的方法，而没有使用任何复杂的导管。

血流压力的影响

在治疗前，所有患者的血流压力均高于正常水平。在治疗后，血流压力有所下降，但仍然高于正常水平。

数据

表2展示了56例患者的血流压力的测量结果。这些测量是在所有参与研究的患者中进行的，且测量结果分别显示在表2中。

Table 2. Maximum Blood Pressure Changes in AVM Patients during Preparation for Proton Beam Therapy

<table>
<thead>
<tr>
<th>Arterial Blood Pressure (mmHg)</th>
<th>Systolic</th>
<th>Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline (range)</td>
<td>116 ± 9 (96-135)</td>
<td>86 ± 7 (71-98)</td>
</tr>
<tr>
<td>Waiting room (range)</td>
<td>151 ± 15* (120-173)</td>
<td>96 ± 12* (74-150)</td>
</tr>
<tr>
<td>Application of frame (range)</td>
<td>160 ± 17* (123-219)</td>
<td>118 ± 7* (94-154)</td>
</tr>
</tbody>
</table>

数据为平均±SD，56例患者。带有*的为显著差异。

* P < 0.01
stimuli such as arterial CO₂. This suggests that increases in arterial blood pressure would be transmitted directly to AVM vessels. However, when the circulatory dynamics of these lesions are considered, the hypothesis that transient arterial hypertension does not cause an AVM to bleed is plausible. Most AVMs are high-flow, low-resistance shunts, and mean blood pressure in the AVM is only about 45–60% of systemic MAP and is less pulsatile. Moreover, blood flow is usually irregular and turbulent and, in a turbulent system, only the square root of the pressure change is transmitted to the vessel wall. Thus, the impact of an abrupt increase in systemic arterial pressure should be attenuated in the AVM. This is supported by other clinical observations. For instance, the majority of AVMs rupture during sleep, when hypertension is unlikely, and there appears to be no relationship between chronic arterial hypertension and AVM rupture. These data taken together with our own substantiate our assertion that there is probably little relationship between arterial hypertension and spontaneous AVM hemorrhage.

Of course, the statistical probability that hemorrhage would occur in any of our 56 patients during the treatment is very low. However, it seems reasonable to speculate that most, if not all, patients treated in this way over the years developed a similar degree of hypertension. Thus, based on the larger group of 1,000 patients with AVMs, we conclude that a procedure that violates generally accepted principles of blood pressure control and that precipitates moderate hypertension (i.e., 20–30 mmHg increase in MAP) is associated with little short-term risk of clinically evident AVM hemorrhage. We believe this clinical experience is convincing, albeit uncontrolled and indirect, evidence that arterial hypertension of this magnitude and duration is unlikely to precipitate spontaneous hemorrhage of intracranial AVMs.

This information may be most useful under circumstances when prevention of hypertension is difficult to achieve, such as during a rapid sequence induction or awake tracheal intubation of a patient with an intact AVM (i.e., no recent hemorrhages). Our clinical experience suggests that, in such situations, blood pressure control might be considered a secondary priority because a brief period of moderate hypertension is unlikely to cause rupture or rerupture of the AVM. Thus, although increases in blood pressure may be undesirable when intracranial compliance is compromised (e.g., by an intracerebral hematoma) or when the AVM is being manipulated by the surgeon, we conclude that prevention of hypertension in patients with intact cerebral AVMs seems necessary only to the extent that it is a conservative and prudent management objective in any patient undergoing anesthesia and surgery.

References