Bursting pressure of experimental aneurysms

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The laser-sealed arteriotomy (LSA) technique was used to create experimental aneurysms in the rat carotid artery. Animals were reexplored 2, 4, and 8 weeks following LSA, at which time the aneurysms were measured and subjected to a bursting strength pressure. In addition, a group of hypertensive rats with LSA was also tested 2 weeks after surgery. The LSA procedure produced aneurysms of a stable size and bursting pressure over the time studied. The bursting pressure technique may be applicable for assessing aneurysm therapy in an experimental setting.

KEY WORDS □9 aneurysm □9 laser □9 bursting pressure □9 arteriotomy □9 rat

In a companion publication, we outlined the use of the laser-sealed arteriotomy (LSA) procedure as a new model of human cerebral aneurysm. This model has the advantage of being reliable, easy to use, and histologically analogous to human cases. The present report details the size and bursting pressure of these experimental aneurysms as affected by time and hypertension (systolic blood pressure ≥ 170 mm Hg).

Materials and Methods

Adult Sprague-Dawley rats, each weighing between 250 and 350 gm, underwent various procedures according to the standards of care outlined by our University's Animal Research Committee. After induction of anesthesia with intraperitoneal pentobarbital (50 mg/kg), the rats underwent bilateral carotid artery exposure, and a 1.0-mm axial LSA procedure was performed on the right side as described previously. The left carotid artery was placed in an approximator clamp for a similar length of time, but no arteriotomy was made. This vessel served as a control. The wounds were closed in two layers with 3-0 silk sutures, and no antibiotics or systemic anticoagulant agents were used.

In another series of hypertensive animals, a 0.5-mm arteriotomy was performed in the same manner on the right carotid artery; the vessel was opened axially in eight rats and longitudinally in six. These animals have been described elsewhere.

At 2, 4, or 8 weeks after the LSA, the animals were reanesthetized and their neck wounds reexplored to detect the presence of aneurysms. The aneurysms were measured using a 1-mm grid under × 40 magnification, and the maximum dimension was recorded. The rats were then sacrificed by intracardiac perfusion with buffered saline at systemic pressures. The entire common carotid artery was dissected free bilaterally, and reserved for a brief time in saline. In each vessel, one end was cannulated with a blunted, scored, No. 21 needle, and fixed in place with a 2-0 silk tie suture. The female end of the needle was then attached to a perfusion pump in parallel with a pressure transducer and chart recorder as previously described. After the tubing and artery were purged of air, the opposite end of the vessel was ligated with 2-0 silk suture, thus forming a closed system. Buffered saline was infused at 99 cc/hr until rupture of the aneurysm or vessel occurred. The system was capable of generating a maximum pressure of 1200 mm Hg.

Results

Of the control vessels that had been placed in a clamp only (without LSA), none burst when subjected to pressures of up to 1200 mm Hg. All aneurysmal vessels except one in the group of hypertensive rats ruptured, typically near the base or neck of the aneurysm as opposed to the dome. Aneurysm size ranged from 0.5
Bursting pressure of experimental aneurysms

<table>
<thead>
<tr>
<th>Group</th>
<th>Aneurysms</th>
<th>Size* (mm)</th>
<th>Bursting Pressure* (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>normotensive</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2 wks</td>
<td>10/11</td>
<td>2.9 ± 2.6</td>
<td>1000 ± 15.3</td>
</tr>
<tr>
<td>4 wks</td>
<td>6/8</td>
<td>1.6 ± 0.9</td>
<td>1066 ± 65.3</td>
</tr>
<tr>
<td>8 wks</td>
<td>7/8</td>
<td>1.7 ± 0.5</td>
<td>1115 ± 58.3</td>
</tr>
<tr>
<td>hypertensive</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2 wks</td>
<td>14/14</td>
<td>3.0 ± 1.7</td>
<td>966 ± 24.7†</td>
</tr>
</tbody>
</table>

* Values are average ± standard deviation.
† One aneurysm did not burst and was not included in the average bursting pressure.

TABLE 1

Bursting pressure of experimental aneurysms

The present investigation represents a further characterization of the LSA model of human cerebral aneurysms. We have demonstrated that in this system aneurysm size is stable over the 8-week period studied and that the bursting pressure among the groups is the same. The hypertensive rats with a 0.5-mm arteriotomy examined at 2 weeks exhibited similar aneurysm size and bursting parameters. Interestingly, in the LSA model, there was no correlation between aneurysm size and bursting pressure.

Discussion

The present investigation represents a further characterization of the LSA model of human cerebral aneurysms. We have demonstrated that in this system aneurysm size is stable over the 8-week period studied and that the bursting pressure among the groups is the same. The hypertensive rats with a 0.5-mm arteriotomy examined at 2 weeks exhibited similar aneurysm size and bursting parameters. Interestingly, in the LSA model, there was no correlation between aneurysm size and the pressure at which rupture took place.

The relationship between increasing aneurysm size and tendency to rupture is well established in the clinical literature, based primarily on autopsy and multiple-aneurysm cases. In a study of 289 individuals with fatal aneurysmal rupture, Crompton reported that the aneurysm that had bled averaged 5 mm, compared to intact aneurysms, which averaged 2 mm. The observation of significant size differences remains valid even in consecutive or unselected necropsy series. In patients presenting with subarachnoid hemorrhage (SAH) who harbor multiple aneurysms, the larger aneurysm is responsible for the bleed in almost 90% of cases.

Aneurysms in this series ruptured most frequently at the base, as opposed to the dome. Suzuki and Ohara, reporting on a necropsy study, postulated that dome thinning is a function of turbulence within the aneurysm. In the current experiment, the aneurysm is formed along a straight segment of vessel so that the vectors of force created by turbulence may not be directed at the dome as in human bifurcation aneurysms. Indeed, histological examination of our experimental aneurysms often showed thinning along the neck, with significant connective tissue reaction along the dome.

The model selected for testing the integrity of the aneurysms was a constant-infusion bursting-pressure system, similar to that used for determining the strength of laser vessel anastomosis. It has the advantage of generating a quantitative measurement which may be used for comparative purposes. In reality, there is no way to directly assess the tendency for an aneurysm to rupture, as this can only be expressed as a mathematical probability. The bursting pressure is an indirect technique to approximate this value.

In the LSA model, bursting pressure was not a function of aneurysm size. This may be due to the relatively minor variation in aneurysm size that the model is capable of generating (31 of 36 aneurysms were between 1 and 3 mm), or perhaps because bursting pressure is not a reliable indication of the susceptibility to rupture. The model is a useful one in that the procedure is easy to perform, aneurysms arise quickly (within 2 weeks), and aneurysm histology is analogous to that of human aneurysms. In addition, the bursting pressure is a reproducible measure of aneurysm strength. This model could therefore be applied to evaluate various standard therapeutic regimens (such as wrapping or the application of glues) or new aneurysm therapies (such as the use of the laser). It could also be used to fashion aneurysms at vessel bifurcations, both extra- and intracranially.

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References

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