Results of early operations for ruptured aneurysms

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In a consecutive series of 219 patients with a ruptured aneurysm of the anterior part of the circle of Willis, 119 patients (54%) made a good recovery and 67 (31%) died. Of 53 patients who did not have surgery, six (11%) made a good recovery and 37 (70%) died. Urgent surgery with evacuation of an associated significant intracerebral hematoma was performed in 30 patients; nine (30%) made a good recovery and 15 (50%) died. Delayed surgery was performed in 55 patients of whom 42 (76%) made a good recovery and two (4%) died. Early intracranial operation (within 48 to 60 hours after subarachnoid hemorrhage (SAH)) was performed in 81 patients who were in Grades I to III prior to surgery. Sixty patients (74%) made a good recovery, and eight died within a month. Five patients were severely disabled and died 2 to 8 months after SAH and surgery. In 17 patients, although the immediate postoperative course was uneventful, evidence of cerebral ischemia developed 4 to 13 days after the bleed and resulted in death in eight patients. A poor outcome was correlated with a history of elevated blood pressure before SAH. Seven patients, of whom six were women of child-bearing age, demonstrated pronounced vasospasm on postoperative angiography; nevertheless, they remained well and free from ischemic symptoms after surgery.

Early operation combined with removal of subarachnoid clots and rinsing the basal cisterns does not eliminate the risk of delayed ischemic dysfunction. Such early surgery, however, improves overall outcome by preventing recurrent bleeding, and may also reduce the frequency of hydrocephalus.

Key Words: cerebral vasospasm • ischemic deficit • early aneurysm surgery • perivascular blood collection • arterial hypertension

The choice of timing of surgical intervention for ruptured intracranial aneurysms has caused controversy for many years. In 1953, Norlén and Olivecrona concluded that intracranial operations for ruptured aneurysms were extremely dangerous during the acute phase, and advised that surgery should be delayed until after the 3rd week of the acute illness. On the other hand, Pool pointed out in 1961 that the mortality rate need not be forbiddingly high in young patients in good condition. The first observation that angiographic spasm does not develop immediately after subarachnoid hemorrhage (SAH) but only at some time toward the end of the 1st week was made by Kägström, et al., who then advocated that surgery should be performed within 48 hours of rupture, even if the mortality rate approached 20%. Symon similarly concluded that the “operation should be undertaken as soon as the clinical condition of the patient permits...”

The value of early (within approximately 48 hours after rupture) as opposed to late (10 days or more after rupture) operations for intracranial aneurysms remains controversial. It appears that most surgeons still favor a delayed operation as the best method of reducing postoperative ischemic complications. However, early surgery is proposed increasingly often in an attempt to improve overall outcome not only by preventing a rebleed but also by avoiding ischemic dysfunction by removal of subarachnoid blood.

On the basis of these views, since August, 1976, we have operated within 48 hours after the SAH on patients in preoperative neurological Grades I–III of Hunt and Hess. In a few cases, surgery was postponed until the 3rd day after SAH. Postoperative angiography was performed in all cases, with the exception of three patients who died in the early postoperative period. We have reviewed our experience to discover the results of this form of management.

Clinical Material

This report focuses on the results in a consecutive series of 81 patients, who presented between August,
Patients who were at home and capable of self care, but who had persisting neurological deficit or mental disturbances, were classed as having a "fair result." Severely disabled patients, totally dependent on others, were classed as having a "poor result." Patients who were severely disabled or survived in a more or less vegetative state and who later died were classed among the deaths even if the immediate cause of death was not ascribable to SAH, but for example to pulmonary embolism.

### Operative Results

#### Early Surgery

The site of aneurysm and the results in the total series of patients operated on early are shown in Table 1.

<table>
<thead>
<tr>
<th>Outcome</th>
<th>ACoA</th>
<th>ICA</th>
<th>MCA</th>
<th>Total Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>No.</td>
<td>22</td>
<td>25</td>
<td>13</td>
<td>60</td>
</tr>
<tr>
<td>Percent</td>
<td>74%</td>
<td></td>
<td></td>
<td>100%</td>
</tr>
</tbody>
</table>

Good Recoveries. The result was regarded as good in 60 patients (74%). Age and sex distribution in this group is given in Table 2. Two patients were classed as having a good recovery although they remained with neurological deficit (diplopia and oculomotor nerve palsy, respectively) due to preoperative damage from aneurysm rupture.

Out of 21 patients in whom the ventricular fluid pressure (VFP) was monitored according to Lundberg's technique, there was a need for CSF drainage in 12 cases. Drainage varied from intermittent for 1 to 2 days to continuous for 14 days, according to the regimen described by Sundbärg and Pontén. Three of these patients had preoperative CSF drainage, which led to obvious improvement and made early surgery appropriate according to the above criteria. Absorption of CSF did not become permanently impaired in any patient in this group.

Fair Results. The operative result was defined as fair in eight patients with early surgery. The age and sex distribution is given in Table 2. Neurological dysfunction included delayed ischemic deficits in six cases. Two patients developed impaired CSF absorption and had a ventriculostrial shunt implanted 2 and 2½ months after SAH, respectively. Intracranial pressure (ICP) had been monitored during the postoperative period in one of these patients, demonstrating a slight elevation of VFP. Two patients needed postoperative ventricular CSF drainage, and

### Table 1

<table>
<thead>
<tr>
<th>Outcome</th>
<th>ACoA</th>
<th>ICA</th>
<th>MCA</th>
<th>Total Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>No.</td>
<td>22</td>
<td>25</td>
<td>13</td>
<td>60</td>
</tr>
<tr>
<td>Percent</td>
<td>74%</td>
<td></td>
<td></td>
<td>100%</td>
</tr>
</tbody>
</table>

*ACoA = anterior communicating artery; ICA = internal carotid artery; MCA = middle cerebral artery.

### Table 2

<table>
<thead>
<tr>
<th>Age (yrs)</th>
<th>Good Recovery</th>
<th>Fair Result</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>F</td>
<td>M</td>
</tr>
<tr>
<td>10–19</td>
<td>2</td>
<td>0</td>
<td>—</td>
</tr>
<tr>
<td>20–29</td>
<td>3</td>
<td>7</td>
<td>—</td>
</tr>
<tr>
<td>30–39</td>
<td>7</td>
<td>6</td>
<td>—</td>
</tr>
<tr>
<td>40–49</td>
<td>7</td>
<td>6</td>
<td>1</td>
</tr>
<tr>
<td>50–59</td>
<td>6</td>
<td>7</td>
<td>1</td>
</tr>
<tr>
<td>60–69</td>
<td>3</td>
<td>5</td>
<td>1</td>
</tr>
<tr>
<td>70–79</td>
<td>1</td>
<td>0</td>
<td>—</td>
</tr>
<tr>
<td>total</td>
<td>29</td>
<td>31</td>
<td>2</td>
</tr>
</tbody>
</table>
Early aneurysm surgery

**TABLE 3**

<table>
<thead>
<tr>
<th>Outcome</th>
<th>ACoA</th>
<th>ICA</th>
<th>MCA</th>
<th>Total Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>good</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>6</td>
</tr>
<tr>
<td>fair</td>
<td>3</td>
<td>3</td>
<td>6</td>
<td>12</td>
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<tr>
<td>dead</td>
<td>4</td>
<td>2</td>
<td>2</td>
<td>8</td>
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<tr>
<td>total</td>
<td>7</td>
<td>6</td>
<td>4</td>
<td>17</td>
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</tbody>
</table>

*ACoA = anterior communicating artery; ICA = internal carotid artery; MCA = middle cerebral artery.

**TABLE 4**

<table>
<thead>
<tr>
<th>Outcome</th>
<th>ACoA</th>
<th>ICA</th>
<th>MCA</th>
<th>Total Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>good</td>
<td>3</td>
<td>2</td>
<td>1</td>
<td>6</td>
</tr>
<tr>
<td>fair</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>dead</td>
<td>5</td>
<td>3</td>
<td>1</td>
<td>9</td>
</tr>
<tr>
<td>total</td>
<td>9</td>
<td>6</td>
<td>3</td>
<td>18</td>
</tr>
</tbody>
</table>

*SAH = subarachnoid hemorrhage; ACoA = anterior communicating artery; ICA = internal carotid artery; MCA = middle cerebral artery.

one of them was later reexamined because of suspected hydrocephalus, which was not verified.

**Deaths.** There were 13 deaths in the series of patients with early surgery. Eight were early (within 1 month after SAH) and five late (2 to 8 months after SAH). Table 2 shows the age and sex distribution.

Eight patients, two with internal carotid artery (ICA) aneurysms, two with MCA aneurysms, and four with anterior communicating artery (ACoA) aneurysms, were in the same grade immediately after surgery as just before. However, after an interval of 2 to 7 days, that is, 4 to 8 days after SAH, they deteriorated. Six of these patients had arterial hypertension prior to SAH, and all except one received anti-hypertensive medication at the time of SAH.

In four cases, the cause of death was more or less occasioned by a technical failure. A 57-year-old hypertensive man with an ACoA aneurysm was found at autopsy to have a clip positioned so that circulation in the left anterior cerebral artery was obstructed. A 72-year-old hypertensive woman with a right ICA aneurysm was found on postoperative angiography performed 3 days after SAH to have occlusion of the choroidal artery. A 66-year-old woman with an ACoA aneurysm had a broad "spontaneous" rupture of the ICA while the position of a brain retractor was adjusted. The fourth patient, a 55-year-old man with an upward-directed broad-based ACoA aneurysm, did not tolerate trapping of the aneurysm.

Neurological deficits persisted in one patient, who was found at autopsy to have a focal infarction. Among the patients in this group, eight had ventricular CSF drainage. Another three patients had ICP monitoring, but no need for drainage was identified.

**Delayed Ischemic Deficits and Outcome.** In the present series, 17 (21%) of the 81 patients with early surgery developed delayed ischemic deficits with onset of symptoms 4 to 13 days after SAH. The site of ruptured aneurysm and results at follow-up examination are given in Table 3. Eight of these patients died (see above); however, two of these had more than one aneurysm and had surgery directed toward an unruptured aneurysm. Preoperative computerized tomography (CT) and angiography had failed to identify which aneurysm had ruptured. Consequently, subarachnoid washout in the vicinity of the ruptured aneurysm was not performed in these two cases. A third patient, who died with delayed ischemic deficits, had had an SAH and aneurysm surgery 12 years earlier. In this case, there were pronounced subarachnoid adhesions, and it was not even possible to clean the anterior cisterns.

In six cases, the outcome was defined as fair and in three as good at follow-up review.

**Arterial Hypertension and Outcome.** Eighteen patients with early surgery were categorized as hypertensive (diastolic pressure exceeding 110 mm Hg on at least two separate measurements before SAH, or a history of previous treatment for arterial hypertension). The site of ruptured aneurysm and result at follow-up for these patients are presented in Table 4. Among the 18 hypertensive patients, nine (50%) developed delayed ischemic deficits in contrast to eight of the 63 normotensive patients (13%). Of the eight hypertensive patients who received antihypertensive medication at the time of SAH, six (75%) developed delayed ischemic deficits.

Altogether, nine (50%) of the hypertensive patients died. Two deaths were due to operative failures, in one case surgery was directed toward an unruptured aneurysm, and in a fourth a focal infarction was found at autopsy (see above). Six patients woke up in the same grade as before surgery but deteriorated between the 4th and 8th day after aneurysm rupture. Among the eight patients with a fair outcome, three (38%) were hypertensive. Of the six hypertensive patients finally defined as having a good recovery, only two had not manifested transient neurological deficits. The final outcome for hypertensive versus normotensive patients is summarized in Table 5.

**Daily Lumbar Puncture.** Of the early surgery group, 42 patients had daily lumbar punctures postoperatively for an average of 5 days in order to aid in removal of blood-contaminated CSF. The volumes evacuated daily ranged from 20 to 30 ml. Lumbar punctures resulted in a lowered VFP for a few hours. The withdrawal of lumbar CSF had no obvious effect on the incidence of delayed ischemic deficits.
TABLE 5

Outcome in patients with early surgery, with arterial hypertension versus normotension*

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Hypertensive</th>
<th>Normotensive</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>Percent</td>
</tr>
<tr>
<td>good &amp; fair</td>
<td>9</td>
<td>50</td>
</tr>
<tr>
<td>dead</td>
<td>9</td>
<td>50</td>
</tr>
<tr>
<td>total</td>
<td>18</td>
<td>100</td>
</tr>
</tbody>
</table>

*The difference in outcome between normotensive and hypertensive patients is significant at p < 0.001 (Fisher's exact test).

Urgent Surgery

Of the 30 patients who required emergency surgery due to the presence of an associated significant intracerebral hematoma, nine made a good recovery, three were classed as fair, and another three as poor results at follow-up review. Fifteen of these patients died (Table 6).

Delayed Surgery

The results in the delayed surgery group are summarized in Table 6. Of the 32 patients who were admitted too late for early surgery but who were subjected to late surgery, 26 made a good recovery, two were classed as fair, and three as poor results; one patient died. Of the 23 patients who were not selected for early surgery, no aneurysm was visualized on the initial angiogram in two patients, both of whom made a good recovery after delayed surgery. Of the remaining 21 patients who were not selected for early surgery but later improved and had delayed surgery, 14 made a good recovery, four were classed as fair, and two as poor results, while one patient died.

In summary, 76% of the patients with delayed surgery made a good recovery, 11% were classed as fair and 9% as poor, and 4% died.

No Surgery

The outcome for the 53 patients who did not have aneurysm surgery is shown in Table 6: 23 patients would have fulfilled the criteria for early surgery; six patients were considered "borderline patients" (that is, patients arriving in time for early surgery but not operated on because of various complicating factors, like being over 70 years old, in combination with advanced pulmonary emphysema, heart dysfunction, or severe arteriosclerotic narrowing of main cerebral arteries); and 24 patients remained in Hunt and Hess' Grades IV or V or deteriorated during the first 12 to 24 hours after SAH, and were at no time considered candidates for aneurysm surgery.

The 23 patients who would have fulfilled the criteria for early surgery but were not operated on were further analyzed. As shown in Table 7, nine patients did not have early surgery as a consequence of too late admission due to patient's delay; poor results were caused by the development of delayed ischemic dysfunction in one case and in combination with repeat SAH in one case; one death was caused by delayed ischemic dysfunction, and two deaths were caused by repeat bleeds. Doctor's delay (that is, delay at a local hospital) made early surgery impossible in eight cases: two patients died of delayed ischemic deficits, and two of rebleeds; one poor result was caused by delayed ischemic dysfunction. Neurosurgical delay (that is, organizational problems, patient's disease primarily misinterpreted) prevented early surgical intervention in six patients, of whom four died, all of rebleeds within 10 days after the first SAH. One

TABLE 6

Overall outcome in 219 patients with a ruptured aneurysm of the anterior part of the circle of Willis

<table>
<thead>
<tr>
<th>Patient Groups*</th>
<th>Good Recovery</th>
<th>Fair Result</th>
<th>Poor Result</th>
<th>Deaths</th>
<th>Total Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>urgent surgery</td>
<td>9</td>
<td>3</td>
<td>3</td>
<td>15</td>
<td>30</td>
</tr>
<tr>
<td>early surgery</td>
<td>60</td>
<td>8</td>
<td>-</td>
<td>13</td>
<td>81</td>
</tr>
<tr>
<td>delayed surgery</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>not selected for early surgery</td>
<td>16</td>
<td>4</td>
<td>2</td>
<td>1</td>
<td>23</td>
</tr>
<tr>
<td>fulfilled criteria for early surgery but admitted too late</td>
<td>26</td>
<td>2</td>
<td>3</td>
<td>1</td>
<td>32</td>
</tr>
<tr>
<td>no surgery</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>fulfilled criteria for early surgery during first 60 hrs post-SAH</td>
<td>5</td>
<td>1</td>
<td>4</td>
<td>13</td>
<td>23</td>
</tr>
<tr>
<td>Hunt &amp; Hess' Grades IV and V</td>
<td>1†</td>
<td>-</td>
<td>-</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>total cases</td>
<td>119</td>
<td>18</td>
<td>15</td>
<td>67</td>
<td>219</td>
</tr>
<tr>
<td>percent</td>
<td>54</td>
<td>8</td>
<td>7</td>
<td>31†</td>
<td>100</td>
</tr>
</tbody>
</table>

*For fuller description see text.
†Follow-up period: 2½ years.
‡Management mortality.
Early aneurysm surgery

poor result was a consequence of persisting delayed ischemic dysfunction.

Of the 23 patients who fulfilled the criteria for early surgery but were not operated on, 13 patients died. Five of these 13 patients (38%) were hypertensive.

Discussion

Our policy is to have all patients with a verified or suspected SAH referred immediately irrespective of neurological grade. This policy is probably reflected in our management mortality, which was 31% for the total series of 219 cases with a ruptured aneurysm of the anterior part of the circle of Willis.

The outcome for the group of patients receiving early surgery (81 cases) versus the outcome for the group of patients who fulfilled the criteria for such surgery but who were not operated early (55 cases) is summarized in Table 8. Our results indicate that early surgery is equal or superior to delayed treatment. In fact, when considering good recoveries only, the results of early surgery were fully comparable to the results in the selected group of patients subjected to conventional delayed surgery (74% and 76% good recoveries, respectively). Early surgery is also accompanied by a markedly shortened hospital stay: in our series, the 60 patients with good recoveries in the group operated on early spent an average of 16 days in the clinic in contrast to more than double that time for patients receiving delayed therapy.

When compared to the probability data of Alvord, et al., for a 2-year survival period with conservative treatment, the results in the present series with early operation indicate that early aneurysm surgery is superior to the natural history of the disease. Only 55% to 65% of the patients in our series with early operation would be alive after 2 years.

We discovered a striking correlation between the presence of arterial hypertension prior to SAH and development of delayed ischemic deficits. In 1963, Allcock and Drake reported that systemic hypertension was accompanied by an increased incidence of angiographic vasospasm, and concluded that arterial spasm worsened the prognosis. Wilkins, et al., found a slightly increased frequency of angiographic evidence of vasospasm in hypertensive compared to normotensive patients. The 1966 Cooperative Study revealed that the category of patients with preexisting arterial hypertension had a 55.3% mortality, as compared to the overall mortality of 36.8% in the whole series. Fisher, et al., found no correlation between vasospasm and arterial hypertension in their analysis, and Samson, et al., in their series of 106 consecutive Grades I and II patients, found no correlation between preexisting arterial hypertension and the development of postoperative neurological deficit.

Several studies indicate that arterial vessels in hypertensive individuals differ morphologically from those of normotensive individuals. Cook and Yates found medial hypertrophy at all artery sizes in brains from hypertensive humans. Russell examined brains from patients who had hypertension during life and found that small penetrating arteries, 50 μ in diameter, showed decreased lumen and increased wall thickness. Spontaneously hypertensive experimental animals are more vulnerable than normotensive animals to bilateral ligation of the common carotid artery, which may indicate a reduced capacity for compensatory vasodilatation. Furthermore, the cerebrovascular resistance during maximum vasodilatation is considerably higher in hypertensive rats than in normotensive rats. This suggests that hypertensive patients may be more vulnerable than normotensive patients to severe arterial narrowing, especially when this narrowing (spasm) affects the penetrating arteries.

In a recent report, it was stated that patients with angiographically demonstrated vasospasm eventually show neurological symptoms. The findings in our series are not in agreement with this statement. Some patients in the present series developed severe angiographic vasospasm (Type 1) but remained free from symptoms and signs of delayed ischemic dysfunction. Other patients with severe delayed ischemic deficits demonstrated only a very moderate narrowing of the larger cerebral arteries at angiography. These find-
ings stress the importance of the distribution of vaso-
spasm and the adequacy of the patient's compens-
atory mechanisms, and demonstrate that spasm in
the conducting arteries is only one factor in the total
outcome that determines how well the brain is per-
fused after SAH. 7

Disturbance of autoregulation 64,49 and intravascu-
lar platelet aggregation with embolization of platelet
thrombi,69 and consequent occlusion of arterioles
and capillaries may occur as a consequence of extra-
vasation of blood into the perivascular space. 8,22,37,46
By assessing the amount of subarachnoid blood
visualized on CT, Fisher, et al.,9 found that blood
localized in the subarachnoid space is the only eti-
ological factor in vasospasm. Consequently, it has been
proposed that early and extensive removal of perivas-
cular blood collections should be effective in minimiz-
ing the occurrence of vasospasm and delayed ischemic
deficits.34,50,41,46,47 Our results fail to provide evidence
that the capricious appearance of cerebral delayed
ischemic dysfunction can be eliminated by our present
operative procedures. However, this does not pre-
clude the fact that subarachnoid blood is the prime
etiological factor in vasospasm and delayed cerebral
ischemia. In cases in which a preoperative CT scan
showed a thick layer of subarachnoid blood and in
which an early postoperative CT scan was performed,
there was consistently residual blood. With improved
techniques for cleaning the subarachnoid spaces, it
might be possible to further reduce the incidence of
delayed ischemic complications.

In a recent series of aneurysms operated on early,41
a high percentage of patients required a ventriculo-
peritoneal shunt because of impaired CSF outflow. In
our series, temporary CSF drainage by catheter to a
frontal horn was performed in 22 cases. In many in-
tances, CSF drainage was started with the onset of
delayed ischemic deficits. Only two patients (3%) de-
veloped permanently impaired CSF outflow accord-
ing to clinical criteria, verified by CT scan and lumbar
infusion test. Both patients had a ventriculotriatal
shunt implanted, and at follow-up review they were
considered as having a fair outcome. Samson, et al.,58
in their series of 106 good-risk surgical patients (79
operated on within 8 days after SAH and 27 operated
on later), reported a 9% incidence of postoperative
hydrocephalus. In our opinion, early aneurysm sur-
gery and washout of blood-contaminated CSF lessens
the risk of subarachnoid fibrosis and impaired CSF
outflow.

The case for delayed surgery was strongly stated by
Drake: 4 "if we could learn how to keep a patient safe
from rebleeding for a week . . . the problems of the
surgery of ruptured intracranial aneurysms would be
nearly solved." In arriving at this view, he was prob-
ably influenced both by the extremely poor results of
early surgery and also by his observations of the con-
dition that his associates had faced when performing
early aneurysm surgery: "it was not all spasm either,
for I watched them struggling with the angry, swollen
brain."7

Today, with microsurgical adjuncts and improved
techniques, dealing with the blood-stained and swollen
brain certainly is not easy, yet does not imply a dis-
astrous struggle. Future improvements in the outcome
of patients with SAH may depend upon a rephrasing
of Drake's statement: if we could learn how to keep a
patient safe from developing delayed cerebral
ischemic dysfunction, then the problem of surgery of
ruptured aneurysms would be nearly solved.

References

1. Alcock JM, Drake CG: Ruptured intracranial aneu-
rysms: the role of arterial spasm. J Neurosurg 22:21-29,
1965
arachnoid hemorrhage due to ruptured aneurysms. A
simple method of estimating prognosis. Arch Neurol
27:273–284, 1972
3. Bohm E, Hugosson R: Results of surgical treatment of
200 consecutive cerebral arterial aneurysms. Acta
mia, and interruption of carotid, or carotid and vertebral
5. Cook TA, Yates PO: A histometric study of cerebral
and renal arteries in normotensives and chronic hyper-
risk as related to time of intervention in the repair of in-
7. Drake CG: Discussion of Symon L: Vasospasm in aneu-
rysms, in Moossy J, Janeway R (eds): Cerebral
Vascular Diseases. Seventh Princeton Conference. New
York: Grune and Stratton, 1971, pp 241–244
8. Echlin FA: Experimental vasospasm, acute and chronic,
due to blood in the subarachnoid space. J Neurosurg
35:646–656, 1971
vasospasm to subarachnoid hemorrhage visualized by
computerized tomographic scanning. Neurosurgery 6:1–9, 1980
10. Fisher CM, Roberson GH, Ojemann RG: Cerebral
vasospasm with ruptured saccular aneurysm — the
11. Fleischer AS, Tindall GT: Cerebral vasospasm follow-
ning aneurysm rupture. A protocol for therapy and pro-
tance of adaptive changes in vascular design for estab-
lishment of primary hypertension, studied in man and in
spontaneously hypertensive rats. Circ Res (Suppl 1)
22:2–16, 1973
bral metabolism after bilateral carotid artery ligation in
normotensive and spontaneously hypertensive rats. J
Neurol Neurosurg Psychiatry 39:212–217, 1976
14. Graf CJ, Nibbelink DW: Cooperative study of intra-
cranial aneurysms and subarachnoid hemorrhage.
Report of a randomized treatment study. III. Intra-
15. Hamby WB: Remarks concerning intracranial aneurys-
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