Intracranial Arterial Spasm: A Clinical Analysis*

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Materials and Methods

The charts and preoperative arteriograms of 259 patients were reviewed. Autopsy examinations were performed on 30 of the 51 patients who died during their hospitalization.

Definitions. A patient arbitrarily was categorized as hypertensive if he had a brachial cuff pressure exceeding 150/100 on at least 2 separate days, or had a history of previous treatment for hypertension. In all patients with spontaneous subarachnoid hemorrhage, the diagnosis was established by lumbar puncture. The responsiveness of each patient at the time of arteriography was categorized as normal, lethargic (drowsy, but able to follow commands or talk), comatose (only able to withdraw from painful stimuli), or rigid (decorticate or decerebrate rigidity). The arteriographic pattern of segmental narrowing of intracranial arteries was identified as spasm provided that the narrowed areas were not: 1) irregular and ragged, suggesting atheromata, 2) in arteries known on occasion to be hypoplastic (as in the proximal anterior cerebral artery), 3) in arteries stretched around mass lesions, or 4) radiographic artifacts due to laminar flow of the contrast medium.

Arteriograms. Carotid and brachial arteriography was performed percutaneously, with Renografin or Hypaque; at least two views were made in the anteroposterior projection and two in the lateral projection in each study.

With five exceptions, each of the 128 patients with spontaneous subarachnoid hemorrhage or intracranial aneurysm had bilateral carotid arteriography preoperatively. Three of the patients had only one carotid artery studied, two because they were so ill and one because of previous carotid ligation. The other two patients had only brachial arteriograms, one with a vertebral artery aneu-

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rysm that presented as a mass lesion, and one with basilar transient ischemic attacks and an asymptomatic aneurysm of the internal carotid artery.

Twelve of the 99 patients with intracranial aneurysms had additional preoperative carotid arteriograms, nine because the diagnosis could not be established with certainty on the first arteriograms, and three because the status of the previous arterial spasm needed to be determined. Twenty of the 99 had preoperative brachial or vertebral arteriography. In 10 of these, with negative carotid arteriograms, aneurysms of the vertebrobasilar system were found.

Each of the other 29 patients with spontaneous subarachnoid hemorrhage had several arteriographic studies. All had initial bilateral carotid arteriograms and either a brachial or a vertebral arteriogram. Twenty also had second bilateral carotid arteriograms, on an average of 8 days after the first.

Of the remaining 131 patients, 111 had unilateral carotid arteriograms and 20 had bilateral carotid arteriograms preoperatively. However, in many of the cases with craniocebral trauma, bilateral opacification of the cerebral arteries was accomplished by manual compression of the contralateral carotid artery during arteriography. Only two had more than one preoperative carotid arteriogram.

Electrocardiograms. Electrocardiograms (EKG) were analyzed to determine whether the EKG abnormalities which frequently accompany spontaneous subarachnoid hemorrhage might coincide with the presence of intracranial arterial spasm. Of the patients with verified spontaneous subarachnoid hemorrhage, 35 had preoperative electrocardiograms within 1 day of carotid (34) or retrograde brachial (1) arteriograms. Twelve of the 35 had more than one EKG during their hospitalization. The 35 patients were divided into three groups for EKG analysis: Group A, aneurysm and intracranial spasm (12); Group B, aneurysm and no intracranial spasm (14); and Group C, no aneurysm and no spasm (9). In addition to the electrocardiograms, the following parameters were also studied in these 35 patients: age, sex, and race; clinical evidence of hypertension or significant heart disease; chronological relationship of EKG to subarachnoid hemorrhage, arteriogram, and operation; serum electrolytes at the time of each EKG; and death during hospitalization.

Other Clinical Information. Twenty patients had intracerebral hematomas. Four of the 11 spontaneous intracerebral hematomas were located in the area of the basal ganglia, 5 were primarily parietal, and 2 were in other locations. Five of the 9 traumatic intracerebral hematomas were frontal and 4 were temporal. Fifty patients had arteriograms for suspected intracranial neoplasms, and in 33 of these patients the diagnosis was later confirmed by craniotomy: glioma, 12; meningioma, 7; metastatic carcinoma, 7; chromophobe adenoma, 5; craniopharyngioma, 1; and colloid cyst, 1.

Results

Basic Clinical Data (Tables 1 and 2). The patients with spontaneous subarachnoid hemorrhage had a higher incidence of hypertension than the other patients, and a greater percentage were females. However, when the basic data (sex, age, presence of hypertension, responsiveness during arteriography, and hospital mortality) from the patients with intracranial arterial spasm were compared with those from corresponding patients with no spasm, the differences were not statistically significant (p > 0.01 using the Chi square test).

Intracranial arterial spasm was seen most frequently in conjunction with subarachnoid hemorrhage, and it appeared roughly related to the amount of blood in the subarachnoid spaces enclosing the circle of Willis. Forty-four (36.7%) of the 120 patients with spontaneous primary bleeding into the subarachnoid space demonstrated arterial spasm. However, spasm was identified in the arteriograms of only three (6.4%) of the 47 patients with proven or suspected subarachnoid hemorrhage resulting from acute craniocebral trauma, and in none of the 72 patients who had not had a subarachnoid hemorrhage or an intracranial infection (including eight patients with unruptured intracranial aneurysms).

Intracranial spasm was also noted in association with active meningitis or subdural empyema. Three of the four patients with these types of infections showed spasm, whereas no spasm was seen in the arterio-
### Intracranial Arterial Spasm

#### TABLE 1

**Clinical parameters**

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>No. of Patients</th>
<th>Sex</th>
<th>Average Age (yrs)</th>
<th>Hypertension</th>
<th>Hospital Deaths</th>
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<tr>
<td><strong>Intracranial Vascular Lesions</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SAH*, aneurysm, spasm†</td>
<td>35</td>
<td>10</td>
<td>25</td>
<td>45.0</td>
<td>20</td>
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<tr>
<td>SAH, aneurysm, no spasm</td>
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<td>23</td>
<td>33</td>
<td>47.6</td>
<td>27</td>
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<tr>
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<td>12</td>
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<td>Aneurysm, no SAH</td>
<td>8</td>
<td>4</td>
<td>4</td>
<td>45.8</td>
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<tr>
<td>Spontaneous intracerebral hemorrhage</td>
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<td>6</td>
<td>5</td>
<td>44.5</td>
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<tr>
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<td></td>
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<tr>
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<td>7</td>
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<tr>
<td>Acute or subacute subdural hematoma</td>
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<td>8</td>
<td>2</td>
<td>45.0</td>
<td>6</td>
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<tr>
<td>Chronic subdural hematoma</td>
<td>11</td>
<td>8</td>
<td>3</td>
<td>64.2</td>
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<tr>
<td>Cerebral contusion</td>
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<td>19</td>
<td>2</td>
<td>29.0</td>
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<tr>
<td><strong>Intracranial Infections</strong></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Encapsulated brain abscess</td>
<td>7</td>
<td>6</td>
<td>1</td>
<td>29.4</td>
<td>0</td>
</tr>
<tr>
<td>Subdural empyema or active meningitis</td>
<td>4</td>
<td>4</td>
<td>0</td>
<td>36.8</td>
<td>1</td>
</tr>
<tr>
<td><strong>Suspected Intracranial Tumors</strong></td>
<td>50</td>
<td>29</td>
<td>21</td>
<td>46.8</td>
<td>11</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>259</td>
<td>149</td>
<td>110</td>
<td>—</td>
<td>91</td>
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</table>

* Spontaneous subarachnoid hemorrhage
† Identified on any preoperative arteriogram. In 30 of the 35 cases, this was present on the first arteriogram.

#### TABLE 2

**Lumbar puncture and cerebral arteriography**

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Subarachnoid Hemorrhage</th>
<th>Responsiveness During Arteriography</th>
<th>Intracranial Arterial Spasm</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Yes</td>
<td>No</td>
<td>L.P.</td>
</tr>
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<td><strong>Intracranial Vascular Lesions</strong></td>
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<td></td>
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</tr>
<tr>
<td>SAH, aneurysm, spasm</td>
<td>35</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>SAH, aneurysm, no spasm</td>
<td>56</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>SAH, no aneurysm</td>
<td>29</td>
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<td>0</td>
</tr>
<tr>
<td>Aneurysm, no SAH</td>
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<td>8</td>
<td>0</td>
</tr>
<tr>
<td>Spontaneous intracerebral hemorrhage</td>
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<td>2</td>
<td>3</td>
</tr>
<tr>
<td><strong>Craniocerebral Trauma</strong></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Intracerebral hemorrhage</td>
<td>2</td>
<td>0</td>
<td>7</td>
</tr>
<tr>
<td>Acute or subacute subdural hematoma</td>
<td>1</td>
<td>1</td>
<td>8</td>
</tr>
<tr>
<td>Chronic subdural hematoma</td>
<td>0</td>
<td>2</td>
<td>9</td>
</tr>
<tr>
<td>Epidural hematoma</td>
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<tr>
<td>Cerebral contusion</td>
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<tr>
<td><strong>Intracranial Infections</strong></td>
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<tr>
<td>Encapsulated brain abscess</td>
<td>0</td>
<td>6</td>
<td>1</td>
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<tr>
<td>Subdural empyema or active meningitis</td>
<td>0</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td><strong>Suspected Intracranial Tumors</strong></td>
<td>0</td>
<td>26</td>
<td>24</td>
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</table>
grams of the seven patients with encapsulated brain abscesses. The following case is illustrative.

**Case 1.** A 55-year-old Negro man was discovered to have a right maxillary tooth abscess on June 10, 1967. He developed fever, headache, confusion, and neck stiffness and was admitted to a local hospital on June 20. Lumbar puncture showed a white blood cell count of 18,600/cu mm, of which 70% were polymorphonuclear leukocytes. By June 22, the patient had developed a left hemiparesis. On June 26, he had three seizures involving his left extremities and was transferred to Duke Hospital.

**Examination.** X-ray examination showed that the patient had right maxillary, ethmoidal, and frontal sinusitis. A right carotid arteriogram showed intracranial arterial spasm, as well as evidence of a right subdural empyema (Fig. 1 left).

**Course.** The following operations were performed: right frontal and ethmoidal sinusotomy, right intranasal ethmoidectomy, right maxillary antrotomy, and right cranial trephination in multiple areas, with evacuation of pus from these various sites. No organisms were ever cultured from the cerebrospinal fluid, or from the pus evacuated from the sinuses or the subdural space.

By July 11, all of the patient's subdural drains had been removed and he was showing progressive improvement. Because of persistent fever a right carotid arteriogram was performed on July 13 (Fig. 1 right). There was minimal displacement of the anterior cerebral arteries to the left without evidence of subdural fluid. This was felt to be on the basis of cerebral edema. No spasm was present, however. The patient continued to improve, and a second postoperative right carotid arteriogram performed on July 24 was normal.

**Interval before Initial Arteriography (Table 3).** In the 131 cases of spontaneous subarachnoid or intracerebral hemorrhage

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**Fig. 1. Case 1.** Anteroposterior views of the right carotid arteriograms performed on June 26, 1967 (left) and July 13, 1967 (right). The right anterior cerebral artery is somewhat hypoplastic. The first arteriogram shows evidence of the subdural empyema with separation of the cortical vessels from the inner table of the skull and displacement of the right anterior cerebral artery to the left. There is spasm of the intradural portion of the right internal carotid artery and the proximal portion of the right middle cerebral artery. The second arteriogram shows no evidence of spasm.
and the 48 cases of acute craniocerebral trauma, initial carotid arteriography was performed in most instances within 48 hours of admission to Duke Hospital. The arteriograms were delayed from 5 to 20 days in five cases due to an initial period of clinical observation (2), marked hypertension (2), or perforation of a peptic ulcer (1). However, many of the patients were kept at a local hospital for a period of time before being transferred to Duke Hospital, and for this reason carotid arteriography was delayed up to 61 days after the hemorrhage or trauma had occurred.

The arteriograms showing intracranial arterial spasm were performed at essentially the same time interval after spontaneous subarachnoid hemorrhage as were those showing no spasm. However, of the 19 patients who had subarachnoid hemorrhage from an intracranial aneurysm and who had carotid arteriography within 24 hours after the hemorrhage, none showed evidence of spasm.

Repeated Preoperative Carotid Arteriograms. Thirty-two of the patients with spontaneous subarachnoid hemorrhage had carotid arteriograms on more than one occasion preoperatively. Intracranial arterial spasm was present on the initial arteriograms of four of these patients, at an average of 9.3 days after the subarachnoid hemorrhage. The four patients all improved clinically, and the spasm was no longer present on the second arteriograms, performed at an average of 20 days after hemorrhage.

Both the initial and repeated carotid arteriograms showed no spasm in 15 patients. These were performed at an average of 5.5 and 13.5 days respectively after subarachnoid hemorrhage.

In an additional 13 patients, the first carotid arteriograms were negative for spasm, but the second arteriograms were positive. Four of these 13 patients had a second subarachnoid hemorrhage between the arteriograms. However, the other nine patients had no such hemorrhage and showed progressive clinical improvement despite the development of intracranial arterial spasm. The initial carotid arteriograms in these nine patients were performed soon after the first subarachnoid hemorrhage (average, 1.1 days) and the repeated arteriograms were done at an average of 8.0 days after hemorrhage. The following two cases are illustrative.

**Case 2.** A 39-year-old Negro woman had a subarachnoid hemorrhage at about 1:30 a.m. on September 1, 1964. She was admitted to a local hospital in a stuporous state but without localizing neurological findings. A lumbar puncture showed bloody cerebrospinal fluid at a pressure greater than 600 mm of water. She was then transferred to Duke Hospital on the evening of the same day.

**Examination.** The patient was lethargic, but would speak in short phrases and would move all extremities equally when stimulated. On the morning of September 2, bilateral carotid arteriograms were performed, and these were thought to be normal (Fig. 2 left and Fig. 3 left). Her blood pressure at that time was 110/80. On September 3 a left retrograde brachial arteriogram was likewise normal. A lumbar puncture done the same day showed bloody and xanthochromic cerebrospinal fluid at a pressure of 200 mm of water. The cerebrospinal fluid pressure on the following day was 160 mm of water. Despite this drop in cerebrospinal fluid pressure and
Fig. 2. Case 2. Anteroposterior views of the right carotid arteriograms performed on September 2, 1964 (left) and September 9, 1964 (right). The first arteriogram is normal. The second shows diffuse spasm of all of the major intradural arteries.

Fig. 3. Case 2. Anteroposterior views of the left carotid arteriograms performed on September 2, 1964 (left) and September 9, 1964 (right). There is an aneurysm of the anterior communicating artery that was best demonstrated in an oblique view on September 9. There is no spasm in the first arteriogram, but the second shows diffuse spasm of all of the major arteries filled.
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progressive improvement in the patient's state of consciousness, bilateral carotid arteriograms performed on September 9 (Fig. 2 right and Fig. 3 right) showed widespread bilateral arterial spasm. This study also demonstrated an aneurysm of the anterior communicating artery that filled primarily from the left carotid injection. Her blood pressure at that time was 120/90.

Course. The patient had a second subarachnoid hemorrhage on September 11 and died 5 days later. At autopsy the suspected aneurysm was verified.

Case 3. A 53-year-old white man had a subarachnoid hemorrhage at about 10 a.m. on July 24, 1964.

Examination. The patient was admitted to Duke Hospital at 1:00 a.m., July 25. At this time he was neurologically normal, and his neck was supple. His blood pressure was 130/80. A lumbar puncture showed grossly bloody cerebrospinal fluid at a pressure of 250 mm of water. Bilateral carotid arteriograms done on July 25 (Fig. 4 left and Fig. 5 left) were normal, as was a left retrograde brachial arteriogram performed on July 27. Lumbar punctures on July 29, August 1, 2, and 3 showed the following opening pressures: 400, 590, 600, and 300 mm of water. The cerebrospinal fluid on August 3 was still xanthochromic. On that same day, bilateral carotid arteriograms were repeated (Fig. 4 right and Fig. 5 right). No aneurysm was demonstrated, but bilateral diffuse cerebral arterial spasm was present.

Course. With repeated lumbar punctures the patient's cerebrospinal fluid pressure returned to normal and his headaches disappeared. He was discharged on August 12 and has remained in good health since that time.

Location of Spasm (Tables 4 and 5). When intracranial arterial spasm was present, it could be seen on all the films made during that particular arteriogram. The intensity and location of the spasm did not appear to change from minute to minute.

As shown in Table 4, intracranial arterial spasm appeared slightly less frequently in association with vertebrobasilar aneurysms than with aneurysms at other sites. Otherwise the distribution of aneurysms in various locations was similar in the patients with spasm and in those with no spasm.

Spasm was more difficult to identify in the posterior than in the anterior intracranial arteries because of dilution of the Renografin or Hypaque by blood from the opposite vertebral artery or from the posterior communicating arteries during retrograde brachial or vertebral arteriography. In addition, in relatively few patients in each diagnostic category was the posterior circulation demonstrated arteriographically. For these reasons, the sites of arterial spasm in the posterior circulation were not studied.

However, in the majority of carotid arteriograms, spasm could be localized to various portions of the anterior intracranial arteries (Table 5). The vascular areas most noticeably involved by spasm were the intradural portions of the internal carotid arteries and the initial segments of the anterior and middle cerebral arteries. Because of the difficulties inherent in the interpretation of differences in the size of small vessels, the branches of the anterior and middle cerebral arteries were not examined for spasm.

In the nine patients with spontaneous subarachnoid hemorrhage in whom bilateral carotid arteriograms demonstrated spasm in relation to a single aneurysm of the anterior communicating artery, the spasm was present unilaterally in seven. The spasm associated with aneurysms of the internal carotid or middle cerebral arteries was primarily ipsilateral to the aneurysm. It never involved contralateral arteries without also involving ipsilateral arteries. Of the nine patients with ruptured vertebral or basilar aneurysms, only one had spasm in the anterior circulation. This spasm was present bilaterally. Similarly, the spasm was bilateral in the cases in whom no aneurysms could be identified.

Electrocardiogram Analysis (Tables 6 and 7). No major differences were found in the EKG patterns among the three groups of patients studied. There did not appear to be any unique features of the electrocardiograms of the patients with intracranial arterial spasm. In all three groups, the nonspecific EKG changes associated with subarachnoid hemorrhage gradually improved with time.

Serial electrocardiograms were performed
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Fig. 4. Case 3. Anteroposterior views of the right carotid arteriograms performed on July 25, 1964 (left) and August 3, 1964 (right). The first arteriogram is normal. The second shows intracranial spasm, especially involving the intradural portion of the right internal carotid artery and the proximal portions of the right anterior and middle cerebral arteries.

Fig. 5. Case 3. Anteroposterior views of the left carotid arteriograms performed on July 25, 1964 (left) and August 3, 1964 (right). The findings are similar to those of the right arteriograms (Fig. 4).
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TABLE 4
Location of intracranial aneurysms

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Internal Carotid</th>
<th>Anterior Communicating or Anterior Cerebral</th>
<th>Middle Cerebral</th>
<th>Vertebrobasilar</th>
<th>Multiple</th>
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<tbody>
<tr>
<td></td>
<td>Right</td>
<td>Left</td>
<td></td>
<td>Right</td>
<td>Left</td>
</tr>
<tr>
<td>Aneurysm, spasm</td>
<td>8</td>
<td>5</td>
<td>10</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>Aneurysm, no spasm</td>
<td>10</td>
<td>11</td>
<td>17</td>
<td>7</td>
<td></td>
</tr>
</tbody>
</table>

on one patient who had repeated preoperative carotid arteriograms showing the disappearance of intracranial spasm. No simultaneous changes were noted in the EKG patterns.

Case 4. A 52-year-old Negro woman with a history of hypertension had a subarachnoid hemorrhage on June 10, 1967. She was comatose on admission to a local hospital. Lumbar puncture on June 13 showed bloody and xanthochromic cerebrospinal fluid at a pressure of 580 mm of water. She gradually improved and was transferred to Duke Hospital on June 17.

Examination. Initially the patient was lethargic and confused, and had nuchal rigidity. From June 17 to July 7, however, she showed progressive improvement. Bilateral carotid arteriograms performed on June 19 demonstrated an aneurysm of the anterior communicating artery that filled only from the left carotid injection. Spasm was present in the proximal portions of both anterior cerebral arteries. Her blood pressure was 140/90 at that time. A left carotid arteriogram on June 26 showed that spasm was still present in the proximal left anterior cerebral artery, though less marked than formerly. Her blood pressure was 130/80. No spasm could be seen on a third left carotid arteriogram on July 3. Lumbar puncture the same day showed the cerebrospinal fluid to be clear and colorless at a pressure of 110 mm of water. Her blood pressure was 120/80.

Course. On July 7, a left frontal craniotomy was performed and the aneurysm was

TABLE 5
Location of intracranial arterial spasm*

<table>
<thead>
<tr>
<th>Location of Aneurysm</th>
<th>No. of Patients</th>
<th>Internal Carotid Artery</th>
<th>Anterior Cerebral Artery</th>
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<tr>
<td></td>
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<td>Right</td>
<td>Extral</td>
<td>Right</td>
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<td>Right middle cerebral artery</td>
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</tr>
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<td>2</td>
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<td>1</td>
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<td>No aneurysm</td>
<td>9</td>
<td>8</td>
<td>0</td>
<td>8</td>
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* In patients with spontaneous subarachnoid hemorrhage and either a single aneurysm or no aneurysm.
clipped successfully. The patient had an uneventful postoperative course and was discharged on July 19.

EKG's. Preoperative electrocardiograms had been obtained on June 20, 21, 22, 23, 26, 27, July 3, and 5. These had shown QT prolongation, T wave distortion, and ST segment depression, which had remained essentially unchanged during the 2-week period. Postoperative EKG's made on July 8, 10, 13, and 14 showed gradual improvement in each of these abnormal findings.

In another case, serial EKG's were made during the development of intracranial spasm. Again, no simultaneous changes were noted in the EKG patterns.

Case 5. A 58-year-old Negro woman with a history of hypertension had a subarachnoid hemorrhage on July 5, 1967. She was admitted to a local hospital in a stuporous state. Lumbar puncture showed bloody cerebrospinal fluid at a pressure of 400 mm of water.

*Examination. The patient was transferred to Duke Hospital on July 6. She had much rigidity but was otherwise normal neurologically. Cerebrospinal fluid pressure by lumbar puncture was 240 mm of water. On July 7 her blood pressure was 150/90, and bilateral carotid arteriograms showed an aneurysm on the proximal portion of the right anterior cerebral artery. No spasm was present.

*Course. On July 10, a right frontal craniotomy was performed and the aneurysm was clipped, as was the right anterior cerebral artery on both sides of the aneurysm. The patient did well for the first 5 days postoperatively, but then gradually became lethargic and developed a left hemiparesis. On July 18 a right carotid arteriogram was performed. There was no filling of the

**TABLE 6**

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>No. of Patients</th>
<th>Avg. Age</th>
<th>Hypertension</th>
<th>Clinical Heart Disease</th>
<th>Abnormal Serum Electrolytes*</th>
<th>Hospital Deaths</th>
<th>Avg. Time (days) SAH to EKG</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. SAH, aneurysm, spasm</td>
<td>12</td>
<td>49.3</td>
<td>7</td>
<td>5</td>
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<td>3</td>
</tr>
<tr>
<td>B. SAH, aneurysm, no spasm</td>
<td>14</td>
<td>49.2</td>
<td>7</td>
<td>7</td>
<td>0</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>C. SAH, no aneurysm, no spasm</td>
<td>9</td>
<td>58.0</td>
<td>6</td>
<td>3</td>
<td>0</td>
<td>5</td>
<td>0</td>
</tr>
</tbody>
</table>

* Measured within 1 day of EKG. Normal values: Na, 135–145 mEq/l; K, 3.6–4.8 mEq/l; Cl, 98–108 mEq/l; Ca, 9.0–10.5 mgm%; CO₂, 24–33 mM/l.

**TABLE 7**

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>EKG (Average Values)</th>
<th>EKG Interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rate</td>
<td>PR</td>
</tr>
<tr>
<td>A. SAH, aneurysm, spasm</td>
<td>85</td>
<td>0.14</td>
</tr>
<tr>
<td>B. SAH, aneurysm, no spasm</td>
<td>70</td>
<td>0.16</td>
</tr>
<tr>
<td>C. SAH, no aneurysm, no spasm</td>
<td>74</td>
<td>0.15</td>
</tr>
</tbody>
</table>

* Nonspecific T wave changes, U waves, or minor arrhythmias.
† EKG evidence of left axis deviation, left ventricular hypertrophy, left ventricular ischemia, subendocardial ischemia, or myocardial infarction.
right anterior cerebral artery; the right internal carotid and right middle cerebral arteries showed definite spasm. Her blood pressure at that time was 130/90. She gradually improved but was still lethargic and had a left hemiparesis at the time of her discharge on August 15.

EKG's. Preoperative EKG's had been obtained on July 8 and 10. Postoperative tracings were made on July 11, 14, 17, 19, 22, 25, 28, August 1 and 15. In each tracing there was evidence of left axis deviation, and there were no significant changes in this pattern between July 8 and August 15.

Discussion

The data presented in this paper are in general agreement with those of previous studies. However, it is unusual to find that in our patients preoperative intracranial arterial spasm was not associated with a significantly increased incidence of hypertension, lethargy, or hospital mortality. It has been our policy to delay operations for aneurysms when intracranial spasm is present, and this may explain in part why the mortality was not greater in these patients.

Intracranial arterial spasm in our patients was seen almost exclusively in association with spontaneous subarachnoid hemorrhage. As in other investigations, it did occur infrequently with craniocerebral trauma and intracranial infections, but then only when the circle of Willis was exposed to blood or inflammation.

According to our data and those of other authors, intracranial arterial spasm can be seen within 1 day of subarachnoid hemorrhage, but it usually is delayed in onset for at least 1 day. It is interesting that of our 20 patients who had a single subarachnoid hemorrhage, two sets of carotid arteriograms showing no intracranial aneurysm, and progressive clinical improvement, eight (40%) developed spasm by the time of the second arteriograms.

The electrocardiographic abnormalities found in some patients with subarachnoid hemorrhage but no apparent heart disease have been well documented during the past two decades. These changes consist mainly of T wave distortions, U waves or T-U fusion waves, prolongation of the QT interval, or elevation of the ST segment. In other words, the electrocardiographic abnormalities point to altered and delayed ventricular repolarization, a pattern that may simulate myocardial infarction. These changes are most marked during the initial period after the hemorrhage and tend to show improvement as the patient recovers. Similar EKG changes have been found to occur in other conditions but most frequently have been described in association with subarachnoid hemorrhage.

Various mechanisms have been postulated to explain these EKG changes associated with subarachnoid hemorrhage, but none is entirely satisfactory. Although coronary arteriosclerosis and abnormal concentrations of serum electrolytes are known to cause similar EKG changes, and are not uncommon in patients with subarachnoid hemorrhage, these two factors in themselves have not provided an adequate explanation. Increased intracranial pressure and left ventricular subendocardial damage have been offered as explanations, but these have not been proven significant. In addition, direct or indirect stimulation of the autonomic nervous system by lesions within the brain or by reflexes arising in the circle of Willis have also been postulated to account for these electrocardiographic abnormalities, which in many ways resemble those resulting from vagal or sympathetic stimulation of the heart. However, changes in cardiac rate and rhythm have not been a prominent feature of the reported preoperative EKG's of patients with subarachnoid hemorrhage. Furthermore, stellate ganglion block, carotid sinus pressure, and intravenous atropine have been found to have little effect on the EKG patterns in such patients.

Because intracranial arterial spasm frequently complicates subarachnoid hemorrhage in the first few weeks, when the EKG abnormalities are most common, we wonder whether the EKG changes and the spasm might be related in some way, such as by a circulating vasoconstrictor or an autonomic phenomenon affecting both cerebral and coronary vessels. However, in the present study there was no statistical relationship between...
intracranial spasm and electrocardiographic abnormalities.

As stated previously, it has been postulated that preoperative intracranial arterial spasm originates locally as a result of arterial distortion and irritation by arterial rupture and perivascular clot formation, and then is propagated to other areas. This is supported by the almost exclusive association of spasm with significant subarachnoid hemorrhage and its primary location in the intradural arteries closely adjacent to rupture aneurysms. Furthermore, the lack of association of spasm with systemic arterial hypertension, electrocardiographic abnormalities, or blood serotonin levels\textsuperscript{27} argues against a generalized neural or humoral etiology. It appears, then, that intracranial arterial spasm begins as a local event, and it is interesting to speculate that substances are elaborated locally, during the process of perivascular clot formation and subsequent clot lysis, that somehow initiate arterial spasm.\textsuperscript{6,13,17,43,58} There has been recent evidence of this in laboratory experiments.\textsuperscript{33,59} and further support comes from the finding that spasm in humans frequently is delayed in onset for several days after a subarachnoid hemorrhage.

Summary

In the present study, various clinical aspects of preoperative intracranial arterial spasm have been analyzed. The charts and arteriograms of 259 patients provided the source of this information.

As in previous studies, spasm was seen most frequently in association with subarachnoid hemorrhage. It was most marked in the major intradural cerebral arteries adjacent to a ruptured aneurysm, and it appeared to last for days once it occurred. From our data, we found no relationship between intracranial arterial spasm and either systemic arterial hypertension or the electrocardiographic abnormalities that accompany subarachnoid hemorrhage. In a significant number of our patients, the onset of spasm was delayed for several days after the subarachnoid hemorrhage.

These findings add further support to the concept that intracranial arterial spasm begins locally at the site of the aneurysmal or arterial rupture, perhaps as a result of substances elaborated during the process of perivascular clot formation and subsequent clot lysis, and then spreads to involve adjacent arteries.

References

Intracranial Arterial Spasm


51. SMEONE, F. A., MURTAGH, F., and RYAN, K. Prolonged cerebral vasospasm: An experi-
134 Robert H. Wilkins, James A. Alexander and Guy L. Odom


