Intracranial arterial narrowing and spasm in acute head injury

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Arteriography in 350 patients with a moderate to severe head injury, including repeated studies in 40 patients, revealed narrowing of one or more of the intracranial arteries in 65 patients (18.6%). Narrowing of the intracranial portion of the internal carotid artery and the first part of the anterior and middle cerebral arteries was found in 18 patients and was believed to be responsible for the clinical symptoms in some. Narrowing of the branches of the cerebral arteries at the site of the cerebral contusion was seen in 33 patients and diffuse narrowing of the intracranial arteries in 12. In two additional patients with gunshot wounds of the brain, there was narrowing of the cerebral artery adjoining a torn vessel. The evidence suggests that vascular spasm is responsible for the narrowing in some patients, while contusion and hemorrhage in the arterial wall is the cause in others. Whatever the mechanism, the occurrence and significance of cerebral arterial narrowing in association with acute head injury needs to be emphasized.

Key Words: acute head injury • cerebral arterial spasm • vascular contusion • arterial narrowing

The importance of arterial spasm in association with subarachnoid hemorrhage from a ruptured intracranial aneurysm is well established. Experimentally, spasm of the pial vessels over the cerebral convexity and of the larger arteries at the base of the brain in cats, dogs, and monkeys occurs after local mechanical stimulation. At neurosurgical operations large intracranial arteries may be observed in spasm following manipulation. In serious head injury, there is an extensive mechanical effect on intracranial structures, and subarachnoid hemorrhage is common. It seems logical to expect spasm of the cerebral arteries in association with acute head injury. Simulated head injury in animals has been shown to produce narrowing of the exposed basilar artery as well as the perforating arteries of the cerebral cortex.

Arteriographic demonstration of arterial narrowing in patients with acute head injury has recently been reported. The incidence of arterial narrowing ranges from 2% to 31%. Wilkins, et al., limited their observations to the major arteries at the base of the brain because of the difficulty they encountered in interpreting differences in the size of branches of the anterior and middle cerebral arteries. Freidenfelt and Sundström, on the other hand, reported cases with spasm of the anterior cerebral and middle cerebral branches as well as other cases with involvement of the intracranial portion of the internal carotid artery.

The present communication is a study of this problem in our patients with head injury.
Material and Methods

We studied the clinical records and arteriograms of patients admitted to the Chulalongkorn Hospital, Bangkok, with head injuries between May, 1962, and June, 1969. Arteriography was performed in 385 head-injured patients, and 350 were included in this study. In 38 patients arteriography was done twice, in one 3 times, and in one 4 times. A total of 393 arteriograms were therefore included. Except for several studies performed by retrograde brachial technique, percutaneous carotid arteriography was done. Conray-280 and, to a lesser extent, Hypaque 50% was used.

It is the policy in our neurosurgical clinic to study by arteriography head-injured patients suspected of having an intracranial space-occupying lesion if the progression is slow and the added time of the procedure is justifiable. Patients who were clinically diagnosed as having cerebral contusion and who did not show the expected improvement were subjected to arteriography to find a possibly correctible lesion. The study was also used in patients with negative burr-hole exploration when hidden hematoma was suspected. The patients in this study, therefore, had a moderate to severe head injury.

Since the middle of 1968 when the spasm and narrowing of intracranial arteries among these patients was recognized, a prospective study has been done in patients who showed narrowing of the intracranial arteries, namely, careful recording of the sequence of injection at arteriography, follow-up cerebral arteriography, and lumbar puncture.

In reviewing the arteriograms, the pattern of narrowing of intracranial arteries was identified. When possible, the caliber of the specific arteries in arteriograms done at different time intervals after the head injury were compared. This proved to be the most reliable indication of any change in the size of the artery. Bilateral arteriograms were available for study in the majority of the patients, and the corresponding arteries on the two sides could be compared. Segmental narrowing of the intracranial portion of the internal carotid artery and diffuse narrowing of major intracranial arteries were usually recognizable in a single film by comparing the caliber of various parts of the arterial tree.

When comparing two arteriograms, the magnification factor and radiographic artifacts such as laminar flow were considered. When there was a question of anatomical variation, arteriosclerotic narrowing, and narrowing due to stretching around a mass lesion, the vessel was considered to be one without traumatic narrowing. Segmental dilatation, occlusion, small aneurysms, and changes in the flow of the cerebral arteries were also encountered but not included in this study.

Results

In 350 patients, 65 (18.6%) were found to have narrowing of one or more intracranial arteries. We have recognized four types of arterial narrowing in association with acute head injuries.

Group A: Localized Narrowing of the Major Cerebral Arteries at the Base of the Brain

Narrowing of the intracranial portion of the internal carotid artery and the first part of the middle and anterior cerebral arteries was found in 18 patients, or 5.1% of the head-injured patients who had arteriographic studies. Their ages ranged from 14 to 42 years, with an average age of 29 years. Table 1 shows the time interval between the head injury and the cerebral arteriography when the arterial narrowing was found. This type of narrowing was seen from the 1st through the 39th day following the injury.

Arteriography was done twice in five patients in this group. Four of them showed narrowing in the first arteriogram done on the 1st, 2nd, 7th, and 11th days after the injury, and the narrowing was absent in the second arteriogram done on the 13th, 15th, 18th, and 23rd days after the injury respectively. In a 23-year-old patient, the first arteriogram done on the third day after injury showed narrowing of the proximal part of the left anterior cerebral artery just after its origin. It was associated with a left temporal intracerebral hematoma. The narrowing could have been mistaken for an anatomical variation. The patient had a temporal craniectomy and removal of the intracerebral clot. He, however, did not improve and the second arteriogram done 4 days later showed narrowing of the intracranial portion of the left internal carotid artery and the proximal portion of both anterior and middle cerebral
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TABLE 1

Time interval between head injury and the cerebral arteriography when the arterial narrowing was found

<table>
<thead>
<tr>
<th>Type of Arterial Narrowing</th>
<th>No. of Cases</th>
<th>No. of Studies</th>
<th>Interval after Head Injury (days)</th>
<th>Summary</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group A: localized narrowing of arteries at base of brain</td>
<td>18</td>
<td>20</td>
<td>1 2 3 4 5 6 7 8 9 10 over 10</td>
<td>maximal interval 39 days</td>
</tr>
<tr>
<td>Group B: localized narrowing of arteries at contusion</td>
<td>33</td>
<td>33</td>
<td>1 2 3 4 5 6 7 8 9 10 over 10</td>
<td>maximal interval 19 days, time not known in 3</td>
</tr>
<tr>
<td>Group C: diffuse narrowing</td>
<td>12</td>
<td>12</td>
<td>1 2 3 4 5 6 7 8 9 10 over 10</td>
<td>maximal interval 20 days, time not known in 6</td>
</tr>
<tr>
<td>Group D: gunshot wound</td>
<td>2</td>
<td>2</td>
<td>1 2 3 4 5 6 7 8 9 10 over 10</td>
<td>time not known in 9</td>
</tr>
<tr>
<td>Total cases of narrowing</td>
<td>65</td>
<td>67</td>
<td>13 14 5 5 1 3 3 0 0 1 13</td>
<td>time not known in 9</td>
</tr>
<tr>
<td>Total no. of studies</td>
<td>350</td>
<td>393</td>
<td>132 49 28 23 5 14 9 2 7 18 84</td>
<td>time not known in 22</td>
</tr>
</tbody>
</table>

arteries. The temporal mass had decreased in size. He then made a slow improvement and no further arteriography was done.

In one 39-year-old man, four arteriograms were made. The patient was admitted in a stupor with a right hemiparesis a few hours after an automobile accident. The first left carotid arteriogram made on the day of admission showed a 16-mm thick subdural hematoma but no arterial narrowing. Temporal craniectomy and removal of the hematoma were accomplished but the patient showed no improvement. The second bilateral carotid arteriogram made on the third day after injury showed residual hematoma in the parietal region. There was again no arterial narrowing. Following reexplanation through a wide craniotomy, he showed some improvement at first and then his condition remained stationary. The third arteriogram on the 30th day after injury revealed narrowing of the terminal portion of the left internal carotid and the adjoining anterior and middle cerebral arteries; a fourth arteriogram on the 39th day was unchanged.

The site of the arterial narrowing in this group of patients is shown in Table 2. The terminal portion of the internal carotid artery was the most common site. Arterial narrowing in one patient began in the proximal anterior cerebral artery and spread to involve the internal carotid and middle cerebral arteries. Narrowing at the intracavernous portion of the internal carotid artery was seen on the first day after injury in another patient who also had a basilar skull fracture involving the sphenoid. The narrowing disappeared in the second arteriogram made 13 days later.

Lumbar puncture was done in 12 patients

TABLE 2

Location of the narrowing of the arteries at the base of the brain (Group A)

<table>
<thead>
<tr>
<th>Location</th>
<th>No. of Occurrences</th>
<th>Bilateral</th>
<th>Right</th>
<th>Left</th>
</tr>
</thead>
<tbody>
<tr>
<td>terminal part of internal carotid artery</td>
<td>12</td>
<td>5</td>
<td>2</td>
<td>5</td>
</tr>
<tr>
<td>intracavernous part of internal carotid artery</td>
<td>1</td>
<td>1*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>anterior cerebral artery</td>
<td>1</td>
<td>1*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>middle cerebral artery</td>
<td>2</td>
<td>1*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>internal carotid, anterior &amp; middle cerebral arteries</td>
<td>3</td>
<td>1*</td>
<td>2</td>
<td>2</td>
</tr>
</tbody>
</table>

* Indicates the same patient at different times.
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TABLE 3
Associated lesions in patients with narrowing of the cerebral arteries as diagnosed clinically and at operation

<table>
<thead>
<tr>
<th>Associated Lesions</th>
<th>Group A: Localized Narrowing at the Base</th>
<th>Group B: Localized Narrowing at Contusion</th>
<th>Group C: Diffuse Narrowing</th>
</tr>
</thead>
<tbody>
<tr>
<td>cerebral contusion with no space-occupying lesion</td>
<td>8</td>
<td>10</td>
<td>7</td>
</tr>
<tr>
<td>cerebral contusion with small space-occupying lesion</td>
<td>2 (1 temporal; 1 basofrontal)</td>
<td>5 (all temporal)</td>
<td>2 (all temporal)</td>
</tr>
<tr>
<td>acute subdural hematoma</td>
<td>4</td>
<td>11</td>
<td>2</td>
</tr>
<tr>
<td>extradural hematoma</td>
<td></td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>intracerebral hematoma</td>
<td>4</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>adjoining fracture of skull</td>
<td>1</td>
<td>4</td>
<td></td>
</tr>
</tbody>
</table>

in this group; 10 were found to have bloody cerebrospinal fluid while the other two had clear fluid. The associated lesions are shown in Table 3. The diagnosis of these lesions was based on the clinical, arteriographic, operative, and postmortem findings. Evidence of injury around the base of the brain and skull was present in seven of the 18 patients.

The following cases were characteristic of this group.

Case 1. A 23-year-old man was rendered unconscious for about 10 min after an automobile accident. He was admitted to a local hospital where he stayed for 24 hrs. After discharge he complained of persistent headache and was referred to us. Examination 2 days after the injury revealed a minimal left-sided weakness and a positive Barré test. There was stiffness of the neck and a positive Kernig's sign. Skull films revealed a linear fracture of the right parietal bone and a diastatic fracture of the lambdoid suture. A bilateral carotid arteriogram showed narrowing of the supracalvarial portion of the right internal carotid artery (Fig. 1A). The inferior margin of the narrowed portion was irregular. The right anterior cerebral artery filled poorly from either the right or left carotid injections. Lumbar puncture released blood-stained fluid with yellow supernatant cerebrospinal fluid. He was treated with repeated lumbar puncture. A second arteriogram 13 days after the first one showed that the narrowing had disappeared (Fig. 1B). The patient made a rapid recovery and has been well during the 5 years' follow-up study.

Case 2. A 21-year-old man was admitted 7 days after an automobile accident in which he was rendered unconscious for a few minutes. Headache and drowsiness had followed but he was still able to walk. On the following day, weakness of the right extremities was noted. His conscious level remained unchanged. The weakness became progressively worse in the following week and he was referred to us. A severe right hemiparesis was found. The first arteriogram, made 7 days after the injury, showed segmental narrowing of the left middle cerebral artery just before its trifurcation. There was no evidence of a space-occupying lesion. He was treated conservatively. The weakness improved spontaneously and rapidly beginning 10 days after the injury. At the end of the third week he was moving all muscle groups and was learning to walk again. The second arteriogram was done 11 days after the first one. It showed that the narrowing was no longer present (Fig. 2).

We believe the smooth circumferential outline of the narrowing and its rapid disappearance within 2 weeks indicate arterial spasm rather than arterial thrombosis. The narrowing was probably responsible for his clinical symptoms which were not typical for contusion or other known lesions.

Case 3. A 40-year-old man was thrown out of an automobile. When taken to a local
hospital, he was found to respond only to pain, and was combative at times. He moved his left arm less than his right one. The plantar response was extensor on the left and flexor on the right. He was referred to us 48 hours after the accident in a more or less unchanged neurological status. Bilateral carotid arteriography under general anesthesia was done without difficulty and showed segmental narrowing of the left middle cerebral artery almost exactly like that in Case 2. There was no space-occupying lesion. Immediately after his recovery from anesthesia, he was found to have a moderate weakness of the right arm and the right side of the face, which improved during the following night. His level of consciousness gradually improved in the following weeks. He showed some dysphasia and mild weakness of the right arm for about 3 months following the injury. In view of the alarming complication on the first occasion, arteriography was not repeated.

We believe that the arterial spasm of the middle cerebral artery was aggravated by the arteriography that precipitated the symptoms of cerebral ischemia.

**Group B: Localized Narrowing of Branches of the Cerebral Arteries at the Site of the Cerebral Contusion**

Narrowing of the branches of the arteries over the cerebral cortex was found in 33 patients, or 9.5% of the head-injured patients in this study. Their ages ranged from 17 to 78 years, with an average age of 34½ years. The narrowing was seen in the arteriograms done 1 to 19 days after the injury.

Arteriography was done twice in 14 patients, all of whom showed changes in the caliber of one or more branches of the cerebral arteries. In 12 of them the artery was larger in the second arteriogram done from 7 days to 2 years after the first one. In the remaining two patients the caliber of the artery was smaller in the second arteriogram done 10 and 12 days following the first study. The time interval between the head injury and the demonstration of arterial narrowing is shown in Table 1.

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Fig. 1. Case 1. Left: Right carotid arteriogram 2 days after an automobile accident showing narrowing of the supraclinoid portion of the right internal carotid artery. Note irregularity at the site of the narrowing and poor filling of the anterior cerebral artery. Right: Right carotid arteriogram 13 days later showing that the narrowing had disappeared.
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The narrowing was found in the branches of the middle cerebral artery in 27 patients and of the anterior cerebral artery in six patients. The associated injuries to the brain are shown in Table 3. In the majority of patients the narrowed vessels were located in the cerebral cortex underlying the subdural or extradural hematoma or the depressed fracture of the skull. The arteries did not appear to be stretched by the mass. Lumbar puncture was done in 13 patients in this group, and the cerebrospinal fluid was bloody in nine, xanthochromic in two, and clear in two.

The following case is characteristic of this group.

Case 4. A 17-year-old boy was hit on the head and during the next 7 days had severe headache, drowsiness, and left hemiparesis which remained unchanged. He was then admitted to the hospital where arteriography revealed a thin subdural hematoma on the right. Following a temporal craniectomy with removal of the hematoma, the patient made a slow improvement. When cerebral arteriography was repeated 10 days later, the subdural hematoma had disappeared. The branches of the middle cerebral artery were definitely smaller in diameter in the first arteriogram (Fig. 3 A and B). The narrowing of the arteries would not have been recognized if the arteriography had not been repeated.

Group C: Diffuse Narrowing of the Cerebral Arteries

Diffuse narrowing of the cerebral arteries was encountered in 12 patients, ages 11 to 71 years (average age, 40½ years). In all but one patient the narrowing involved all intracranial arteries, and the circulation appeared slow. One patient had normal internal carotid arteries and narrowing of the rest of the cerebral arteries. One patient in this group had a second arteriogram 4 months later, which showed the caliber of the cerebral arteries to be definitely larger than in the arteriogram 20 days after the injury. The remaining 11 patients had arteriography only once. The majority of the patients had...
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Fig. 3. Case 4. Right carotid arteriogram done on the day of admission (left) and 10 days later (right) showing a change in the caliber of the branches of the middle cerebral artery (arrow) located at the site of the cerebral contusion.

this type of narrowing in the first 48 hours, but in some it appeared as late as 20 days after the injury. Lumbar puncture was done in eight patients: in four the cerebrospinal fluid was bloody, one was xanthochromic, and it was clear in the other three patients. The cerebrospinal fluid pressure was within normal ranges in three and was moderately increased up to 250 mm of water in five.

Group D: Spasm Associated with Penetrating Injury of the Cerebral Arteries

This type of narrowing was seen in two patients. The first patient was an 11-year-old boy who was accidentally shot with a pistol. The wound of entry was in the middle part of the forehead, and there was no wound of exit. He was unconscious with spasticity of the arms and flaccidity of the legs. Angiography 2 hours after the accident showed an anterior cerebral artery terminating abruptly into a round shadow of contrast material (Fig. 4). There was no filling of both anterior cerebral arteries beyond this point in either the right or left carotid injections. At autopsy the bullet tract was seen entering the right frontal tip, passing through the genu of the corpus callosum into the lateral ventricles. Complete interruption of the left anterior cerebral artery was seen, while the right artery was intact without occlusion or rupture. The failure of arteriographic filling of the right anterior cerebral artery was thought to be due to spasm of the arteries adjacent to the point of active arterial bleeding.

The second patient also had a gunshot wound in the frontal region. Indriven fragments of bone were seen close to an extravasation of contrast material from a pericallosal artery. Segmental narrowing of the pericallosal artery proximal to the point of rupture was seen.

Discussion

Cerebral arterial narrowing occurring in association with head injury is more common and of greater significance than indicated by the few reports on this subject. We believe it is responsible for clinical symp-
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Fig. 4. Left: Right carotid arteriogram in an 11-year-old boy who had a gunshot wound at his right frontal region, showing one anterior cerebral artery terminating abruptly in a round shadow of contrast material. There was no filling of the branches of the anterior cerebral arteries. Right: Line drawing of the same arteriogram. Dotted lines indicate the right anterior cerebral artery which was not filled in the arteriogram but was found to be patent at autopsy.

toms and affects both the course and the prognosis.

Many pathological states are known to be responsible for narrowing of an artery secondary to trauma.

Myogenic Spasm

Spasm of the peripheral arteries is a well-known clinical entity following trauma to the limbs, and its effect can be quite detrimental. \(^1\) Spasm of the intracranial arteries following head injury has been reported by many authors. \(^9,10,15,27\) The supra-cavernous portion of the internal carotid artery is most commonly affected. Subarachnoid hemorrhage in head injury is not uncommon, and the presence of blood in the subarachnoid space can cause arterial spasm. \(^4,14\)

Direct mechanical irritation such as jarring or pulling on the arteries at the base of the brain has been observed to produce spasm. \(^3,19,21\) In serious closed head injury the internal carotid artery, which traverses from skull to brain, would be subjected to the effect of deformation of the skull and discordant craniocerebral movements. Experimental evidence has shown that the arterial spasm produced by mechanical stimulation was transient, lasting only 3 to 35 min, in contrast to the more persistent spasm produced by arterial puncture or subarachnoid blood. \(^5,14,23,24\) Whether the mechanical stimulation produced experimentally can simulate the quantity and quality of mechanical effects of human head trauma remains a moot matter.

Experimental evidence has shown that the peripheral branches of the cerebral arteries are capable of going into spasm. \(^4,16,17\) Spasm has also been reported in the pericallosal artery and the Sylvian branches of the middle cerebral artery close to the area of cerebral contusion. \(^9,10\) In our present series, the narrowing of the artery disappeared in the subsequent arteriograms made as early as 7 days after the first one. The narrowing was smooth and circumferential, and extended over a few centimeters. Myogenic spasm is probably responsible in at least some of these patients.

With laceration of the cerebral arteries, the resulting bleeding could be compared with that from a rupturing aneurysm. The adjoining artery would go into spasm by the
same mechanism. Simeone and his colleagues showed that needle puncture of the cerebral artery consistently produced spasm in monkeys. Our two patients in Group D illustrate this point.

Trauma to the Arterial Wall

Contusion, hemorrhage, and intimal rupture in the wall of the internal carotid artery, resulting in narrowing or occlusion, have been reported secondary to blunt injury to the neck, basal skull fractures, and intracranial injuries. In patients who have persistent narrowing of an artery for a long time after injury, and in those with an irregular outline of the narrowing, actual damage to the arterial wall may be more likely than spasm.

We could not find a logical explanation for the diffuse narrowing seen in patients in Group C.

Cerebral ischemia secondary to the arterial narrowing and spasm can cause neurological deficit. Delayed onset of hemiparesis after a head injury was seen in our Case 2, and was also reported by Jackson and Back. The arterial spasm found in the arteriograms was probably responsible.

Cerebral arteriography appears to carry a risk for patients with cerebrovascular diseases and spasm of the cerebral arteries. One of our patients had an increase in his neurological deficits after the cerebral arteriography, which revealed a narrowing of the middle cerebral artery. The narrowing appeared to be asymptomatic before the arteriographic study.

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References


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