ARTERIOGRAPHIC DEMONSTRATION OF SPASM OF THE INTRACRANIAL ARTERIES

WITH SPECIAL REFERENCE TO SACCULAR ARTERIAL ANEURISMS*

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The percutaneous method of puncturing the carotid artery allows cerebral angiography to be performed on repeated occasions in the same patient. The cerebral arteries thus visualized with the same technic on different occasions generally are identical but sometimes differ strikingly in caliber. When the artery has the smaller caliber it is said to be in arterial spasm. It is the purpose of this paper to present our observations of such spasm and to indicate its possible significance.

MATERIALS AND METHODS

For this study there were available about 400 groups of angiograms made on 350 patients. The patients were mostly adults and suffered from a variety of medical and surgical neurologic disorders. We have found only 12 instances, among 10 of these cases, which have met our criteria of arteriographically demonstrated spasm. This study is devoted to the intracranial portion of the internal carotid artery, its subdivisions, the anterior and middle cerebral arteries and their main branches. It is concerned only with vessels of at least 1.5 mm. in internal diameter.

Arterial spasm can be definitely recognized arteriographically when a vessel is of larger caliber in a subsequent angiogram than was demonstrated at a previous study made under identical conditions. This study is concerned with spasm of only moderate degree, rather than with maximal spasm or total occlusion of the vessel, which obviously cannot be recognized arteriographically. Minimal spasm, with alterations of caliber of less than 0.5 mm., will not be considered because of the difficulties involved in precise measurement.

Marked spasm slows the blood stream. In such cases arteriograms with routine timing show filling of only the proximal portion of the intracranial arteries. Repeating the arteriogram with delayed timing of the exposure (by 1/2 to 5 seconds) will show full arterial filling. When spasm is relieved the velocity of blood flow becomes normal as demonstrated by full arterial filling with routine timing.


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PSEUDOSPASM AND SOME OTHER SOURCES OF ERRONEOUS INTERPRETATION

Nonfilling of a Length of Artery. During the carotid injection of diodrast®, there is probably a slight increase of intra-arterial pressure. However, the instant the injection is over, the pressure in the carotid artery cephalad from the needle becomes less than normal because of the mere presence of the needle within the arterial wall and its lumen. At this instant blood rushes into the injected vessels from the other carotid and from the basilar artery via the circle of Willis. An exposure made at this moment reveals dye in the proximal part of the injected carotid artery and the distal portions of the anterior and middle cerebral arteries, whereas the circle of Willis and the adjacent portions of these vessels are not visualized. This appearance, due entirely to a slight delay in exposure of the arteriogram, has been called pseudospasm.²

Localized Narrowing of Vessels from Causes other than Spasm. The angiographic appearance of cerebral arteriosclerosis is most often that of unusually wide and tortuous vessels. Nevertheless, localized arterial narrowings may be due to arteriosclerosis. These narrowings may appear not only as obvious plaques with almost rectangular edges, but also as gradual taperings similar to the narrowing of spasm. Furthermore, a given vessel may be congenitally narrow. Accordingly we have required a variation in caliber of

Fig. 1. Case 1. Aneurism at bifurcation of left internal carotid artery. Spontaneous subarachnoid hemorrhage Sept. 21, 1950. Arteriograms after left carotid injection. Chamberlain-Towne views: (A) On October 2 with spasm of aneurism itself and adjacent arteries at bifurcation of left internal carotid artery. (B) On November 3 without spasm. Left lateral views: C as A; D as B. Arrows point to aneurism, internal carotid and middle cerebral arteries.
Fig. 2. Case 2. Aneurism of anterior communicating artery. Subarachnoid hemorrhage July 21, 1950 and Sept. 21, 1950. Chamberlain-Towne projection of arteriograms. (A) Left carotid injection; spasm at bifurcation of left internal carotid artery, July 28. (B) Right carotid injection; spasm at bifurcation of right internal carotid artery. (C) Left carotid injection; spasm of left anterior cerebral artery, September 9. Aneurism larger than in B. (D) Left carotid injection after intravenous injection of papaverine hydrochloride and inhalation of amyl nitrite, October 31. No spasm in anterior part of circle of Willis. Fundus of aneurism thrombosed. This was confirmed at postmortem examination. Arrows point to spastic vessels. Compare with Fig. 3.

Fig. 3. Case 2. Aneurism of anterior communicating artery. Lateral arteriograms made after left carotid injection on (A) July 28, (B) September 29, (C) October 31. Note the variation in caliber at the terminal portion of the internal carotid artery, minimal in July. The aneurism is best seen in B. Compare with Fig. 2 A, C, D. Arrows point to internal carotid artery.
FIG. 4. Case 3. Aneurism at bifurcation of right middle cerebral artery. Spontaneous subarachnoid hemorrhage Jan. 4, 1951. Arteriograms made after right carotid injections. Anteroposterior views: (A) January 15; (B) March 20. Lateral views: (C) January 15; (D) March 20; Chamberlain-Towne views: (E) January 15; (F) March 20. All films show the aneurism at the bifurcation of the right middle cerebral artery. The three views made in January all show constriction of the right internal carotid artery, its terminal branches and the aneurism itself. This spasm is absent from the films made in March.

the same vessel as seen on two different films before accepting it as a definite example of spasm.

CASES OF ARTERIOGRAPHICALLY PROVED SPASM

There were 6 cases of untreated saccular arterial aneurism of or near the anterior part of the circle of Willis in which spasm was visualized. In each case there had been spontaneous subarachnoid hemorrhage; twice in Case 2. In 5 cases spasm was present in earlier films and absent from later films; in 1 case the reverse. These 6 cases were selected out of a total of 29 cases where aneurisms had been demonstrated arteriographically. In 18 of these cases there was no evidence of arterial narrowing in the angiograms. In the remaining 5 cases spasm was suggested by narrowed vessels at a single angio-
graphic session but it remains unproved since comparative films are not available.

There were 2 cases of postoperative narrowing of cerebral vessels following carotid ligation in the neck. In one there had been total ligation and in the other (Case 5) partial ligation. We have had 2 additional cases of carotid ligation in the neck (1 total, 1 partial) where postoperative films demonstrated no vasoconstriction when compared with pre-operative. In our cases

![Image](image_url)

**Fig. 5. Case 5.** Aneurism arising at junction of anterior communicating and right anterior cerebral arteries. Spontaneous subarachnoid hemorrhage March 9, 1951. Arteriograms made after right carotid injection, all on the same date. No drugs were administered during angiography. Lateral views: (A) showing practically no spasm; (B) showing marked narrowing of the intradural and subarachnoid course of the internal carotid artery; (C) Chamberlain-Towne view without vasoconstriction; (D) view of right optic foramen showing spasm of the terminal portion of the internal carotid artery, the anterior cerebral artery and the aneurism itself.

of partial ligation the same carotid artery was injected. In those of total ligation the contralateral carotid was injected and the question of narrowing was determined in the middle cerebral artery of the ligated side.

Generally the spasm was maximal in degree at the site of the lesion and extended along adjacent arteries for 1 or more cm. in milder degree. Often the spasm was marked intracranially but was absent in the internal carotid artery proximal to the anterior clinoid process.

In most cases, the difference in caliber was seen by comparing arteriograms made on different days, usually several weeks apart. Except for Case 4 and the influence of drugs, all arteriograms made at the same session showed the same degree of spasm.
DISCUSSION

Physical and Physiological Factors in Production of Spasm. Any change in caliber of the arteries due to the presence of the needle, the pharmacodynamic effect of the dye and the minimal rise of intracarotid pressure from the injection of the dye would have been relatively constant for all cases.

Uncontrolled variables include chemical constituents of the blood such as the circulating hormones and carbon dioxide. However, such factors could not reasonably be expected in themselves to cause localized or unilateral changes in caliber of the vessels. We emphasize the role of the local factor in most of our cases, where all arteriograms made in various projections on the same occasion showed the same localization and degree of spasm.

The Nature of Arterial Spasm. Strange as it may seem, the normal function of the smooth muscle in the walls of the larger arteries has never been elucidated. However, under certain abnormal circumstances, the lumen of the artery narrows and the artery is said to be in spasm. In 20 monkeys a clip was placed on the middle cerebral artery to occlude the vessel. In 10 of these cases spasm appeared in the internal carotid artery and lasted from 1 to 10 minutes. In a case of suprasellar cyst in a child where the frontal lobe was elevated, one of us (A.E.) has seen severe spasm of the internal...
carotid artery, apparently as the result of traction. Dr. Robert Bassett on two occasions has gently touched the internal carotid artery in its subarachnoid course. This procedure produced spasm of the artery and allowed him to apply a metal clip which previously had been too small. It is not known for certain whether this spasm is due to the muscular or the elastic tissue or to both, or what factors determine the degree or duration of the contraction.

The linear extent of the spasm may vary from 1 to several cm. This is made understandable by recalling that both muscular coats of an artery are basically spiral. The so-called longitudinal muscular coat is a long spiral and the so-called transverse muscular coat is a short spiral. The duration of the spasm may be brief or more prolonged. It lasts at least a few months, in some cases following ligation or the “near miss” of a high velocity missile.

In general, the stimulus producing spasm seems to be traction on the artery. It may be longitudinal or lateral traction, the application of a ligature or metallic clip, or the impaction of a solid embolus. The production of arterial spasm by a fractured bone is presumably of the first type, by a high velocity missile of the second. We have seen spasm most frequently in cases of aneurism and its presence suggests traction on the vessel wall.

Other conditions where we have seen narrowing of the anterior and/or middle cerebral arteries strongly suggestive of spasm are: acute severe internal hydrocephalus, severe cerebral edema, intracerebral hemorrhage, malignant hypertension without arteriosclerosis, and the syndrome of “cerebral collapse” (presumably due to oligemia and predisposing to subdural hematoma). Spasm in the femoral artery has been produced experimentally by acute severe arterial hypotension. Spasm probably occurs in association with arteriosclerosis and with thrombosis of the internal carotid artery and its larger branches. The mechanism of spasm from intrinsic lesions of the arterial wall is unknown.

*The Role of Spasm in Cases of Aneurism.* It is probable that arterial spasm plays an important role following spontaneous rupture of saccular aneurisms of or near the circle of Willis. If there were no reactive contractile force in the wall of the aneurism or adjacent artery, the original pin-point leak would be followed by a large tear. Then, instead of only a few, all of these patients would die from the first hemorrhage. According to this hypothesis, the cases that are immediately fatal are those where the arteries do not go into spasm.

We have reviewed all our cases of angiographically proved intracranial arterial aneurisms with subarachnoid hemorrhage and without surgical treatment. There were 12 instances of hemorrhage (among 11 patients) where arteriograms were made 23 days or earlier after the hemorrhage. In each case except 1 there was demonstrable arterial narrowing (proved to be spasm in 7 instances by later normal angiograms). On the other hand, in another group of 10 cases where arteriograms were made 26 days or longer after the hemorrhage, there was no evidence of spasm. These data seem to support the suggestion that arterial spasm occurs after rupture of an intra-
cranial aneurism. Persistence of vasoconstriction may indicate continued or additional stretch of the aneurismal wall. Certainly spasm disappeared after a few weeks in those of our patients who survived.

Furthermore, spasm may play a considerable role in the production of intra-aneurismal thrombosis, as in Cases 2 and 4. Finally, excessive spasm may produce unfavorable effects by impairing the circulation of the brain in the area nourished by the affected artery.

On the other hand, we have had 3 cases of large unruptured aneurisms of the circle of Willis, all without evidence of arterial constriction.

**SUMMARY**

Spasm of the larger intracranial arteries (internal diameter at least 1.5 mm.) has been demonstrated in 12 instances among 10 cases by means of comparative arteriograms. The affected vessels were the internal carotid, anterior cerebral, and middle cerebral arteries and their branches.

Seven examples of spasm occurred in 6 cases of saccular arterial aneurism of or near the circle of Willis. These were selected from a total group of 29 cases of angiographically demonstrated aneurisms that had recently ruptured. Arteriograms made soon (23 days or earlier) after a subarachnoid hemorrhage from such an aneurism generally revealed arterial spasm; those made later (26 days or more) revealed none.

Other instances of spasm were associated with: total or partial ligation (1 each) of the carotid artery in the neck, postoperative astrocytoma, localized intracerebral hemorrhage and edema, and severe intrinsic lesion of the arteries.

The common element in the production of spasm in all cases seemed to be abrupt traction on the arterial wall. Usually the spasm was maximal at the lesion but extended several cm. along adjacent arteries in lesser degree. Except for Case 4, all arteriograms made at the same session revealed the same location and degree of spasm.

**REFERENCES**