Experimental and Clinical Study of the Development of Spasm of the Cerebral Arteries Related to Subarachnoid Hemorrhage

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One of the most severe complications of the rupture of aneurysms is the resultant spasm of cerebral arteries leading to ischemia, edema, and infarction of large areas of the brain, including some remote from the rupture. It may be postulated that the leading role in the development of the arterial spasm is played by the subarachnoid hemorrhage inevitably accompanying the rupture of an aneurysm. That is why the problem of arterial spasm is so important in surgery of arterial aneurysms of the brain. Nevertheless, our knowledge of the origin and control of arterial spasm is amazingly limited.

The complexity of the problem is aggravated by the fact that its manifestations in individual patients against the uniform background of a subarachnoid hemorrhage vary greatly. Angiographic examinations prove that the arterial spasm may develop immediately after rupture of an aneurysm, as well as several days later. The spasm is usually limited to the main artery on which the aneurysm developed, but it may also spread over the arterial tree, and even to other arteries of the circle of Willis or the contralateral hemisphere. Moreover, after a hemorrhage the arteries surrounded by blood are not always spastic, while spasm may be noted in arteries not surrounded by blood. Finally, the spread and severity of arterial spasm are also not directly dependent on the extent of the hemorrhage or the size, shape, or site of the arterial aneurysm.

The spasm may last several hours or disappear several minutes after intra-arterial injection of various antispasmodic drugs; it may also persist for many days or even weeks. A sequence of factors often underlies the variations in development, intensity, extent, and duration of the arterial spasm both after surgery and after angiography. These factors include reactions peculiar to the individual such as those of the vascular system, and the patient's age and general state of health.

The following three cases were followed both in the acute and late periods (after surgery and after angiography without operation); they illustrate several of these factors rather convincingly.

Case Reports

Case 1. This 30-year-old woman complained of reduction of vision, especially in the left eye, of 2 years' duration. The patient showed a chiasmal syndrome with predominant damage to the left optic nerve plus hyperactive tendon reflexes in the right arm. Pneumocisternography showed caudal displacement of the chiasmal cistern. Carotid angiography showed leftward displacement of the supracoiloid section of the left internal carotid artery, and sharp narrowing of the lumen of the A-1 section of the left anterior cerebral artery (Fig. 1 left). The diagnosis of a tumor of the tuberculum sellae with direct pressure on the left optic nerve and left internal carotid was verified at operation and total extirpation was accomplished.

Angiography performed 6 days postoperatively revealed a considerable narrowing of the supracoiloid part of the left internal carotid artery (Fig. 1 center). The neurological status, however, showed only a slight increase in the severity of the general cerebral symptoms. Angiography repeated 2 months postoperatively demonstrated almost complete disappearance of the spasm of the internal carotid artery (Fig. 1 right).

Case 2. This 36-year-old man with recognized primary hypertension suffered a sub-
arachnoid hemorrhage without loss of consciousness 3 months prior to admission to the Institute; a paralysis of the third left nerve developed soon after the hemorrhage. There was paralysis of the third left nerve, reduction of the left corneal reflex, weakness of the seventh nerve, and slight predominance of the right tendon reflexes. Carotid angiography demonstrated an aneurysm of the supraclinoid part of the left internal carotid artery with good collateral flow via the anterior communicating artery. The aneurysm was isolated by clipping its neck. Bleeding from the temporopolaris vein was controlled by clipping the venous ostium and applying a piece of muscle. Mild speech disturbances developed after the operation, and right pyramidal signs increased. By the 7th postoperative day the state of the patient had become still worse, with general cerebral symptoms and increasing pyramidal signs and aphasia. Angiog-