Intracranial Arterial Spasm:
A Clinical Analysis*

ROBERT H. WILKINS, M.D., JAMES A. ALEXANDER, M.D.,
AND GUY L. ODOM, M.D.

Division of Neurosurgery, and the Department of Surgery, Duke University Medical Center,
and the Durham Veterans Administration Hospital, Durham, North Carolina

It has become apparent during the past two decades that patients suffering from spontaneous subarachnoid hemorrhage frequently also have intracranial arterial spasm, a complicating factor that adversely affects their neurological status and prognosis. Despite the common occurrence of this phenomenon, its exact etiology and pathogenesis are not known.

Intracranial arterial spasm in humans can be induced by mechanical stimuli such as surgical trauma to the cerebral arteries, but the mechanisms that produce preoperative spasm are less well understood. This type of spasm has been found to have the following characteristics: it is seen most frequently in association with subarachnoid hemorrhage; it is usually most marked in the major cerebral arteries ipsilateral to a ruptured aneurysm and involves primarily the parent artery; it is almost exclusively an intradural phenomenon; it usually lasts for days to weeks and does not seem to vary in severity from minute to minute; and it does not appear to be caused by arteriography. Because of these clinical features, as well as the results of various laboratory investigations, it has been postulated that preoperative intracranial arterial spasm originates locally as a result of arterial distortion and irritation by aneurysmal rupture and perivascular clot formation, and is propagated to other areas by the nerves and smooth muscle fibers of the involved cerebral arteries.

Our recent clinical experience with intracranial arterial spasm, as analyzed in the present paper, adds further support to this concept.

Received for publication December 18, 1967.

* Supported in part by U. S. Public Health Service Grants HD-00668-09, NB02369, and NB04368 and a grant from the Walker P. Inman Foundation.

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 atheromas, 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-
aneurysm 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 angiography. In 10 of these, with negative carotid arteriograms, aneurysms of the verteobasilar system were found.

Each of the other 29 patients with spontaneous subarachnoid hemorrhage had several angiographic 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 cranio-cerebral trauma, bilateral opacification of the cerebral arteries was accomplished by manual compression of the contralateral carotid artery during angiography. 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 cranio-cerebral 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-