THE NONFILLING PHENOMENON DURING ANGIOGRAPHY IN ACUTE INTRACRANIAL HYPERTENSION
REPORT OF 5 CASES AND EXPERIMENTAL STUDY*

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(Received for publication April 23, 1962)

NONFILLING of the cerebral vessels during carotid angiography in cases of acute intracranial hypertension is a recognized but poorly understood clinical fact. These patients usually are moribund and often require artificial respiration throughout arteriography. The angiographic picture of acute intracranial hypertension resembles that of thrombosis of the internal carotid artery. Collateral circulation through the external carotid artery sometimes is demonstrated in cases of thrombosis but never in acute intracranial hypertension. In the presence of great intracranial pressure the internal carotid artery usually does not fill with contrast medium beyond the carotid siphon intracranially; while the external carotid artery and its branches are well demonstrated. Because patent internal carotid arteries have been found at necropsy, the mechanism of this nonfilling phenomenon has been unknown.

Riishede and Ethelberg reported 5 patients with intracranial space-occupying lesions in whom this angiographic picture was observed. They concluded that the nonfilling phenomenon was the result of decreased cerebral blood flow secondary to herniation of the brain by the severely increased intracranial pressure.

Horwitz and Dunsmore reported 4 similar cases. They also related the angiographic findings to acutely increased intracranial pressure and postulated that reflexes mediated by the diencephalon had altered the intracranial circulation.

Newton and Couch presented 1 case of pseudo-occlusion of the internal carotid artery as demonstrated by angiography. Massive intracerebral and intraventricular hemorrhages and patent internal carotid arteries were found at necropsy.

Pribram reported 11 well documented cases of acute intracranial hypertension. He postulated that in some of his cases the abrupt rise in intracranial pressure could have been caused by a blood clot or tentorial herniation blocking the aqueduct of Sylvius. After tapping the ventricles in 1 patient, he was able to demonstrate the intracranial vessels by arteriography. He surmised that an acute rise in intracranial pressure was responsible for the nonfilling phenomenon, and he suggested that in similar patients lowering of the intracranial pressure would permit visualization of the cerebral vessels.

In the past 12 months, 5 patients have been seen at the North Carolina Baptist Hospital with acute intracranial hypertension in whom the cerebral vessels failed to fill during carotid arteriography. In each instance the needle was placed correctly within the lumen of the vessel, but only the common and external carotid arteries filled well with contrast medium.

CASE REPORTS

Case 1. A 19-year-old man was in an automobile accident a few hours before admission. He was in
deep coma when brought to the hospital and tracheostomy was performed in the Emergency Room.

Bilateral carotid angiograms showed apparent obstruction of both internal carotid arteries at the level of the siphon (Fig. 1A). The patient ceased spontaneous respiration during the procedure and died soon afterwards.

Postmortem examination was not obtained.

Case 2. The day before admission a 33-year-old white woman complained of intense headache and nausea, accompanied by weakness of her right arm and dysphasia. On the day of admission she had a generalized seizure, extensor rigidity developed, and she was admitted in coma. Both pupils were dilated and paralyzed and she stopped breathing in the Emergency Room. Intubation was performed and artificial respiration was initiated.

A right ventricular tap revealed the pressure to be greater than 600 mm. of water. Air was injected and roentgenograms showed deviation of the right lateral ventricle to the right. A left ventricular tap and injection of air revealed poor filling of the left lateral ventricle.

Bilateral carotid arteriograms then were done and showed nonfilling of the intracranial portion of both internal carotid arteries (Fig. 1B).

The patient died the day following angiography and postmortem study revealed a left frontotemporal abscess and herniation of the cerebellar tonsils. The internal carotid arteries were patent.

Case 3. An 11-year-old white boy was admitted to the hospital because of coma. Six years previously the patient underwent a Blalock procedure for tetralogy of Fallot and he had done well until 5 days prior to the present admission. At that time he complained of a left frontal headache with nausea and vomiting, and he was treated symptomatically with improvement. The night before admission he complained of a severe headache. He went to bed but the next morning he could not be aroused and was brought to the hospital.

On physical examination the patient was comatose, areflexic, and flaccid, with positive Babinski's sign bilaterally. Pupils were dilated and paralyzed and bilateral papilledema was present. Respiration was irregular and he was mildly cyanotic.

Bilateral twist-drill holes were placed in the frontal region and needling on the left yielded clear fluid under increased pressure. Twenty cc. of ventricular fluid were withdrawn; air injected into this region was not shown on roentgenograms. Bilateral carotid arteriograms demonstrated no intracranial filling, although there was good filling of the common and external carotid arteries.

Bilateral occipital burr holes then were made and tapping on the left side yielded cloudy fluid