AVOIDANCE OF FALSE ANGIOGRAPHIC LOCALIZATION OF THE SITE OF INTERNAL CAROTID OCCLUSION

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Surgical attempts to re-establish circulation to the brain after occlusion of the carotid artery\(^1-3\) have made localization of the exact site of obstruction an absolute necessity. Recognition by angiography of the actual point of occlusion if it lies in the cervical portion of the internal carotid artery is not difficult. Usually this site is represented by a blunt end of the opaque column or by a complete absence of the internal carotid artery. However, if the opaque column tapers gradually to a slender point, this does not necessarily represent the site of occlusion. It may, and in fact usually does, represent the advancing end of the opaque column proceeding slowly toward an obstruction higher in the artery, usually in the intracranial portion. Therefore, a single exposure or even two exposures made primarily for the neck during or shortly after the injection may be misleading. Postmortem findings in Case 1 alerted us to this fact and by simply making greatly delayed serial exposures, we have been able to localize accurately the proximal end of complete or nearly complete obstructions, wherever they may be.

METHODS

In all cases the contrast material used was 50 per cent Hypaque. Manual injection of from 6 cc. to 8 cc. as rapidly as possible through a \#18 spinal needle is employed.

At the Baptist Memorial Hospital a simple device takes four lateral films during one injection over a period of about 3 seconds, and similarly three anteroposterior films. At the City of Memphis Hospitals two laterals are exposed, the last after manual removal of the first cassette. The interval is about 1 second. Anteroposterior films are routine, but only one exposure is made per injection. Diagnosis of the site of obstruction is attempted on the lateral films as a rule.

Patients are medicated with Nembutal and a mild analgesic, usually codeine; general anesthesia is employed only in pediatric cases. Procaine, 1 per cent, is infiltrated at the site of puncture and around the common carotid artery. Five cc. is the amount usually necessary.

Exposure time is either one-fifth or one-tenth second. The first film is

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taken near the end of injection, which itself requires 1 to 1\frac{1}{2} seconds. If a gradually tapering opaque column on routine arteriogram is seen, then an additional injection is made in which exposures are delayed up to 6 seconds.

CASE REPORTS

Case 1. JGH #281945. A white female housekeeper apparently in her late fifties was found comatose in her room. History was not immediately available. Blood pressure was 220/110; pulse rate was 96. Fundi showed hypertensive changes and she responded to pain with movements of the right side only. The left hemiplegia included the lower face.

Spinal fluid was clear; pressure was 146 mm. of water.

Forty-one hours later carotid angiograms showed no opaque medium in the right internal carotid artery above a point a few centimeters above the bifurcation of the common carotid artery (Fig. 1). Exploration of this area was planned, but the patient expired before this could be carried out.

Autopsy revealed generalized arteriosclerosis and a grey, adherent thrombus in the intracranial portion of the right internal carotid artery at the bifurcation, extending into the middle cerebral artery. The right hemisphere was infarcted. There was no antemortem thrombus in the cervical portion of the internal carotid artery.

Case 2. JGH #288782. A 19-year-old colored female was well until the morning of admission when she vomited and became incontinent and aphasic.

Physical examination disclosed a left hemiparesis, aphasia, normal blood pressure and a pulse rate of 48. Spinal fluid was clear with a pressure of 230 mm.

Twenty-eight hours after the onset of symptoms, carotid angiography showed at first an apparent obstruction of the right internal carotid artery in the neck (Fig. 2a). By delaying exposure up to 6 seconds from the completion of injection, the opaque column was seen to progress cranial to a few millimeters beyond the origin of the ophthalmic artery (Fig. 2b). There was slight filling of the right anterior cerebral artery from the contralateral side.

A few hours later, under local anesthesia, the right internal carotid artery was opened above its origin in the neck in hopes that suction from below plus pressure from across the circle of Willis might dislodge the obstruction. A slight, nonpulsatile retrograde ooze appeared from above. A #8 French catheter was inserted into the artery as far as possible and suction was applied without result.

Through a right frontotemporal craniotomy, the middle cerebral artery was exposed and found to be nonpulsatile. The contiguous cortex was of normal color, but quickly became cyanotic with retraction. A firm plaque was palpable within the internal carotid artery at its bifurcation, apparently occupying the entire lumen. After occluding the middle cerebral artery at an accessible location, saline was injected into the middle cerebral artery proximal to the clamp through a #22 spinal needle while suction was applied again to the catheter below in an attempt to dislodge the obstruction, without success. On removing the needle, bleeding from the artery was slight and easily controlled with Gelfoam. No attempt at intracranial endarterectomy was made. The patient died on the 3rd postoperative day.

At autopsy the lumen and lining of the right internal carotid artery were grossly normal in its cervical portion. The middle ear on this side was purulent. The distal centimeter of the internal carotid artery showed narrowing of the lumen and calcified intimal plaques. A solid clot occluded this portion of the artery just distal to the ophthalmic artery and extended into the middle cerebral artery, occluding it.