Observations on the Tolerance of the Intracranial Arteries to Catheterization*

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Reversible segmental or diffuse narrowing of the larger cerebral arteries has been documented in the following circumstances: 1) direct mechanical, chemical or electrical stimulation,4,10 2) traction during surgery,9 3) subarachnoid hemorrhage from saccular aneurysms,1,7 and 4) secondary to angiographic contrast media when the arteries are in a state of hyperirritability.11 Further we have observed, angiographically, segmental narrowing persisting for weeks at the sites of applications of clips in postoperative patients with saccular aneurysms. In conditions with more generalized cerebral arterial involvement and slowing of the cerebral-circulation time, such as acute increased intracranial pressure,8 cerebral trauma,8 systemic hypertension and inhalation of hyperbaric oxygen,8 the importance of arterial spasm is less certain.

In a recent communication relating to the effects of other forms of arterial trauma, the responses of the larger cerebral arteries to the direct and saltatory passage of spherical plastic emboli were described.9 This technique of artificial embolization has been useful in the treatment of certain inoperable arteriovenous malformations and the associated intraluminal trauma, similar to pathological embolization, does not induce spasm. Moreover, evidence indicates delayed proximal and distal dilatation may be a characteristic response. However, extension of this technique to include manipulation of delicate catheters or emboli within the intracranial arteries, as conceivably useful for the urgent treatment of certain bleeding saccular aneurysms, would create much greater intraluminal trauma and possibly spasm similar to that following stimulation of the external wall. Further potential obstacles to catheterization are extreme tortuosity of the arteries of the cervical trunk and possible occlusion or thrombosis of the arteries by the catheter itself.

After preliminary laboratory work a satisfactory method for catheterization has been devised, and reported here with are initial clinical observations of the arterial effects. These include maneuvering a catheter beyond the circle of Willis, inflation of the tip of a catheter within the terminal internal carotid artery and intraluminal occlusion of an aneurysmal orifice during acute increased intracranial pressure with subarachnoid hemorrhage.

Experimental Background

Forming the average configuration of an internal carotid artery are five sites of major angulation with a sixth site proximal to the carotid canal in approximately 5 per cent of patients. A scale glass model of the internal carotid artery was designed and with this various conventional and improvised plastic and rubber catheters were tested. The catheters which passed most easily through this model were tested in cadavers. In only a few instances catheters with very flexible tips could be passed to the terminus of the internal carotid artery. Mostly, there was a tendency for the tip to form a pocket in the wall of the artery at the acute anterior angulation of the siphon. Occasionally, the catheters ruptured the thin arterial wall at this site. For these reasons it was decided that an embolus must be used to carry the catheter.

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Fig. 1. External glass catheterization chamber.

As shown in Fig. 1, initially the catheter is coiled in the glass bulb in a length permitting passage to the predetermined site. The connecting tubing to the artery then is opened and with gentle irrigation the embolus leading the catheter passes into the arterial lumen and is carried by the flow of blood the full length of the introduced tubing. The connecting tube is then clamped and the bulb is irrigated through a side arm to remove the accumulated blood. If additional distance of catheterization becomes necessary, more tubing is introduced into the bulb and the process is repeated. With this apparatus a 2.5 mm. spherical embolus could carry a flexible Silastic tube accepting a #22 needle. It was perfected in dogs using the common carotid artery as a side arm and catheterizing into the thoracic aorta.

Clinical Observations

Case 1. A 33-year-old housewife and waitress was admitted to Georgetown University Hospital on Jan. 20, 1962 for treatment of a cerebral arteriovenous malformation.

She had been well until 5 years previously when she noted a tendency to drag the left foot followed by progressing weakness gradually including the entire leg, and within 1½ years, the left arm and face. Bilateral carotid angiograms (Dr. Paul M. DeLuca, Endicott, N.Y.) demonstrated an arteriovenous malformation in the midportion of the right hemisphere. It was considered inoperable. One and a half years prior to admission she had a generalized seizure and thereafter remained on anticonvulsant medication. During the year before admission she experienced frequent episodes of transient "numbness" of the left arm and leg and occasional throbbing, generalized and right-sided headaches. The left hemiparesis forced her to curtail work as a waitress.

Examination. Blood pressure was 110/70. She was alert and had no dysphasia. A systolic murmur was audible over the left eye. There was a left spastic hemiparesis involving equally the face, arm and leg. Grip in the left hand was 50 per cent of normal, and her left ankle tended to invert after 4 or 5 steps. Strength in extension of the fingers was only 10 per cent of normal. Sense of position and graphesthesia were severely impaired in the left hand, and there was extintion with simultaneous stimulation over the left arm and leg. The left arm and leg were about 2 to 3° F. colder than the right.
Routine laboratory findings were normal. An electroencephalogram was normal. Roentgenograms of chest and skull were normal. There was no cardiac enlargement.

Bilateral, serial carotid angiograms demonstrated an arteriovenous malformation, the nidus of which occupied a \(4 \times 4 \times 3\) cm. zone in the right insula and sylvian fissure. It was supplied primarily by enlarged sylvian branches and penetrating branches from the middle cerebral trunk. A medial portion of the malformation, in the region of the internal capsule, filled from the left side across the anterior communicating arteries to the proximal right middle cerebral trunk.

**Operation.** The malformation was embolized in two stages using the right internal carotid artery. At the first procedure 30 spherical Silastic emboli containing metallic radiological markers and measuring 2.5 mm. and 3.0 mm. were introduced stepwise with radiological control. This eliminated a large portion of the sylvian contribution. During the second stage the responses of the middle cerebral trunk and terminal internal carotid artery to an embolus held stationary and manipulated over a short distance were tested. A 2.5 mm. embolus attached to a 4-0 silk suture was introduced and permitted to pass to a point a few mm. proximal to the internal carotid bifurcation. Angiography showed no local or generalized alteration in the diameter of the adjacent arteries (Fig. 2). The embolus was easily retrieved aided by momentary reversal of the flow by clamping the proximal common carotid artery. A 3.0 mm. embolus was then introduced to a point in the middle cerebral trunk and maneuvered back and forth over several mm., again without angiographic evidence of arterial reaction. After adding more length of attached suture the embolus passed into the nidus of the malformation. Gentle traction on the suture could shift the position of the embolus over a short distance, but the traction necessary for retrieving provoked immediate narrowing of the terminal portion of the internal carotid artery. Therefore, the suture was cut and the freed end was permitted to float into the malformation.

**Postoperatively** the patient's neurological status was unaltered but she complained of severe right retro-orbital pain which persisted for approximately 3 weeks before subsiding abruptly.

During 6 months after discharge the disability in use of left hand progressed slightly. Therefore, she was readmitted and at a third stage of embolization the terminus of the middle cerebral trunk was occluded with three 3.5 mm. emboli. She has been followed for 10 months thereafter. She has resumed working as a waitress and feels that progression of her disability has ceased. She has had no recurrent headaches.

**Comment.** These observations suggest that a catheter led by an embolus can be maneuvered through the terminal internal carotid artery and middle cerebral trunk without inducing spasm of the adjacent arteries and from these locations may be retrieved easily when aided by momentary reversal of flow in the internal carotid artery. However, the traction necessary to remove the catheter from a site distal to the middle cerebral trunk probably distorts the terminus of the internal carotid artery and spasm at this site promptly ensues.

**Case 2.** A 51-year-old housewife was admitted to Georgetown University Hospital on Sept. 10, 1962 for treatment of a cerebral arteriovenous malformation.

At 14 years of age she became aware of incoordination of the left hand. Over the ensuing years this gradually increased to a left spastic hemiparesis. At age of 21 years she had the first of a series of focal tonic-clonic seizures, usually starting in the left index and middle fingers. Recurrent seizures were controlled on anticonvulsant medication, but in recent years she experienced
frequent sensory seizures in the left arm and leg. Her most disabling symptom was daily right-sided throbbing headache. Through much of the day she carried an ice bag on the right side of her head claiming this offered some relief.

One year prior to admission a carotid angiogram at Mt. Sinai Hospital, Detroit (Dr. Abe S. Goldstein) demonstrated a large arteriovenous malformation and in April 1963 she was referred to the National Institute of Neurological Diseases and Blindness. More angiographic studies revealed an aneurysm of the junction of the posterior communicating artery and the right internal carotid artery which was the major feeding artery to the malformation. These lesions were judged inoperable, and she was referred to Georgetown University Hospital.

Examination. Blood pressure was 115/80. General physical findings were within normal limits except for a Grade II blowing systolic murmur at the apex. There was left hemiatrophy involving mostly the extremities. A blowing continuous murmur was audible over both eyes and temples, more pronounced on the right. There was a left spastic hemiparesis with greatest involvement of the arm. She was barely able to use the left hand for eating and combing her hair. There was slight impairment in graphesthesia in the left hand.

Routine laboratory findings were normal. Roentgenograms of the skull showed slight thickening of the calvarium and increased vascular markings in the right frontal bone. A rheon-cephalogram indicated abnormal cerebral circulation in the right frontal area and possibly deficient circulation in the territory of the right anterior cerebral artery.

Serial right carotid and vertebral angiography were performed (Fig. 3). The nidus of the malformation measured 4×5 cm. It occupied the posterior frontal cortical area and extended medially in a conical shape to the lateral ventricle. There was no significant angiographic contribution from the basilar circulation. The medial portion of the malformation was supplied by both anterior cerebral arteries which angiographically filled from the left side.

Operation. All seven of the 3.5 mm. emboli employed were arrested at proximal sites in the feeding sylvian arteries, completely eliminating the middle cerebral contribution to the malformation. A catheter consisting of delicate flexible Silastic tubing with an enlarged inflatable tip was then introduced. Using Polaroid films the tip was maneuvered to the intraluminal orifice of the saccular aneurysm and inflated with 80 per cent Hypaque to its maximum of 6 mm. (Fig. 4). The inner arterial diameter at this site had been measured at 5 mm. on previous angiographic films. Angiography with the catheter in place showed that the diameter of the artery had increased at the site of the balloon permitting passage of the medium around it and faint filling of the aneurysm. The balloon was permitted to deflate and the catheter was withdrawn with brief proxima

![Fig. 3 (left). Large arteriovenous malformation of right posterior frontal lobe with an associated saccular aneurysm arising from internal carotid artery.](image1)

![Fig. 4. (right). Same case as in Fig. 3, showing the tip of a catheter inflated to a diameter of 6 mm. with 50 per cent Hypaque at intraluminal orifice of the saccular aneurysm. Metallic markers indicate emboli placed previously in major feeding arteries of malformation.](image2)
occlusion of the common carotid to reverse the flow in the internal carotid artery. The internal carotid was then permanently occluded in continuity with a tantalum clip.

Course. During the 3rd and the 4th postoperative days there was slight increase in the spasticity of the left hand with moderate increase in the cortical sensory loss. This, however, cleared within a few weeks. There was immediate alleviation of the former pain in her head, and by 6 months later she had not had any significant headache and was carrying on her prior activities.

Comment. The measurements made from angiograms indicate that the terminal internal carotid artery dilated slightly in response to rapid segmental intraluminal distention. The maximum safe inflation of the balloon had been reached precluding testing of the reaction to firmer distention and complete occlusion of the artery.

Case 3. A 31-year-old housewife was admitted to Georgetown University Hospital 1 hour after the abrupt onset of a subarachnoid hemorrhage. She became unconscious within a few minutes after onset.

 Examination 1 hour later revealed that blood pressure was 100/60, pulse rate 110, and respiratory rate 30. She was unresponsive to painful stimulation with equal but unresponsive pupils and flaccid extremities.

Lumbar puncture showed a pressure of 400 mm. of cerebrospinal fluid and the fluid was bloody grossly. Bilateral carotid angiograms showed a saccular aneurysm at the junction of the internal carotid and ophthalmic arteries on the left, 3 mm. distal to the anterior clinoid process (Fig. 5). A few hours after admission she became bilaterally decerebrate. A tracheotomy was performed. Her blood pressure gradually declined and an intravenous infusion of Aramine was used to maintain it at 90 mm. Hg systolic.

Operation. About 12 hours after admission the bifurcation of the left common carotid artery was exposed and the catheterization bulb was joined to the external carotid artery. As the preliminary maneuver a spherical Silastic embolus, 2.5 mm. in diameter, with an attached suture was introduced to measure the precise distance to the aneurysmal orifice. When the embolus was held stationary at the intraluminal orifice it passed into the neck of the aneurysm (Fig. 6). Angiography immediately thereafter showed contrast medium passing the site of the aneurysm and filling the intracranial arteries without filling the aneurysm (Fig. 7). There was slight narrowing of the internal carotid artery at a site 3 mm. distal to the obliterated aneurysmal orifice. Angiography was repeated twice during the interval of an hour that the embolus remained in place and on each occasion revealed similar findings. With removal of the embolus there was brisk retrograde flow from the internal carotid artery. Angiography continued to show absence of filling of the aneurysm with filling the distal arteries despite
slight increased narrowing 3 mm. distal to the aneurysm.

During the procedure it became necessary to initiate artificial respiration. She survived 10 more hours with artificial support of her respiration and blood pressure.

At autopsy there was an adherent clot in the neck of the aneurysm but the dome of the aneurysm was not clotted. There was no obstruction to flow through the parent circulation.

Comment. Apparent decrease in contrast visualization of the intracranial arteries distal to the aneurysm while the embolus was in place and immediately after its removal was in part secondary to dilution of the medium by saline in the external glass catheterization chamber. Furthermore, it is likely that the contrast medium (50 per cent Hypaque) played a role in causing the spasm distal to the aneurysm. About 90 cc. were used over a 2-hour interval.

Discussion

These experimental and clinical observations suggest that intraluminal manipulation of the intracranial arteries about the circle of Willis is possible technically and tolerated by these arteries when the forces involved are approximately the same as the systolic blood pressure. Also, withdrawal of catheters from arteries immediately adjacent to the circle of Willis is not difficult when aided by momentary reversal of flow in the internal carotid artery. However, passage of a catheter or embolus distal to the middle cerebral trunk may be hazardous because the traction necessary to retrieve it can distort the proximal arteries, resulting in spasm.

Catheterization as well as embolization of the intracranial arteries may have therapeutic usefulness particularly in the treatment of aneurysms and arteriovenous malformations. A variety of tips for the catheter is possible, and a single observation, reported here, suggests that in certain locations an embolus held stationary at the intraluminal orifice of an aneurysm will be forced by the hemodynamics thereby produced into the orifice and remain impacted there.

Summary

A technical method for catheterization of the intracranial arteries is described with observations upon the reactions of the intracranial arteries in 2 patients with arteriovenous malformations, and 1 patient with a bleeding saccular aneurysm.

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References


