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, these increased intracranial pressure with subarachnoid hemorrhage.

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. 8 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 herewith 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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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 extinction with simultaneous stimulation over the left arm and leg. The left arm and leg were about 2 to 3° F. colder than the right.