LOCAL SPASM IN CEREBRAL ARTERIES*

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This work began as an effort to find a local agent which could be used at
the time of surgery to prevent or relieve local cerebral arterial spasm
consequent to irritative vascular manipulation. The need for such an
agent was suggested by strong clinical evidence that certain postoperative
hemipareses were caused by spasm of capsular arteries in response to
mechanical irritation (Penfield and Lende, to be published).

Although there has been much controversy concerning the role of vaso-
spasm in the production of neurological deficits, there has been clear evi-
dence that local spasm is a mode of cerebral arterial response since 1925
when Florey,3 working in Sherrington’s laboratory, induced local spasm in
cerebral arteries of the cat by local mechanical and electrical stimulation.
Spasm similarly induced was photographed in 1931 by Riser et al.10

A variety of direct observations have since been made of such local vaso-
spasm. Echlin2 in 1942 photographed induced constrictions. He was able to
show on injection of vital dye into the general circulation that these spasms
could produce ischemia as evidenced by the restriction of dye peripheral to
the focal constrictions. He further demonstrated that spasm appeared to be
independent of autonomic supply.

Penfield8 in 1937 illustrated with an artist’s drawings local spasm which
he had observed in a pial artery following an epileptic seizure of a patient
undergoing surgery.

The introduction of solid marble-dust emboli into the carotid arteries of
dogs was followed by local arterial constrictions which were photographed
by Villaret and Cachera11 in 1939.

In studies of arterial occlusion in monkeys Harvey and Rasmussen6
noted marked constriction in the proximal middle cerebral artery upon
manipulation which preceded clipping and cutting of the artery.

Recently, transient local arterial spasm has been photographed in rats
through skull windows in a critical work on hypertensive encephalopathy
by Byrom.1

Gillingham5 has observed cerebral arterial constriction in patients dur-
ing the course of surgery for intracranial aneurism, and he has used locally
applied papaverine in an attempt to allay the spasm.

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These substantial reports of local cerebral arterial spasm would suggest that such a mechanism might well operate in the abnormal situation as a significant factor in production of neurological deficit. This present study is concerned with the nature of such spasm, particularly as influenced by topical application of drugs.

MATERIALS AND METHODS

Thirty-four cats, 8 dogs, 3 monkeys (Macaca mulatta) and 1 guinea pig were used. Craniotomies were performed under Nembutal anesthesia. Stimulation by mechanical means was usually done by insertion of a nerve hook about the artery with subsequent traction on the vessel, or by rubbing and displacing the vessel with a firmly wadded piece of cotton. The most practical method of stimulation was found to be electrical. Bipolar electrodes about 2-3 mm. apart were applied to the wall of the vessel, usually for 10 seconds. A 60-cycle AC stimulator was used and generally a 9-volt stimulus. This value was used as a standard maximal stimulation.

Photography was by an Exacta camera with extension tubes and a 24-mm. lens. An electronic flash was the source of light. Focusing was done by a rack and pinion with an arm for holding the camera. This entire device was mounted on the animal head holder. Photographs were usually made with Kodak plus X black and white film. In several experiments Kodachrome color film was used. Chronic experiments were done on 2 cats to test drugs for local or systemic toxicity, using one hemisphere as a control.

Exposure of the basilar artery in the dog was by approach through the palate and pharynx with the mouth widely opened. Sympathectomies were generally performed by midcervical section of the sympathetic trunk. The chronic sympathectomy was prepared for another experiment by resection of the superior and inferior cervical sympathetic ganglia.

A number of drugs (Table 1) were evaluated for their action in prevention or relief of cerebral arterial spasm. Comparison of drug actions in these respects was done by a method of local application which confined the agent to the desired vessels. It was found that saturation of a small piece of compressed cotton ("cotton-oid") would restrict drug action to the limits of the cotton without spreading to adjacent areas as it did with application of drops. Drugs were applied standardly for a 5-minute period. As a control procedure an artery of comparable size was stimulated at about the same time as the treated vessel and reactions were compared at like intervals. Photographs were usually taken before and after application of the drug, and following a standard stimulation at 10 sec., 40 sec., 2 min. and 5 min.

RESULTS

Local constriction of cerebral arteries was observed to follow quite reliably upon local irritative stimulation of the vessel either by electrical current or by mechanical means. Although mechanical irritation is a stimulus more like that incident to human surgery, it was found that electrical stimulation was more easily handled, gave a more constant stimulus value for use in comparing effects of the drugs, and generally gave a more persistent constriction. Also, manipulation and traction commonly resulted in slight bleeding sufficient to obscure photographic detail.