HYPOTHERMIA AND CEREBRAL VASCULAR LESIONS

I. EXPERIMENTAL INTERRUPTION OF THE MIDDLE CEREBRAL ARTERY DURING HYPOTHERMIA*

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Intracranial surgery in the treatment of cerebral vascular anomalies has been sharply limited by the numerous hazards attendant such procedures. Not only is the task of achieving hemostasis frequently difficult, but it is often made more formidable by the risk of producing a neurological deficit through the interruption of a major vascular channel. A technique that could minimize or obviate this risk would make it possible to perform definitive and curative surgery for many lesions that now are commonly considered inoperable. This communication presents an experimental approach to this problem.

It has been well established that permanent occlusion of the middle cerebral artery in the normothermic dog produces an area of infarction of significant magnitude.6,8,11,14 This is proportional to the degree and to the rapidity of the occlusion.9 It was therefore postulated that a marked reduction of cerebral metabolism at the time of occlusion would provide more advantageous conditions for the establishment of collateral circulation, thereby resulting in the modification or prevention of infarction. The following experiments suggest that such conditions may be attained with the use of hypothermia.

METHODS

Thirty-seven mongrel dogs weighing between 10.0 and 23.0 kg. and unselected as to age and sex were used in this investigation.

Twenty-four hours before each experiment, the dogs were given long-acting Benzathine penicillin G, 600,000 units, intramuscularly, and diphenylhydantoin sodium, 0.4 gm., orally. The diphenylhydantoin sodium was administered daily through the 5th postoperative day in order to suppress convulsions.

Anesthesia was achieved with intravenous pentobarbital sodium, 30 mg./kg. Body hair was removed with clippers, and the dogs were intubated with a cuffed No. 38 Fr. endotracheal catheter. The catheter was attached to an automatic posi-

* The opinions or assertions contained herein are the private ones of the writer and are not to be construed as official or reflecting the views of the Navy Department or the naval service at large.

The pilot study preliminary to this investigation was reported at the Annual Meeting of the Federation of American Societies for Experimental Biology, April 11–15, 1955.14 The author was then a member of the Department of Neurological Surgery, Columbia University College of Physicians and Surgeons and the Presbyterian Hospital, New York City.
tive-negative pressure closed-system respirator† which delivered 100 per cent oxygen at a rate of 24 respirations per min. The positive pressure was adjusted between 7 and 11 mm. Hg and the negative pressure between 1 and 4 mm. Hg in order to maintain a tidal exchange of 200–400 ml. A thermistor was inserted 300 mm. into the esophagus for the recording of body temperature. The animals were immersed to the shoulders in ice water until the esophageal temperature fell to 25°C. They were then transferred from the ice bath to the operating table. Body temperature decreased an additional 1–3°C, while the animals were being prepared for surgery.

With the dog in the right lateral recumbent position, a left subtemporal craniectomy was performed using aseptic technique. The dura mater was incised and the pyriform lobe was gently retracted to expose the middle cerebral artery. The artery was freed from its meningeal investment for the first few millimeters of its course, great care being exercised not to disrupt any important adjacent vascular structure. The artery was then transected between silver clips at its origin. Arrow No. 1 in Fig. 1 indicates the point of transection. The dura mater was approximated over strips of absorbable gelatin sponge and the incision was closed in layers utilizing stainless steel screening to cover the craniectomy defect. Two variations of this procedure were also utilized. One consisted of a “crotch isolation”; i.e. division of the middle cerebral artery combined with occlusion of the ipsilateral internal carotid, posterior communicating, and anterior cerebral arteries. The second was a segmental resection of the middle cerebral artery from its origin to the first major cortical bifurcation. This included coagulation and section of each central (striatal) branch and the anterior choroidal artery which, in the dog, arises from this segment of the middle cerebral artery. The section of artery removed corresponds to that indicated between arrows No. 1 and 2 in Fig. 1.

At the termination of the operative procedure, each animal was rewarmed in a water bath in which the temperature was maintained 10°C. higher than that of the dog, until a body temperature of 35°C. was reached. Rewarming was then discontinued and the animal was allowed to spontaneously regain the normothermic state. All animals were observed for 18–22 days. They were then reanesthetized and sacrificed by formalin perfusion. The brains were removed and studied grossly and microscopically.

Occlusion of the middle cerebral artery in the normothermic dog produces a triad of clinically detectable manifestations. These are: (1) ipsilateral forced circling movements, (2) a contralateral hemiparesis, and (3) a contralateral temporal homonymous visual field defect. A visual field defect was said to be present when no reaction was obtained in response to the examiner’s finger movements or when the animal obviously bumped into objects placed in the suspected field of vision. The animals were examined daily and the presence or absence of any or all of the above signs was recorded. No attempt was made to evaluate the clinical signs during the first 48 hours postoperatively, since it was difficult to differentiate the transient signs caused by operative handling from those resulting from permanent brain damage. The subsequent course was divided into 5 periods, as follows:

† Designed by Dr. D. A. Holaday and constructed by the Metal Model Shop of the Naval Medical Research Institute with parts supplied by the Ohio Chemical & Surgical Equipment Company.