HYPOTHERMIA IN THE TREATMENT OF CEREBRAL TUMOURS*

C. B. SEDZIMIR, M.D., AND J. W. DUNDEE, M.D.

Regional Neurosurgical Centre, Walton Hospital, and the University of Liverpool, Liverpool, England

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Interest in the use of hypothermia at the Regional Neurosurgical Centre, at Liverpool, arose from the experience gained by our colleagues in anaesthesiology in general and in thoracic surgery. Our first case, in October 1953, was one of removal of an olfactory groove meningioma in a debilitated elderly patient in whom two previous attempts at surgery had to be abandoned because of bleeding. Despite the transfusion of 4 litres of blood, only about one-quarter of the tumour could be removed at already the second attempt during an 11-hour operation at normal temperature. With a moderate degree of hypothermia (30°C.) combined with trimetaphan (Arfonad)-induced hypotension, total removal of the tumour was completed in 4 hours without the necessity of blood replacement. Encouraged by this success, during the next months operations were performed on patients with aneurysms, using the hypothermia-hypotension combination, and hypothermia was induced as a therapeutic measure in a child with severe primary brain-stem injury and in other conditions.21,22 We have now employed hypothermia in patients with practically every type of tumour, both above and below the tentorium, when complete removal seemed possible.

RATIONALE

In a preliminary report6 in April 1954, we pointed out that the aim was not simply to use hypothermia, but to combine it with controlled hypotension (produced by ganglion-blocking drugs) when required. The title of this communication, "Safer Hypotension," illustrates our views on one of the indications for hypothermia in neurosurgery. It is not necessary to elaborate on the benefits of hypotension to the neurosurgeon, especially during removal of vascular tumours or surgery of aneurysms. It has been shown that the decrease in cerebrovascular resistance during chemically induced hypotension permits adequate cerebral oxygenation at low blood pressures in normal subjects.5,16,23 There is evidence, however, of interference with cerebral circulation in some subjects11 and cerebral damage has been demonstrated by the flicker-fusion test17 and by the electroencephalogram after controlled hypotension.24 Any already existing interference with cerebral blood flow, such as is likely to be present in patients with cerebral tumours or intracranial haemorrhage, will accentuate the effects of hypotension, and

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cerebral hypoxia is likely to result. By virtue of its effect in reducing consumption of cerebral oxygen, hypothermia will protect the brain from the effects of anoxia.

Aserman has described “retractor anaemia” as a cause of death in 3 neurosurgical patients following induced hypotension. He suggests that, in the presence of intracranial hypertension, pressure by a brain retractor may be transmitted to comparatively distant parts of the brain, depriving them of their blood supply and causing irreversible and possibly widespread damage. These effects should be avoided by the use of hypothermia.

It was hoped that hypothermia alone would induce a sufficient degree of hypotension. This has not materialised as shown by the necessity of use of trimetaphan in almost half the patients in our first 50 cases. This will be discussed fully later.

Rosomoff and his colleagues have shown that, in addition to reducing cerebral metabolism, hypothermia decreases intracranial pressure. Cerebrospinal fluid pressure and venous pressure vary proportionately with the temperature. Hypothermia reduces brain volume by 4.1 per cent at 25°C.; and its combined effects increase the intracranial space not occupied by brain by 31.8 per cent. There is no evidence to suggest that ganglioplegic drugs produce any decrease in the volume of the brain whereas many writers have testified to the beneficial effects of hypothermia in this respect in clinical cases. Lundberg et al. have suggested that hypothermia may be able to diminish or counteract swelling of the brain by diminishing the risk of general hypoxia and decreasing the cerebral blood pressure and blood flow.

The major advantage of hypothermia to the neurological surgeon is a prolongation of the permissible time of total occlusion of the afferent circulation of the brain. The main value of this is in vascular surgery, but occasionally it may be necessary in removal of tumours. With the technique to be described, complete occlusion of the middle cerebral artery for periods up to 15 minutes has been carried out in this Centre, without producing apparent cerebral damage.

TECHNIQUE

This has been described previously in detail, and will only be mentioned briefly in this publication.

Preoperative medication is with chlorpromazine, with or without meperidine and/or Phenergan. Light general anaesthesia is induced with Thiopental, the larynx is sprayed thoroughly with 4 per cent lidocaine and oral intubation is carried out with a flexometallic tube after succinylocholine. The maintenance of anaesthesia is with a large flow of nitrous oxide-oxygen (with a T-piece open circuit) and ether is used for the first 10–20 minutes to aid the peripheral vasodilatation.

Surface cooling is commenced, once the patient is asleep, by application of ice bags all over the body and laying the patient on a large ice pack. The position of the ice bags is changed frequently and sometimes the patient is tipped in the head-down position at regular intervals to promote redistribution of the cooled blood. The ice