THERAPEUTIC HYPOTHERMIA IN CASES OF HEAD INJURY*

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The combination of a safe hypothermic technique and general anaesthesia during neurological operations is now an established procedure and its advantages have already been discussed. Following its evolution, our attention was turned to the possible use of hypothermia as a therapeutic measure in cases of severe head injury and of spontaneous intracranial haemorrhage. This communication discusses and evaluates its use in a series of severe head injuries, from December 1953 to October 1957.

It is not necessary to enlarge upon the clinical manifestations and poor prognosis of a primary brain-stem injury, nor upon the pathology and mechanism of the shifts of the brain which may lead to a secondary brain-stem lesion. Once extradural and acute subdural haematoma are excluded, or dealt with surgically, we are left with the problems associated with diffuse neuronal paralysis or destruction, contusion of the brain, laceration and cerebral haemorrhage, giving rise to progressive swelling of the brain. This leads to an ever increasing cerebral circulatory embarrassment. The tendency to pyrexia is pronounced and oxygen requirements of the tissues rise steeply.

Hypothermia reduces the cerebral metabolism and oxygen consumption, thus protecting the endangered neural elements. Survival and function at a lower metabolic level may still continue within areas rendered anoxic or ischemic by oedema or by vascular insufficiency. Time is gained for the oedema to subside or the circulation to become re-established, and the neural tissues can survive to resume their activity at more customary metabolic levels. Furthermore, as the intracranial pressure is considerably lowered under hypothermia, this may help to avert the serious consequences of foraminal herniations. Rosomoff in an experimental study stated:

"The use of hypothermia aborts the development of brain swelling following experimental brain trauma at 25°C. The pathology of the lesions is also affected in that there is less widespread destruction of neural elements, less hemorrhagic diathesis, and diminished host reaction to injury during hypothermia. Animals kept hypothermic 18 hours and then rewarmed survive 5 times longer than their normothermic control equivalents."

Sponging with iced water in cases of hyperthermia is a valuable and long-

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recognised measure, but frequently the temperature can be reduced only insignificantly or is entirely refractory to the sponging, because of cutaneous vasoconstriction and shivering in response to the cold. If it does fall, it rarely approaches normal, let alone below. For persistent brain oedema, dehydration therapy has only a limited application. Major neurosurgical procedures of splitting the tentorium or resection of the herniated uncus are rarely practicable.

The administration of chlorpromazine and an analgesic increases the efficacy of exposure to cold air and sponging with iced water by inducing cutaneous vasodilatation and reducing the shivering. This we call the "exposure therapy," and using this method only, the temperature may drop from 40–41°C. (105°F.) to the region of 34°C. (95°F.). When this simple exposure therapy fails, a more effective technique of surface cooling must be employed. The patient is covered with ice-bags, more drugs are given if shivering occurs, and the temperature is gradually reduced to 34°C. (95°F.) before the ice is removed. It should not be allowed to drop below 30°C. (90°F.), which we consider to be the limit of safety in cases of head injuries. The hypothermia should be maintained, if at all possible, until there is no clinical deterioration, when the temperature is tentatively allowed to rise.

The improvement gained during the period of hypothermia may be maintained subsequently, even when the temperature has risen to about 38°C. (100°F.). The exposure therapy is then continued until the danger to life has passed, usually coinciding with recovery of consciousness. Should deterioration be evident at any time, cooling with ice must be re-employed after further surgical exploration, if the latter is judged to be necessary.

Lazorthes et al.\(^3\) gave a warning that hypothermia may mask the usual signs of massive intracranial haemorrhage. This observation is important, particularly during the indiscriminate use of hypothermia by clinicians unfamiliar with this method or with the supervision of cases of cranial trauma. In these cases there must be very strict vigilance, and with the moderate degree of hypothermia here advocated, \textit{the experienced eye will detect signs of deterioration without difficulty}. If deterioration ensues, appropriate surgery should be performed immediately, as illustrated by a number of our cases.

**MATERIAL AND RESULTS**

In the following group of 30 patients to whom this treatment was given, the only selection lay in the severity of the condition. Only the most serious head injuries qualified and no one was too ill if alive on admission to hospital. The patients have been divided into two groups:

1. Those, apparently moribund on admission, with signs of primary brain-stem damage.
2. Those rapidly deteriorating despite surgical and conservative treatment.

There were 10 patients in the first group: 8 of these were children aged between 3 and 10 years; 2 were male adults aged 18 and 20.