HYPOTHERMIA IN THE TREATMENT OF CRANIOCEREBRAL TRAUMATISM*

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The treatment of craniocerebral injuries has been improved considerably during the past twenty years thanks to the development of new techniques of examination, a better understanding of the possibilities and indications for surgical intervention and the correction of neurovegetative derangements and of respiratory and metabolic disturbances. However, there are still unsolved problems in this field and perhaps therapeutic hypothermia may provide the solution for some of these. Since the end of 1951 we have treated patients with craniocerebral injury with hypothermia. This form of treatment was created in 1941 in the United States by Fay and reintroduced in France by the authors in a different form, inspired by Laborit and Huguenard. It has often been criticised but has been adopted by many.

I. THE PROBLEMS THAT ARE RAISED

1. Anatomicopathological Data. The traumatism produces lesions about which reactions develop. The different phases of this process follow each other and are interwoven. The reparatory phase, not always achieved, follows the others.

The lesions are seldom unique and limited, but on the contrary are usually multiple and dispersed through different levels.

The perilesional reactions, often extensive and durable, play a part in the evolution of the disease which is often important and lasting. They are by definition reversible, if treated early. If they are prolonged they result in irreversible secondary lesions (epilesions) which may cause death. (a) The reactions at the cellular level are associated with a hypercatabolism of the proteins and hydrocarbons, a hyperpermeability of the cellular membranes with a loss of potassium from the cells and a retention of hydrosodium. (b) The vasomotor reactions may produce a local asphyxia followed by vasodilatation and vasoplegia, hyperpermeability of the capillaries and diapedesis. Vasomotor disturbances tend to extend like a spot of oil and often end in generalized vasomotor disturbances with serious reactions in the brain stem. (c) The general cerebral reactions develop from the interstitial edema produced by the vasodilatation, with extravasation of plasma and variable degrees of cellular infiltration.

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The intracranial hypertension is at first compensated for by the squeezing out of cerebrospinal fluid and of intracranial venous blood. Soon a vicious circle develops when herniations of the temporal or occipital regions appear. These compress the brain stem, block the fluid pathways and produce venous stasis, thus aggravating the edema. Compression of the cerebral arteries causes ischemia and cerebral anoxia is the terminus.

Irritation of the brain stem is the common denominator of all of the preceding phenomena. The brain stem may suffer directly from the initial trauma and again from compression produced by the intracranial hypertension and the herniation. It in turn affects the entire organism unfavorably.

2. Clinical and Humoral Data. The vegetative disturbances evolve in two phases: A vagal phase, often unperceived because it is short, follows the traumatism immediately. The patient is pale, hypotensive, bradycardiac, bradypneic and easily hypothermic. It may be associated with the discharge of acetylcholine described by Bornstein.\(^1\) A hypersympathicotonic phase follows it; the patient becomes febrile, hypermetabolic, hyperpneic, tachycardiac, etc. The reactional nature of the clinical picture is evident. The respiratory and circulatory disturbances, the visceral dysfunctions, renal in particular, even the acute pulmonary edema and antemortem hemorrhages in the digestive tract are expressions of the irritation of vegetative centers. Protein hypercatabolism is evidenced by an increase in urea in the blood and urine. The escape of cellular potassium results in a negative potassium balance, while the entry of sodium into the cells is revealed by a hypernatremia without hypernatruria.

The systemic phenomena resulting from the cerebral troubles contribute in turn to their aggravation. The hypoxia and hypercapnia increase the cerebral vasodilatation together with the arterial hypertension. Coma is a superadded cause of metabolic disturbances. The elevation of temperature increases central sympathetic excitability.\(^5\)

As the lesions from which these patients die are often secondary to the reactions, therapy must be antireactional and prophylactic in preventing secondary lesions. Toward this end our treatment should be so directed as (1) to diminish cellular metabolism and if possible to restore potassium to the cells; (2) to inhibit vegetative reactional and endocrine systems; (3) to restrict intracranial hypertension and (4) to increase cerebral resistance to anoxia. In this sense hypothermia seems to be a logical form of treatment from many points of view.

II. HOW HYPOTHERMIA RESOLVES THESE PROBLEMS

Two series of observations, one from North America and one from France, have led to this form of treatment.

(1) Some investigators have induced a deep hypothermia (30°C) by means of intense refrigeration, rendered tolerable by narcosis. Hypothermia lowers general metabolism (to 50 per cent at 30°C to 30 per cent at 25°C); lowers cerebral metabolism and increases the resistance of the brain to hypoxia. This has been demonstrated by Lougheed and Kahn,\(^8\) Botterell...