Efficacy of Barbiturates in the Treatment of Resistant Intracranial Hypertension in Severely Head-Injured Children

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Dear Sir,

This is an interesting paper [Pediatr Neurosci 1989;15:13–17] despite the lack of any formal study being present. In their retrospective review of head-injured children the authors found a group of children who had an intracranial pressure (ICP) over 20 Torr despite therapy and who then received pentobarbital. Six children died; in 14 the ICP was well controlled, but we are given no indication of their outcome, and 7 children continued to have episodes of intracranial hypertension despite the barbiturates. This latter group of 7 children is focus of the paper. The authors were impressed that 5 of the 7 made a good recovery or were moderately disabled, and only 2 were vegetative survivors. Prior to the mid-1970s it was not fashionable to treat ICP, unless it was over 40 Torr. The authors have rediscovered that despite elevated ICP recovery can occur, even when there are transient periods of zero perfusion. This is important, since it may be too easy to abandon therapy because the cerebral perfusion pressure (CPP) is below some arbitrary number, e.g., 50 Torr. There are no studies in children to suggest that a CPP of less than 50 Torr is even associated with decreased cerebral blood flow. Even in adult animals and humans the evidence for increased anaerobic glycolysis does not occur until a CPP of 25–30 Torr. An elevated ICP is only one element in the complex pathophysiology of head trauma, and its significance should not be overestimated. It is a very important parameter because as yet it is one of the few we can treat, but the value of controlling it and the effects of not controlling it within a given, still arbitrary, range are dependent on the brain pathology, the presence or absence of pressure autoregulation, the degree of ischemic damage, seizure activity, etc. It should come as no suprise that survival is not simply equated with ICP control.

I do not agree with the authors’ conclusions that the survival in these children was due to some mysterious effect of the barbiturates. They state that there was no correlation between the time barbiturates were required, the blood levels achieved, or in fact the effect on the ICP. While it may be that the barbiturates produced some benefit, this paper can not be used as support for that thesis. Did all the 14 children in whom the ICP was controlled have a good outcome? In the last paragraph the authors state that the barbiturates interfere with the declaration of brain death. This is not so. They do interfere with the use of the clinical examination and the EEG but not with the evidence of prolonged no flow due to the ICP = SAP or to the value of cerebral blood flow studies.