

Chapter 17. Critical pathway for the treatment of established intracranial hypertension in pediatric traumatic brain injury

A critical pathway, developed by consensus, is presented in Figures 1 and 2. We developed a treatment algorithm for established intracranial hypertension, wherein the order of steps is determined by the risk/benefit ratio of individual treatment maneuvers. The considerations involved are outlined in the chapter specific to each step.

As discussed in the section on intracranial pressure (ICP) treatment threshold, the absolute value defining unacceptable intracranial hypertension is unclear. Although a general threshold of 20 mm Hg has been presented, there will be situations where such pressures are too high as well as instances where higher ICP values are acceptable. These considerations are relevant to the decision to pursue any step in the escalated treatment of ICP.

This critical pathway is a committee consensus and, therefore, must be viewed as class III (“expert opinion”) evidence. As such, it should be interpreted as a framework that may be useful in guiding an approach to treating intracranial hypertension. It can and should be modified in an individual case by any circumstances unique to the patient as well as by the response of the ICP to individual treatment steps.

This algorithm applies to severe traumatic brain injury. The decision to monitor ICP and apply this algorithm to children or infants with lesser degrees of brain injury is left to the physician.

Critical Pathway

The most fundamental maneuver in managing the pediatric patient with severe traumatic brain injury, outside of the surgical evacuation of intracranial

mass lesions, is the insertion of an ICP monitor. Once this has been accomplished, treatment can be directed at controlling ICP and cerebral perfusion pressure (CPP). A number of general maneuvers may be applied to this group of patients early during their treatment, including control of fever, avoidance of jugular venous outflow obstruction, and maintenance of adequate arterial oxygenation. The initial Paco_2 should be maintained at the low end of eucapnia (35 mm Hg). In addition, regardless of the presence or absence of intracranial hypertension, CPP should be maintained. The exact value to be targeted should be predicated on age and may be modified by advanced cerebral physiologic monitoring.

When intracranial hypertension occurs, the adequacy of sedation and analgesia should be checked and augmented as needed. In euvoletic patients, the head of the bed may be elevated to approximately 30° and the response of ICP and CPP monitored for efficacy. The addition of neuromuscular blockade to the treatment regimen also may be considered at this point.

If ICP remains elevated despite adequate sedation, analgesia, and elevation of the head of the bed (with or without neuromuscular blockade), the initial ICP-specific therapeutic intervention should be CSF drainage when ventricular access is available. Should an ICP monitor lacking the capability of CSF drainage have been placed, consideration should be given to obtaining ventricular access.

If CSF drainage is ineffective in controlling ICP or is not available, hyperosmolar therapy should be considered. There is insufficient evidence to support prioritizing the use of mannitol vs. hypertonic saline as a first choice. The patient's volume status should be closely

observed during mannitol administration, and the upper limits of 320 mOsm/L for mannitol and 360 mOsm/L for hypertonic saline should be observed. If hyperosmolar therapy proves ineffective, the level of ventilation may be increased to obtain Paco_2 levels of 30–35 mm Hg. Measurement of cerebral blood flow, jugular venous saturation, or tissue oxygen tension should be considered when hyperventilation is increased.

At all times during the treatment of intracranial hypertension, the possibility that a surgical mass or an unexpected intracranial lesion may have developed should be considered. Therefore, under conditions of intractability or loss of ICP control or when second-tier therapy is being contemplated, clinicians should consider repeating a computed tomography scan.

For intracranial hypertension refractory to the previously described techniques, second-tier therapies should be considered when the physician believes that the patient may benefit if ICP control can be accomplished. Second-tier therapy includes ICP-lowering treatment modalities that have been demonstrated to improve outcome at some level of evidence but that have not been subjected to trials comparing them to alternate second-tier treatments to establish relative risk/benefit ratios. Details of the literature addressing the various second-tier therapies presented here are found in other chapters. Some considerations relevant to differentiating between these differing second-tier maneuvers are contained in the algorithm. The precise indications for selecting and applying second-tier therapies in an individual patient are left to the discretion of the managing physician.

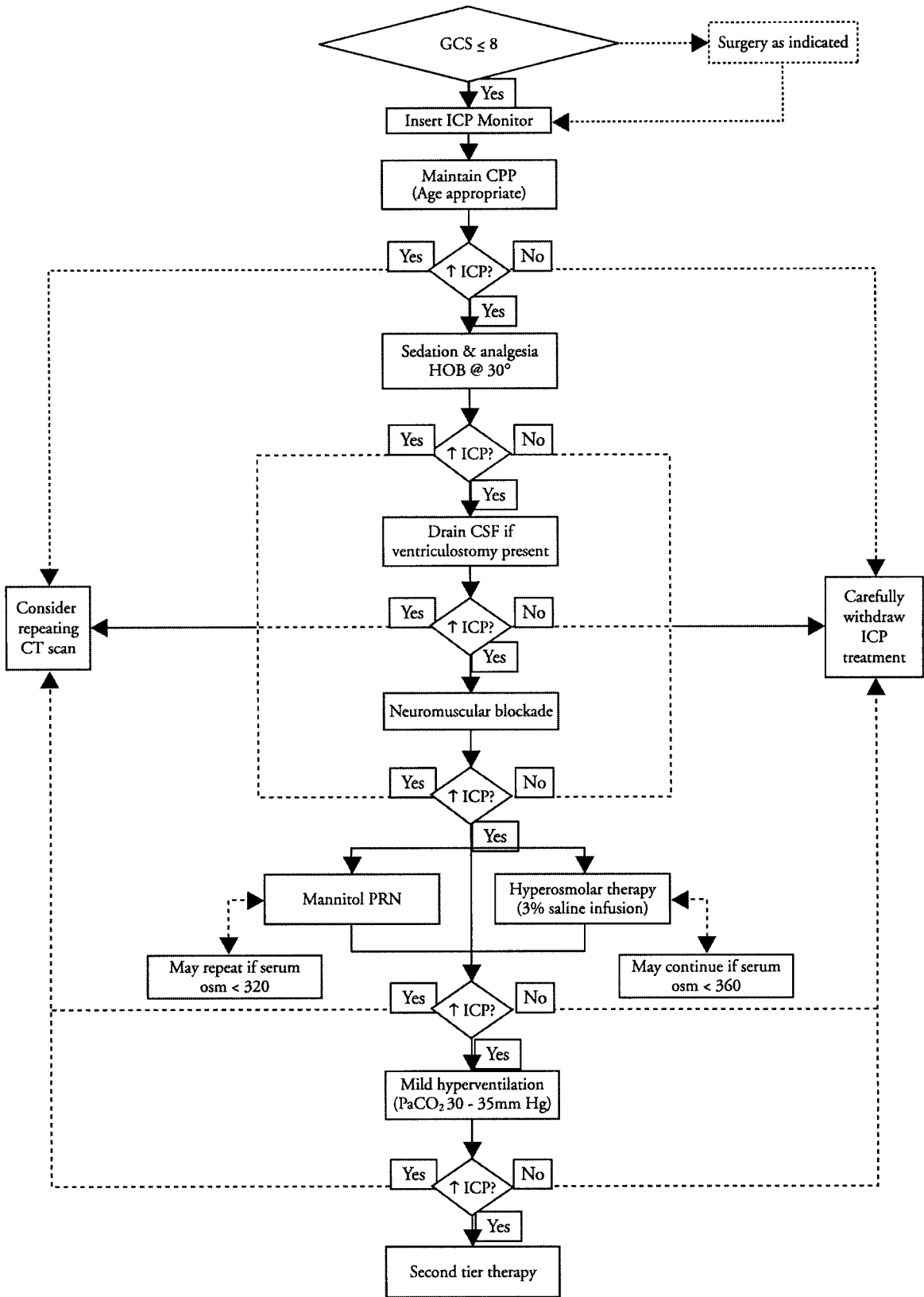


Figure 1. First tier. *GCS*, Glasgow Coma Scale; *ICP*, intracranial pressure; *CPP*, cerebral perfusion pressure; *HOB*, head of bed; *CSF*, cerebrospinal fluid; *CT*, computed tomography; *PRN*, as needed.

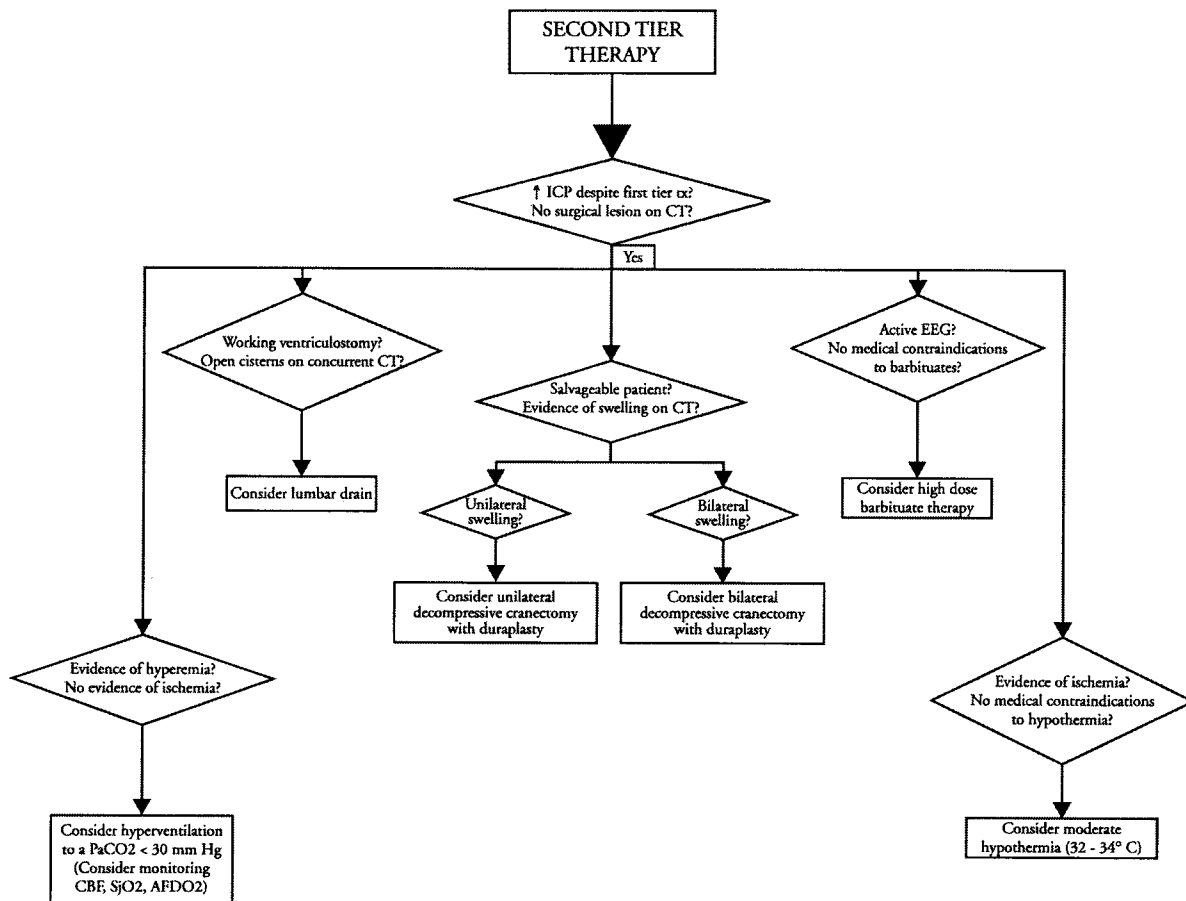


Figure 2. Second tier. *ICP*, intracranial pressure; *CT*, computed tomography; *EEG*, electroencephalogram; *CBF*, cerebral blood flow; *SjO₂*, jugular venous oxygen saturation; *AJO₂*, arterial-jugular venous difference in oxygen content.