



Therapeutic Momentum: Scenarios in Patients with Neurotrauma

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In clinical practice, the element of the patient-clinician relationship can lead to dynamics where treatment decisions can be clouded with the clinician's personal biases or unrecognized acumen judgment errors. This translates to therapeutic momentum (TM). Therapeutic momentum was a term proposed by Rodrigo et al in 2012¹⁾, they describe as: In situations when doctors do not stop or because of personal clinical decisions they do not interrupt therapeutic strategies without any benefit and contrary to evidence that supports maintaining treatment. In addition to the definition, we propose 2 classes of Therapeutic momentum: When the doctor has the deleterious effects of maintaining a therapy, and when the physician is unaware of the deleterious effects of maintaining a therapy.

The concept of TM is strongly presented in the realm of traumatic brain injury (TBI). The examples of therapeutic momentum in BTI may include but are not limited to: fluid therapy (Hypertonic-Mannitol) without evidence of increased intracranial pressure, anticonvulsants - keeping post-trauma antiepileptics for more than 7 days, gastroprotection (maintaining proton inhibitors without evidence of digestive tract bleeding), neuroimaging (performing control neuroimaging in unstable patients with no obvious clinical indication), and invasive intracranial pressure monitoring (maintaining intracranial pressure monitor when intracranial hypertension has resolved)

We propose an algorithm for TM in circumstances where we consider strategies that are not effective in patients with TBI (Fig. 1). Truly the progression of TBI through the stages of care can lead to TM moments and each step deviation can lead the patient to a path of declined status. The moment of initial management includes optimization of perfusion pressure, airway control, avoid hypotension¹⁾, hypercarbia, correction of coagulopathy, control of temperature, and decision to proceed to surgery.

Decompression after 48 hours if intracranial pressure (ICP) and cerebral perfusion pressure (CPP) can be controlled is the preferred pathway²⁻⁴⁾. Early decompression might be required from epidural hemorrhage, marked ICP not controlled with medical management, or obstructive hydrocephalus⁵⁾. Without trying medical management and going right to surgery might lead to unnecessary systemic

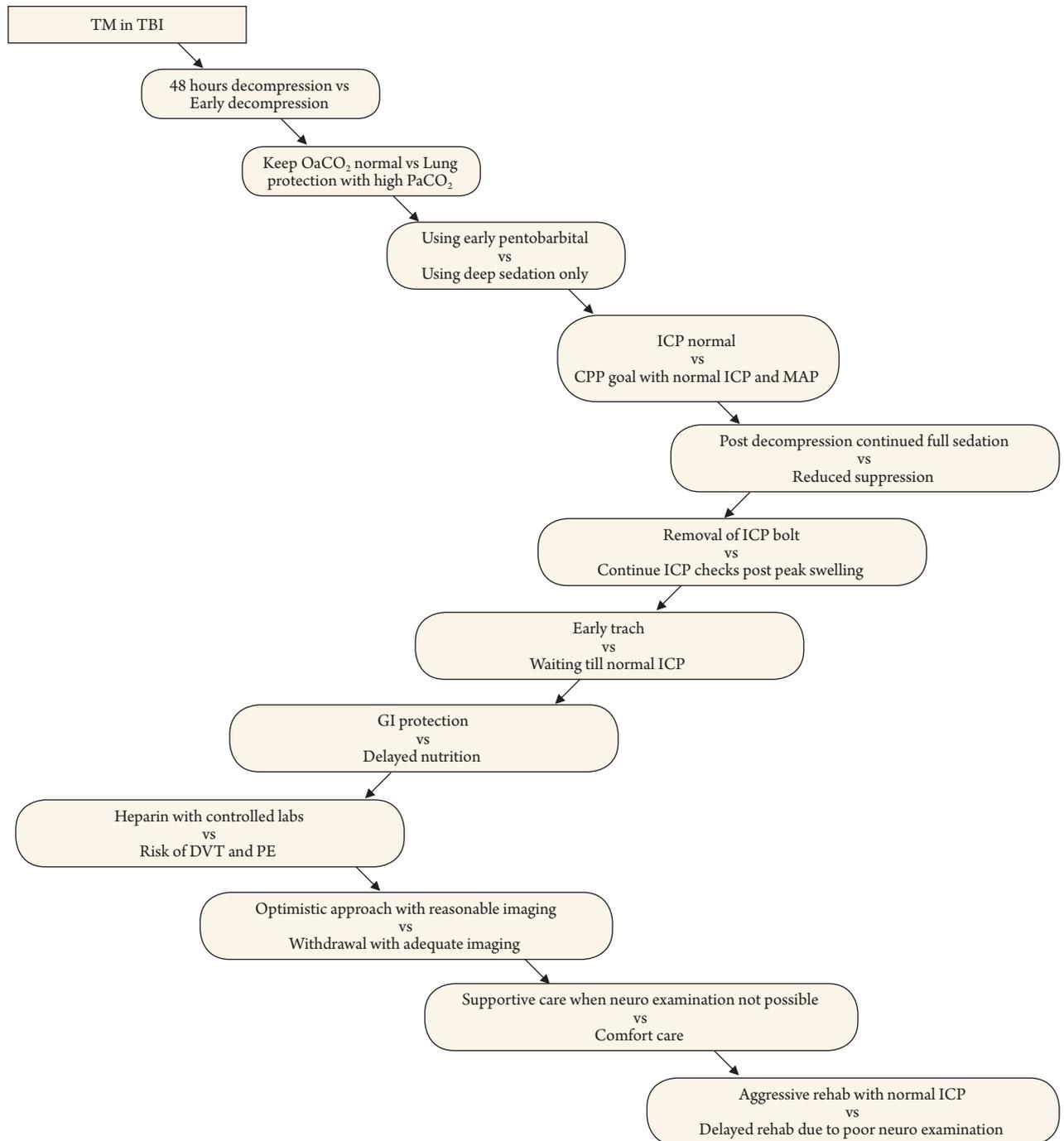


Fig. 1. Algorithm for therapeutic momentum in circumstances where we consider strategies that are not effective in patients with traumatic brain injury.

compromise. Most of these patients also have systemic injuries including pulmonary. Protective lung ventilation is the goal in any intubated patient, although increased PaCO₂ can lead to cerebral edema and the balance can shift towards a negative outcome. TM can lead the clinician to oxygenate more than what is required and hypo or hyperventilate leading to low or high PaCO₂ receptively. At this stage, some patients may benefit from short-term extracor-

poreal circulation if the service is available. Position of the head makes a difference with relative hyperperfusion and decreased venous return. While achieving central venous access or during intubation, keeping the head of the bed flat for a longer period will lead to reducing central venous return, hence worse cerebral edema. The same goes for choosing internal veins for placement of central access.

TBI induced cerebral edema can be controlled with deep sedation and early initiation of the general anesthesia approach may help with reduction in longer sedation needs. Pentobarbital reduces cerebral metabolic rate (CMR) and central temperature. A rather progressive escalation of sedation due to concern for deep sedation effects, it may be prudent to go right to this agent. If the ICP is controlled but the patient has lower mean arterial pressure (MAP), this might cause the number for CPP to be lower. At this stage, following ICP is better than staying the single number of CPP. This can happen with younger patients who do not have high diastolic pressures. Rather than continuing following CPP with a bolt, the bolt can be removed, or if external ventricular drainage is present to just manage the cerebrospinal fluid (CSF) diversion rather than ICP.

There are critical steps during the intensive care management of TBI patients and TM can sidestep some of these to benefit factors that may not help with the progression. These are full suppressive sedation in the early phase after decompressive craniectomy to avoid any detamponade effect of cerebral edema; early tracheostomy if possible before ICP become resistant and high for a prolonged period; active prevention of deep vein thrombosis (DVT) with agents like heparin and not taking a chance of a terrible catastrophe of pulmonary embolism; and initiation of trophic feed to avoid hyperglycemia with the full goal caloric feeding. All elements are simultaneous and concurrent in the management progression.

Due to intense and prolonged resuscitation of TBI patients, TM can lead clinicians to err towards the early track to a cessation of efforts and comfort care. Rehabilitation starts from the early phase and involvement of a full spectrum of services assist in decision making and eventual discharge to a long-term treatment facility for continued acute rehabilitations.

In summary, TM in TBI management is very much part of our

practice. The recognition of this and keep avoid distraction in the TBI course is considered valid and important for the practice of medicine. Early education for the trainees and proper streamlining of the thought process will help keep TM in check. Further research in this field will be required and repeated reminders in certain aspects for senior clinicians.

NOTES

Conflict of interest

No potential conflict of interest relevant to this article was reported.

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