Adrenergic Suppression Modalities in Acute Traumatic Brain Injury

Scott Meyer[1] Fotis Souslian[2] Eric Nussbaum[3] Luis Rafael Moscote-Salazar[4] Tariq Janjua[4,5]

[1] Department of Neurosurgery, Christus Spohn Hospital, Corpus Christi, Texas, United States
[2] Department of Neurosurgery, CHI Health St. Francis, Grand Island, Nebraska, United States
[3] Department of Neurosurgery, National Brain Aneurysm and Tumor Center, Minneapolis, Minnesota, United States
[4] Department of Research, International Consortium of Neurological Research, United States
[5] Department of Critical Care Medicine, Physician Regional Medical Center, Naples, Florida, United States

Address for correspondence Luis Rafael Moscote-Salazar, MD, Department of Research, International Consortium of Neurological Research, United States (e-mail: Rafaelmoscote21@gmail.com).

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The relationship between catecholamine surge and acute traumatic brain injury (TBI) has been shown in the literature.[1–4] The catecholamines released during acute TBI lead to a higher metabolic rate, increased systemic blood pressure blood flow, and hyperemia in the brain. The brain autoregulation is linear due to TBI,[5] this leads to uncontrolled perfusion and hence cerebral edema. Also, this pathophysiological process is directly related to a significant and potentially life-threatening increase in intracranial pressure (ICP). In patients with TBI, multiple pharmacological tools are used to prevent and treat high ICP. These include nonselective beta-blockers for suppression of hypertensive response, beta-blockers to suppress adrenergic surge, hypertonic saline, osmotic diuresis with mannitol, and sedative agents including midazolam, lorazepam, propofol, pentobarbital, and inhaled anesthesia. In the more extreme cases, an induced coma is introduced with pentobarbital or inhaled anesthesia. This is to decrease cerebral metabolic rate and sometimes even to the point of complete loss of brain activity.

The adrenergic stimulation during TBI causes inhibition of thick lymphatic removal of cellular debris and toxins, thus worsening cellular injury and cerebral edema (Hussain). In addition to this adrenergic suppression may then improve the glim flattish drainage and removal of these toxins from the brain, thus reducing injury and cellular damage. This indirectly impacts high ICP (►**Fig. 1**).

Beta-blocker therapy in acute TBI can decrease the physiological effects of the hyperadrenergic state. This effect should be in context with maintained cardiac index and hence continue stable cerebral perfusion. Sedated agents with a central effect like propofol, dexmedetomidine, and benzodiazepines can reduce systemic hypertensive response with reduced adrenergic outflow. Pentobarbital interferes with the impact of plasma catecholamines on physiological factors.[6,7] The increased sympathetic outflow and surge after TBI is abutted which can impact TBI outcome. This adrenergic inhibition has been shown to improve ICP by interfering with the sympathetic surge that occurs after TBI. A single dose of pentobarbital in patients who present with low Glasgow Coma Scale and TBI with evidence of hyperemia and swelling on the initial computed tomography scan seems to break the cycle of the sympathetic surge. This effect of pentobarbital has a multipronged effect by interfering with the sympathetic outflow cascade.

The development of a hyperadrenergic state in patients with TBI is a major pathophysiological process.[8,9] This elevation of blood levels of catecholamines and the action of adrenergic lead to pathophysiological changes including tachycardia, hypertension, tachypnea, hyperemia, peripheral vasoconstriction, rise in lactic acid, hypercarbia, hypoxemia, increased ICP, and agitation. The elevation of catecholamines is associated with a hypermetabolic state, hence reduced outcomes. Proper use of therapeutic modalities including

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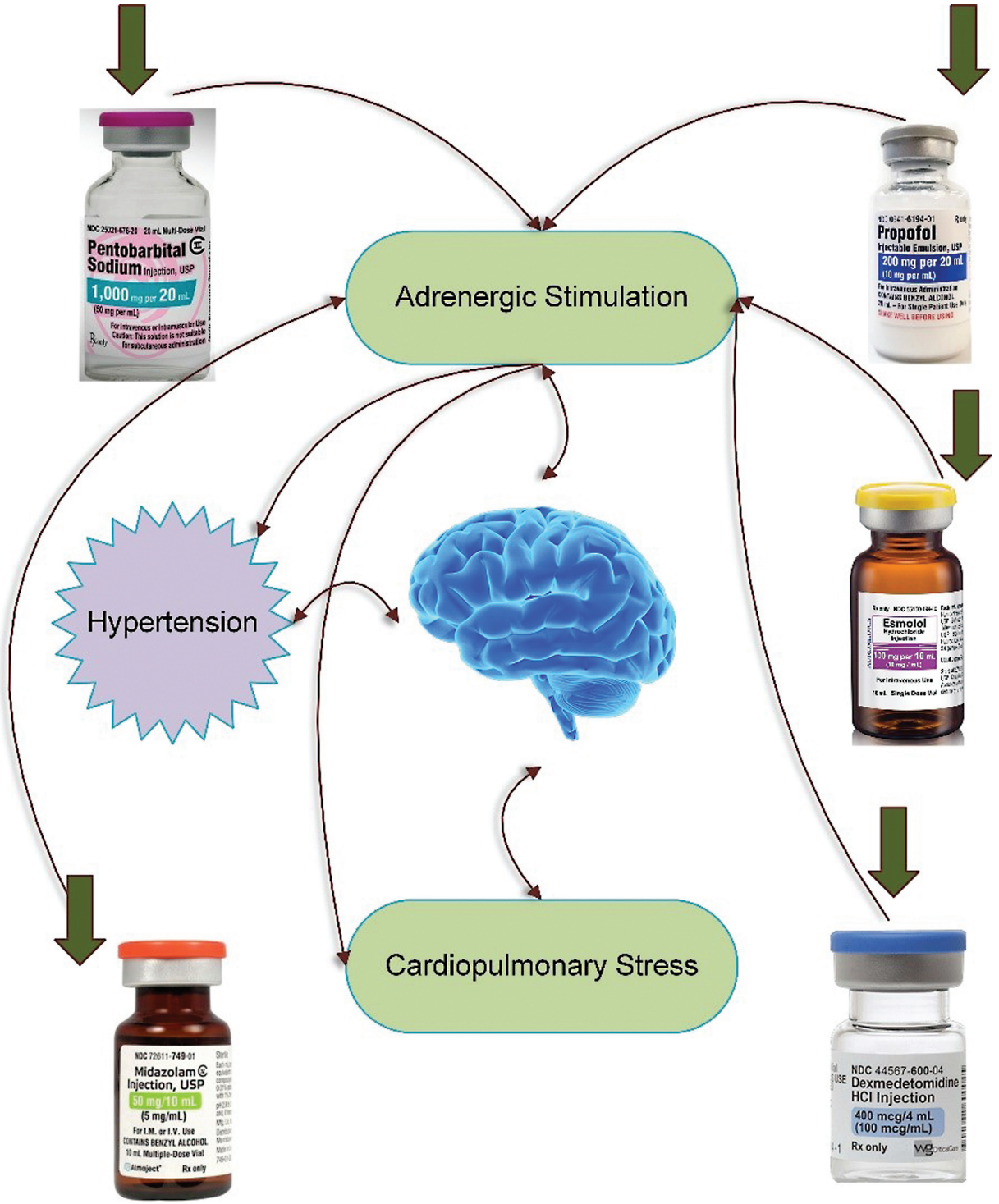

Fig. 1 Effect of adrenergic stimulation, agents used to reduce that impact on acute traumatic brain injury (TBI).

beta-blockers, propofol, dexmedetomidine, and pentobarbital will impact ICP and TBI outcomes. Further studies are required where this impact is confirmed with cardiopulmonary dynamics monitoring.

Conflict of Interest

None declared.

References

1 Florez-Perdomo WA, Laiseca Torres EF, Serrato SA, Janjua T, Joaquim AF, Moscote-Salazar LR. A systematic review and meta-analysis on effect of beta-blockers in severe traumatic brain injury. Neurol Res 2021;43(08):609–615

2 Ding H, Liao L, Zheng X, et al. β-blockers for traumatic brain injury: a systematic review and meta-analysis. J Trauma Acute Care Surg 2021;90(06):1077–1085

3 Khalili H, Ahl R, Paydar S, et al. Beta-blocker therapy in severe traumatic brain injury: a prospective randomized controlled trial. World J Surg 2020;44(06):1844–1853
4 Zagales I, Selvakumar S, Ngatuvai M, et al. Beta-blocker therapy in patients with severe traumatic brain injury: a systematic review and meta-analysis. Am Surg 2023;89(05):2020–2029
5 Figaji AA, Zwane E, Fieggen AG, et al. Pressure autoregulation, intracranial pressure, and brain tissue oxygenation in children with severe traumatic brain injury. J Neurosurg Pediatr 2009;4(05):420–428
6 Baum D, Halter JB, Taborsky GJ Jr, Porte D Jr. Pentobarbital effects on plasma catecholamines: temperature, heart rate, and blood pressure. Am J Physiol 1985;248(1 Pt 1):E95–E100
7 Mazerolles M, Senard JM, Verwaerde P, et al. Effects of pentobarbital and etomidate on plasma catecholamine levels and spectral analysis of blood pressure and heart rate in dogs. Fundam Clin Pharmacol 1996;10(03):298–303
8 Moscote-Salazar LR, Janjua T, Florez-Perdomo WA, Vasque H. Hope or hype: beta-blockers in traumatic brain injury. Indian J Neurotrauma 2021;18:98
9 Elenkov IJ, Wilder RL, Chrousos GP, Vizi ES. The sympathetic nerve–an integrative interface between two supersystems: the brain and the immune system. Pharmacol Rev 2000;52(04): 595–638

