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Blood pressure targets in neurosurgical patients: How high and how low should we go?

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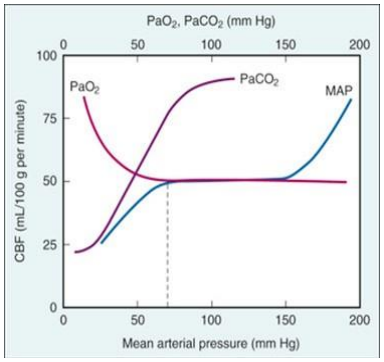
Introduction: The limits of cerebral autoregulation curve are now considered dynamic rather than, as originally described 60 years ago, static. Acceptance of the dynamic nature of cerebral autoregulation, along with new concepts of pathophysiology of intracranial events over the past decade and evidence from new randomized controlled trials (RCTs), have led to a revision of blood pressure (BP) targets for various intracranial events.

Discussion: Pathophysiology and systemic events following an acute intracranial event: Over several decades it has been widely believed that, following an intracranial event, BP increases secondary to the Cushing response, which is essential to maintain cerebral perfusion pressure (CPP). However, as per the current concept, following an intracranial event there is a massive sympathetic discharge with catecholamine surge leading to hypertension and tachycardia.1 The Cushing reflex is a late phenomenon, indicative of severe intracranial injury and associated with hypertension and bradycardia rather than tachycardia. The cytokine release following an acute intracranial event may lead to increased capillary permeability and pulmonary edema.2 The catecholamine surge may lead to ST and T wave changes, tachyarrhythmias, wall motion abnormalities and troponin rise.3 Both cardiac and pulmonary events are markers of the severity of intracranial hypertension.2,3 Since BP rise is primarily due to catecholamine surge, it is prudent to treat systemic hypertension following an acute intracranial event without fearing the risk of hypoperfusion.

Cerebral autoregulation curve: The cerebral vasculature is able to maintain contrast cerebral blood flow with varying fluctuation in the mean arterial pressure (MAP). Originally described by Lassen in 1959, it was thought to be static.4 The lower limit was described as 60mm Hg of MAP and the upper limit in the range of 130- 150 mm Hg. Currently, this range is considered to be dynamic rather than static and likely influenced by hypoxia, hypocarbia, hypercarbia, catecholamine surge and anesthetic agents during general anesthesia. Therefore, the range of MAP of cerebral autoregulation is likely to be in a narrower range rather than the 60-150 mm Hg originally thought. The lower limit has recently been described as 70 mm Hg of MAP, and arterial BP should be measured with a transducer level at the upper part of the cranium.5

Following an intracranial event and depending on the severity of the injury, blood brain integrity is disrupted and cerebral autoregulation is lost, and cerebral blood flow then directly depends on MAP.

Cerebral autoregulation



Cerebral autoregulation. Changes in cerebral blood flow (CBF) caused by independent alterations in PaCO2, PaO2, and mean arterial pressure (MAP). PaO2, Alveolar oxygen partial pressure; PaCO2, partial pressure of carbon dioxide in arterial blood.

Image from Lemkuil BP, Drummond JC, Patel PM. Central nervous system physiology: Cerebrovascular. In Hummings HC, Egan TD, eds. Pharmacology and Physiology for Anesthesia: Foundations and Clinical Application. Philadelphia, PA: Elsevier

BP targets in Traumatic Brain Injury (TBI): The hypotension in TBI is defined as systolic BP less than 110 mm Hg as per the 2016 guidelines of the Brain Trauma Foundation (BTF).6 The prior, 2007 guidelines defined hypotension at 90 mm Hg or less. Based on retrospective data showing increased mortality and adverse outcomes for patients with systolic BP of 90-110mm Hg, the BTF revised their guidelines.6 In 2016, they also revised their cerebral perfusion pressure target to 60-70 mm Hg from the earlier (2007) guideline of 50-70 mm Hg.6 Retrospective analysis showed that patients with severe TBI had worse outcomes when their CPP was between 50 and 60 mm Hg.6

Acute Ischemic Stroke (AIS): Prior to reperfusion therapy (intra-arterial clot extraction, intra-arterial thrombolysis and/or stent placement), systolic BP should be in the range of 140-180 mm Hg as per the 2014 SNACC consensus statement.7 A recent statement from the American Heart Association/ American Stroke Association (AHA/ASA) also recommends systolic BP of less than 185 mm Hg prior to starting intravenous thrombolytic therapy.

Spontaneous intracerebral hemorrhage (ICH): Per the INTEnsive Blood Pressure Reduction in Acute Cerebral Hemorrhage Trial (INTERACT 2) and the Antihypertensive Treatment of Acute Cerebral Hemorrhage (ATTACH 2) multicenter trials, systolic BP can be safely reduced to 120-140 mm Hg without adverse 90-day neurological outcomes.8,9 But, systolic BP less than 120 mm Hg is likely to be associated with acute kidney injury per the secondary outcome measure of the ATTACH 2 trial.9

Spontaneous aneurysmal subarachnoid hemorrhage (aSAH): As with spontaneous ICH, spontaneous aSAH in patients with unsecured intracranial aneurysm presents the dual concerns of hypertension leading to possible rebleeding, and hypotension leading to possible hypoperfusion and cerebral ischemia. For these reasons, systolic BP was historically kept at 180 mm Hg. However, the 2011 consensus statement of the Neurocritical Care Society Conference and subsequent statement of the AHA/ASA recommended a lower level target of 160 mm Hg.10,11 As these twin concerns are similar for both groups pf patients , the results of INTERACT 2 and ATTACH 2 trials of spontaneous non-traumatic ICH can be extrapolated to spontaneous aSAH patients. Thus, the target level prior to securing aneurysm is 120-140 mm Hg.10, 11

Deep Brain stimulation, macroelectrode implantation and microelectrode recording; BP should be reduced to 140 mm Hg systolic since higher BP prior to macroelectrode implantation is associated with intracranial bleeding and adverse outcome. Deep Brain stimulation is also associated with hypertensive episodes. Thus, systolic BP should be maintained between 120 and 140 mm Hg.12

Which antihypertensive agents?

Vasodilators such as hydralazine, nitroglycerine and nitroprusside should

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not be used to reduce BP since resulting vasodilatation can lead to cerebral hyperperfusion and elevated ICP, which may persist for longer periods. The BP should be reduced with beta blockers or calcium channel blockers such as nicardipine and clevidipine as per the Neurocritical Care Society consensus statement and AHA/ASA recommendations.10,11 However, beta blockers should be avoided in patients presenting for deep brain stimulation electrode implantation as their use suppresses the tremors and makes the task of assessing the success of stimulation more difficult.

Conclusion: Following are the target pressures in various acute intracranial events.

Intracranial event	Target Pressure
TBI	CPP 60-70 mm Hg
ICH	120-140 mm Hg systolic
aSAH	120-140 mm Hg systolic
AIS prior to reperfusion	140-180 mm Hg systolic

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