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BLOOD PRESSURE MANAGEMENT IN ACUTE CEREBROVASCULAR BRAIN INJURY

Abstract

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Stroke alters the cerebral autoregulation. We present a case series of three stroke types. Intracerebal hemorrhage, ischemic infarct and SAH (subarachnoid hemorrhage) each requiring ICU care and BP blood pressure optimization to maintain CPP and MAP. The stroke types that were managed require different MAP (mean arterial pressure), CPP (cerebral perfusion pressure), systolic blood pressure (SBP) and diastolic blood pressure (DBP) to maintain adequate cerebral perfusion. Blood pressure optimization in stroke has a major role in neuroprotecetion. The write up also stipulates recommended ranges of CPP, MAP, ICP (Intracranial Pressure), SBP and DBP. Judicious administration of intravenous antihypertensive (IAH) agents is also discussed. Keywords: Stroke, Autoregulation, CPP, MAP, ICP, BP management, Intravenous Antihypertensives

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Introduction

Diseases of the vascular system which include stroke are the second most common cause of death worldwide. Stroke can be classified into two major categories: ischemic and hemorrhagic. About 80% of strokes are ischemic and 20% are hemorrhagic. The proportion of hemorrhagic strokes is higher in Asia when compared to the West[1]. Hemorrhagic stroke differs from subarachnoid hemorrhage(SAH) in its location. Intracranial aneurysm is responsible for about 85% of SAHs; 10% are represented by nonaneurysmal conditions; 5% are represented by other medical lesions of cerebral artery, coagulopathy, neoplasms or drug abuse conditions such as inflammatory or non-inflammatory.[2]

Under normal circumstances cerebral autoregulation minimizes deviations in cerebral blood flow (CBF) when cerebral perfusion pressure (CPP) changes. Cerebral autoregulation acts through vasomotor effectors that control cerebrovascular resistance (CVR). Previous studies have convincingly documented the ability of this physiologic system to maintain relatively constant CBF when CPP is within the range 50-170 mm Hg[3]. Stroke causes disruption of the cerebral autoregulation (Fig 1). All efforts are made by compensatory mechanisms to maintain adequate CBF and CPP. However the regulatory mechanism is compromised and all treatment efforts should target an optimum BP, MAP and CPP range to ensure adequate cerebral blood flow.

The objective of this write up is for to understand the reasoning behind the targeted blood pressure in acute cerebrovascular brain injury. Not just in terms of SBP (systolic blood pressure) and DBP (diastolic blood pressure) but also CPP and ICP (intracranial pressure). We present three cases of acute cerebrovascular brain injury; hemorrhagic stroke, subarachnoid hemorrhage and malignant cerebral infarction. The cases present different scenarios where BP control was achieved after targeting different parameters.

Cases and discussion

#.Hemorrhagic stroke

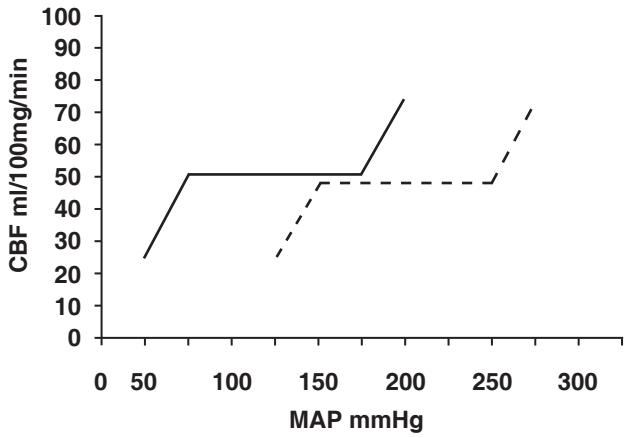
A 38 year old male experienced sudden onset of dizziness and severe headache. It was followed by loss of consciousness for about an hour. He was rushed to ER, on the way his BP was measured to be 200/120 and was given Catapress. At the ER, he was drowsy and dysarthric. The pupils were isocoric and equally reactive to light. Left hemiparesis,hyporeflexia and Babinski's sign were also elicited. Plain cranial CT scan revealed right putaminal hemorrhage. The patient was diagnosed of essential hypertension 7 years ago and was on Imidapril 10 mg od. There was family history of hypertension. He was a smoker and occasionally alcoholic. The patient was admitted to the NCCU for control of BP to prevent rebleed and herniation. He was given mannitol, 3NaCl 200cc q4hrs, and nicardipine.

Primary intracranial hemorrhage is predominantly a result of chronic hypertension and degenerative changes in cerebral arteries. Large hemorrhage causes displaced to the opposite side of the cranium which is often followed by brainstem compression[4]. Blood pressure (BP) is frequently, and often markedly, elevated in patients with acute ICH. These elevations in BP are greater than that seen in patients with ischemic stroke[5]. Studies suggest that systolic BP above 140 to 150 mm Hg within 12 hours of ICH is associated with more than double the risk of subsequent death or dependency[6,7]. INTERACT (INTensive Blood Pressure Reduction in Acute Cerebral Hemorrhage Trial) trial revealed lower hematoma expansion rates associated with intensive BP treatment and ATACH Antihypertensive Treatment in Acute Cerebral Hemorrhage) trial also confirms the feasibility and safety of early rapid BP lowering in ICH using intravenous Nicardipine. The BP of the patient at NCCU admission was 190/100. Nicardipine was started at 15 mg /hr and it was titrated according to SBP changes. The target SBP range was set at 140-160 mm Hg and MAP target was set at about 110 mm Hg. After three days of Nicardipine infusion the patient archived the target BP. Medical decompression and other supportive management were continued.

Recommendation for primary intracerebral hemorrhage

- Monitor and maintain SBP of about 160 mm Hg or MAP of about 110 mmHg.[1]

Figure 1: Autoregulatory Curve



CBF = cerebral blood flow
MAP= mean arterial pressure
Normotensive ———
Hypertensive - - - -

#.Subarachnoid hemorrhage

A 60 year old female suddenly started to experience severe throbbing frontal headache and episodes of vomiting. The patient was taken to a hospital where her BP was 200/110. She was given catapres and metoclopromide. The symptoms persisted and the patient was brought to ER. At the ER the BP was 150/70 and she was noted to have seizure for about 1 minute. She had spontaneous eye opening, answered some question and followed some commands. Pupils were 3mm equally reactive to light and isocoric. There was spontaneous movement of all the extremities. Babinski's sign was noted on the right side. She was a hypertensive on micardis 40 mg od and there was also family history of hypertension. A CT was done which revealed subarachnoid hemorrhage in bilateral fronto parietal region. Patient was transferred to NCCU for BP control, seizure prevention, monitoring of vasospasm, treatment of raised ICP and prevention of herniation. She was given nimodipine, valproic acid, leviteracetam, mannitol, fentany, dexmedometidine and Nicardipine Aneurysms form small, thin-walled blisters protruding from arteries of the circle of Willis or its major branches. Their rupture causes a flooding of the subarachnoid space with blood under high pressure.[8] Aneurysm rebleeding is associated with very high mortality and poor prognosis for functional recovery in survivors. The risk of rebleeding is maximal in the first 2 to 12 hours, with reported rates of occurrence between 4% and 13.6% within the first 24 hours. More than one third of rebleeds occur within 3 hours and nearly half within 6 hours of symptom onset, and early rebleeding is associated with worse outcome than later rebleeding.

SBP of >160 is associated with rebleeding.[9] The BP at the time of admission at NCCU was 150/ 100. Nicardipine infusion was started at 2 mg/hr and was titrated according to the SBP, Amloipine 10 mg od was also given. Target SPB of <150 was set. The TCD revealed vasospasm of anterior communicating artery. An anterior communication artery saccular aneurysm was seen on CT angiogram. It was followed by a 4 vessel angiogram which revealed a Saccular aneurysm on the left cerebral-anterior communication artery and there was also another highly suspicious aneurysm in the inferomedial aspect of distal left A1-anterior communicating artery junction. The former was successfully secured by coiling. A TCD was done the next day. Mean flow velocities and Lindegaard ratio were suggestive of ongoing vasospasm. Upper limit of SBP was raised to 220 and therapeutic hypertension was induced maintaining SPB around 170 -200. Subsequent TCD showed a down trending of vasospasm. There was no vasospasm after 10 day from ictus. Medical decompression, medicines forvasospasm and pain were continued.

Recommendation for unsecured aneurismal SAH

SBP< 150mmHg in the pre operative(unsecured aneurysm) phase seems to be reasonable.[1]

SBP of <220 in post operative(secured aneurysm) phase can be maintained if there is ongoing vasospasm[10].

#. Malignant ischemic infarct

A 63 old male was seen well going to bed.The next day he was unable to rise from the bed. He was also noted to be drowsy with right sided weakness and slurring of speech. The Patient was brought to ER where he was lethargic, followed some commands, pupils were 3mm right, 6mm left, the ER for the BP of the patient was 200/100 (MAP 133.3) . The patient was drowsy and the pupils were anisocoric. Left pupil was 3mm right pupil was 5mm. Bilateral Babinski was also noted. He was a diagnosed case of ischemic heart disease a hypertension, atrial fibrillation and underwent CABG 5 years ago. The 2decho showed EF of 35-40% and a LV thrombus. CT scan showed a showed acute left middle cerebral artery ischemic infarct with midline shift. The patient was given mannitol, atorvastatin, citicholine, ivabradine, imdur, enalapril, bisoprolol, clopidogrel, pantoprazole, and spironolactone. At The patient was immediately admitted to NCCU for management of intracranial hypertension and subfalcine herniation. The Patient was given Mannitol, 2% Nacl, Phenobarbital, Albumin, Amiadarone, Trimetazidine, Atorvastatin.

Elevated blood pressure is common during acute ischemic stroke. Extreme arterial hypertension and hypotension are detrimental.[11] Both types of stroke cause events that lead to an increase in ICP. The human cranium has a fixed amount of space with three main components: CSF, brain parenchyma and blood. If any space occupying lesion or any of these constituents volume is increased beyond the compliance, an elevation of ICP is inevitable. Malignant infarct is associated with extensive edema and raised ICP. MAP and SBP do not reflect the intracranial **CBF. To monitor ICP invasive ICP monitoring is required.**[12] ICP monitoring also sets thresholds for medical and surgical decompression. *The MAP of the patient at Admission to NCCU was 135. Nicardipine infusion was started at the rate of 10mg/hr and titrated according to the BP. Initially a MAP goal was set at 100 to 120 mm Hg. However after the insertion of ICP probe and IJ lumen ICP, CVP and CPP were targeted instead of MAP. New targets were ICP of < 15 mm H20, CVP of 8-12 mm H20 and CPP of 50-70. In subsequent days the patient did not reach an ICP of 15 mm H20 or more the CPP ranged from 55-73 and CVP fluctuated between 7-10mm H20. The patient did not undergo any surgical decompression and was managed medically*

Recommendations for Acute Ischemic Stroke

- Allow “permissive hypertension” during the first week to ensure adequate CPP but ascertain cardiac and renal protection
- Treat if SBP>220 or DBP>120 or MAP>130
- Defer emergency BP therapy if MAP is within 110-130 or SBP=185-220 mmHg or DBP=105-120 mmHg, unless in the presence of Acute MI, Congestive heart failure, Aortic dissection, Acute pulmonary edema, Acute renal failure and Hypertensive encephalopathy
- Treat with small doses of IV antihypertensives patients who are potential candidates for rtPA therapy who have persistent elevations in SBP >185 mmHg or DBP >110 mmHg. Maintain BP just below these limits.
- Use the following locally available intravenous anti-hypertensives in acute stroke:

Table 1:

Drug	Dose	Onset of Action	Duration of Action	Availability/ Dilution	Stability	Adverse Reactions	Action
Nicardipine	1-15 mg/hour	5-10 mins	1-4 hours	(10 mg/10 ml amp); 10 mg in 90 ml NSS/ D5W	1 to 4 hours	Tachycard ia, headache, flushing, dizziness, somnolence, nausea	Inhibits calcium ion from entering slow channel, producing coronary, vascular, smooth muscle relaxation & vasodilatation
Hydralazine	IV push 10-20 mg/dose q 4-6 hours as needed, may increase to 40 mg/dose	10-20 mins	3-8 hours	25 mg/mL amp; 25 mg/tab	4 days	Tachycard ia, flushing, headache, vomiting, increased angina	Direct vasodilatation of arterioles & decreased systemic resistance
Labetalol	5 mg IV push over 2 mins, repeat with incremental dose of 10, 20, 40, 80 mg until desired BP is achieved or a total dose of 300 mg has been Administered	2-5 mins	2-4 hours	5 mg/ml in 40 ml vial; 250 mg in 250 mL NSS/ D5W	72 hours	Orthostatic hypotension, drowsiness, dizziness, lightheadedness, dyspnea, wheezing & bronchospasm	Alpha- & b etablocker. Betaadrenergic blocking activity is 7x > than alpha-adrenergic blockers. Produces dosedependent ¯ in BP without significant ¯ in HR or card iac Output
Esmolol	0.25-0.5 mg/ kg IV push 1-2 mins followed by infusion of 0.05 mg/kg/min. If there is no response, repeat 0.5 mg/kg b olus dose & infusion to 0.10 mg/kg/ min. maximum infusion rate=0.30 mg/ kg/min	2-10 mins	10-30 mins	100 mg/10 ml vial; 2,500 mg in 250 mL D5W/NSS	48 hours	Hypotension, bradycardia, AV block, agitation, confusion, wheezing / bronchoconstriction, phlebitis	Short-acting beta-adrenergic blocking agent. At low do ses, has little effect on beta2 receptors of bronchial & vascular smooth muscle

Recommendations for ICP monitoring in raised ICP

- Maintain CPP 60-70 mm Hg[1] (Treat ICP > 20)[12]

Recommendations for Arterial hypotension in Acute ischemic stroke

- Baseline SBP <100 or DBP< 70 mmHg is associated with higher rates of neurological worsening, poor neurological outcomes and death.[1]
- Cause of arterial hypotension should be sought : aortic dissection, volume depletion, blood loss, ad decreased cardiac output or arrythmiaas
- Correct hypovolemia with NSS and treat arrhythmias to optimize cardiac output
- Available vasopressor agents include dopamine, dobutamine and phenylepinephrine and adrenaline.

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