Arterial Hypotension and Hypertension

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Carotid Endarterectomy (CEA)

Overview

Atherosclerosis is a systemic disease, and, accordingly, patients with carotid plaque also often have coronary artery disease. Moreover, long-standing essential hypertension is frequently a comorbidity along with its effects on cardiac function (e.g., diastolic dysfunction or left ventricular hypertrophy). Both hypertension and hypotension are common.

In patients with poorly controlled hypertension, the blood pressure range for normal cerebral autoregulation is shifted to the right (Fig. 45.1). Moreover, in patients with advanced carotid artery disease, cerebral autoregulation is often impaired, making regional cerebral blood flow (CBF) exquisitely sensitive to perfusion pressure. Carotid sinus baroreceptor manipulation can cause bradycardia and hypotension.

Severe hypertension (>180/110 mmHg) increases overall morbidity and mortality. The most common complication of hypertension is myocardial ischemia. Hypotension can worsen ischemic neurologic deficits. Postoperative hypertension can lead to hyperperfusion syndrome. Active blood pressure management is essential.

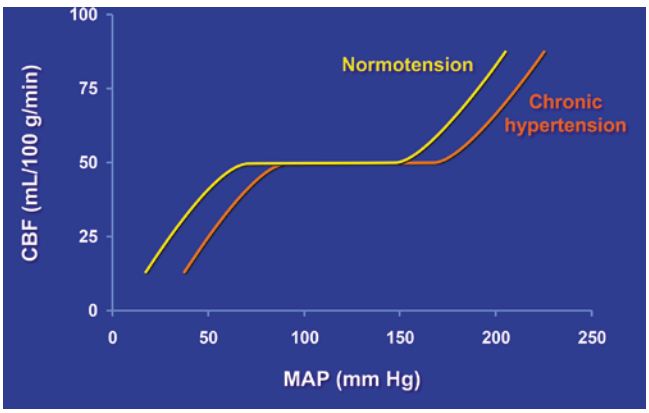

Fig. 45.1 The relationship between cerebral perfusion pressure (CPP) and mean arterial pressure (MAP) shows autoregulation of CPP across a range of MAP. The curve is shifted to the right in patients with chronic hypertension

Hypertension and Hypotension in CEA: Prevention

Chronic hypertension should be well controlled before CEA. Ordinary intraoperative alterations in the level of stimulation should be anticipated and treated early with small doses of therapeutic medication. An arterial line is recommended for beat-to-beat assessment and precise control of blood pressure.

Blood pressure is maintained at preoperative, awake values until the carotid artery is cross-clamped. If there are no contraindications, blood pressure is then increased by as much as 20% during cross-clamping to improve perfusion *via* collateral vessels. Small doses of phenylephrine or ephedrine are appropriate. Careful phenylephrine infusion is often used although it is associated with a higher risk of myocardial ischemia compared to lightened anesthesia. Coughing and other causes of blood pressure spikes during emergence should be averted to minimize stress on the freshly repaired artery and the potential complication of neck hematoma.

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Hypertension and Hypotension in CEA: Crisis Management

Table 45.1 summarizes intraoperative events requiring blood pressure management, signs or indications for treatment, and suggested therapy for hypertension and hypotension during CEA.

Table 45.1 Intraoperative events requiring blood pressure management, signs or indications for treatment, and suggested therapy for hypertension and hypotension during CEA

Event/pathology	Signs/indications for treatment	Suggested therapy
Direct laryngoscopy, intubation, incision	Hypertension, tachycardia	Deepen anesthetic
		Lidocaine
		Fentanyl/remifentanil
		Esmolol/metoprolol
		Nicardipine/clevidipine
		Nitroglycerin
Post-induction	Hypotension	Lighten anesthetic
		Phenylephrine
		Ephedrine
Chronic untreated hypertension	Severe or refractory intraoperative hypertension	Nitroprusside
		Nitroglycerin
		Esmolol
Intravascular volume depletion, hemorrhage	Hypotension, tachycardia, systolic pressure variation	Intravenous fluid
		Blood
		Phenylephrine
		Ephedrine
Carotid cross-clamping	Blood pressure low or low-normal	Increase baseline blood pressure by 10–20% (phenylephrine, decreased anesthetic dose)
Carotid sinus baroreceptor manipulation	Hypotension, bradycardia	Vagolytic (atropine, glycopyrrolate)
Decrement in neurologic monitoring (e.g., EEG slowing)	Hypotension	Raise blood pressure with phenylephrine, ephedrine, lighten anesthetic
Emergence, coughing	Hypertension	Beta-blocker (labetalol, esmolol)
		Lidocaine
		Remifentanil

Hypertension and Hypotension in CEA: Key Points

- The autoregulation curve is shifted to the right in patients with chronic hypertension.
- Prevention of blood pressure lability is achieved by anticipating stimuli and using small doses of therapeutic drugs.
- Normotension is the goal during CEA before and after cross-clamping of the carotid artery. During cross-clamping, blood pressure should be mildly elevated from baseline (approximately 20% is appropriate).

Aneurysm/AVM Repair

Overview

Tight control of blood pressure during aneurysm repair or arteriovenous malformation (AVM) resection is essential. Hypertension increases transmural pressure and risks vessel rupture and subarachnoid hemorrhage, brain edema, and postoperative cerebral hyperperfusion. Conversely, hypotension lowers cerebral perfusion pressure (CPP) and risks ischemia, especially in areas of vasospasm. Moreover, acute hypertension may be a sign of aneurysm rupture and intracranial hypertension.

Hypertension and Hypotension in Aneurysm/AVM Repair: Prevention

Anticipation of stimulating events prevents abrupt blood pressure changes. A preinduction arterial line is routinely used. Small doses of pressors guard against marked reductions in blood pressure following induction of anesthesia. Dramatic blood pressure increases may occur with laryngoscopy, pinion placement, incision, dural opening, and surgical manipulation. Lidocaine, esmolol, or short-acting opioids will diminish hemodynamic stimulation from these events. Nimodipine and nicardipine, calcium channel blockers, may improve neurologic outcome by preventing the development of vasospasm after aneurysmal hemorrhage.

Hypertension and Hypotension in Aneurysm/AVM Repair: Crisis Management

If an aneurysm ruptures intraoperatively, immediate temporary proximal occlusion of the culprit vessel by the surgeon is necessary. Maintaining cerebral perfusion should continue to be the goal while the surgeon gains control.

If vasospasm develops, more aggressive therapy combining hemodynamic augmentation ("triple-H therapy"), angioplasty, and intra-arterial infusion of vasodilator drugs is used. Excessive hypertension risks rebleeding.

Table 45.2 summarizes intraoperative events requiring blood pressure management, goal of intervention, and suggested therapies.

Hypertension and Hypotension in Aneurysm/AVM Repair: Key Points

- Strict maintenance of normotension minimizes abrupt changes in transmural pressure and optimizes collateral circulation.

- Anticipation of stimulating events is key to optimal blood pressure management.
- If rupture occurs, blood pressure should be kept in the high normal range to maintain CPP and minimize ischemia.
- Inducing mild hypertension following subarachnoid hemorrhage improves neurologic outcome, but marked hypertension increases the risk of rebleeding.

Table 45.2 Intraoperative events requiring blood pressure management, goal of intervention, and suggested therapies

Event	Goal of blood pressure management	Suggested therapy
Dissection, manipulation of the aneurysm or AVM, placement of temporary clips on the parent vessel, aneurysmal clip ligation, and closure	Normotension	Deepen anesthetic
		Lidocaine
		Fentanyl
		Labetalol, esmolol
		Nicardipine
		Phenylephrine
		Ephedrine
Following temporary clipping of parent artery	Normotension, or may need to increase blood pressure approximately 20% to maintain CPP	Phenylephrine
		Ephedrine
Rupture	Normotension; may need to increase blood pressure to maintain adequate CPP if intracranial pressure rises, balance with need to control hemorrhage	Maintain intravascular volume
		Phenylephrine
		Ephedrine
Vasospasm	Induced hypertension as part of "triple-H" therapy (hypervolemia, hypertension, hemodilution)	Volume expansion
		Phenylephrine
		Dopamine

Acute Traumatic Brain Injury (TBI)

Overview

Traumatic brain injury (TBI) per se is associated with hypertension either due to increased circulating catecholamines or as a response to intracranial hypertension. Cerebral autoregulation is impaired which increases the risk of hypertension causing hyperemia, vasogenic edema, and intracranial hypertension. In the setting of intracranial hemorrhage (ICH), minimizing hypertension has been shown to reduce hematoma growth. Hypotension can develop when TBI is severe or accompanied by systemic injuries and significant blood loss.

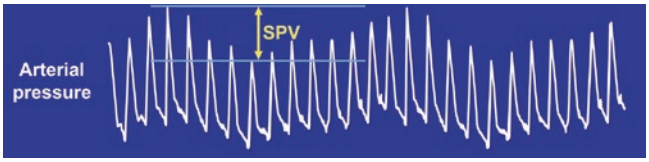

Fig. 45.2 Systolic pressure variation (SPV) is the measured difference between minimum and maximum systolic pressure within one respiratory cycle. Increased SPV reflects volume-sensitive hypotension

Hypertension and Hypotension in Acute TBI: Prevention

Intra-arterial pressure monitoring is customary, although lines may need to be placed during or after surgical incision to facilitate immediate surgical intervention and thereby limit secondary brain injury.

Hypotension is usually a sign of hypovolemia from other injuries. Hypovolemia is best assessed by clinical signs including arterial blood pressure and systolic pressure variation (Fig. 45.2). Urinary output may be confounded as a guide if mannitol has been given.

Hemorrhage should be treated with immediate and aggressive volume resuscitation. Glucose-containing solutions should be avoided because they are associated with worsened neurologic outcomes. To prevent hypotension in the patient receiving large amounts of mannitol, diuresed fluid should be replaced. Control of bleeding and volume resuscitation takes precedence over immediate surgical intervention, as systolic pressure of less than 80 mmHg leads to poor outcomes.

Hypertension and Hypotension in Acute TBI: Crisis Management

Targets for treating hypertension are based on intracranial pressure (ICP) and CPP. Although controversial, CPP should be 70 mmHg or greater. Overaggressive fluid administration may worsen intracranial edema and ICP.

The 2007 guidelines set a systolic blood pressure target of less than or equal to 180 mmHg. Recent data strongly suggest that a lower systolic pressure of 140 mmHg reduces hemorrhagic growth and risk of poor outcomes. No specific agents have been shown to be superior.

Table 45.3 summarizes differential diagnoses and suggested therapeutic approaches to hypertension and hypotension in TBI.

Table 45.3 Differential diagnoses and suggested therapeutic approaches to hypertension and hypotension in TBI

Sign	Differential diagnosis	Therapeutic approach
Hypertension, tachycardia	Catecholamine release	1. Lower ICP
	Increasing ICP	2. Ensure adequate anesthesia, oxygenation, ventilation
	Inadequate anesthesia	
	Coughing, bucking	3. Avoid hypocapnia unless herniation evident
		4. Beta-blocker (avoid vasodilators in a closed cranium)
Hypertension, bradycardia, irregular respiration	Impending herniation (Cushing phenomenon)	Manage blood pressure based strictly on direct ICP monitoring
Hypotension, tachycardia	Hypovolemia	1. Isoosmolar colloid or crystalloid solutions
	Hemorrhage from systemic injuries	2. Transfusion
	Acute mannitol diuresis,	3. Vasopressor or inotrope
	Central diabetes insipidus	1.1.4. Vasopressin for diabetes insipidus
	Chronic diuretic therapy	
	Severe traumatic brain injury (variable heart rate)	

Hypertension and Hypotension in Acute TBI: Key Points

- TBI alone is associated with increased catecholamines and arterial hypertension.
- Arterial hypertension may be a marker of intracranial hypertension and impending herniation.
- Limiting hypertension after ICH reduces hematoma formation.
- Hypotension should be treated as hypovolemia, until proven otherwise.

Suggested Reading

Carotid Endarterectomy

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