

Intraoperative Hypotension & Hypertension

Determinants of Blood Pressure

Blood Pressure (BP)

- BP represents the force exerted by circulating blood on the walls of blood vessels.
- A product of 1) cardiac output and 2) vascular tone.

Cardiac Output (CO)

- $CO = HR \times SV$

Heart Rate (HR)

- Dependent on the interplay between the sympathetic and parasympathetic nervous systems.
- In infants, SV is fixed, so CO is dependent on HR.
- In adults, SV plays a much more important role, particularly when increasing HR is not favorable.

Determinants of Blood Pressure

Stroke Volume (SV)

- Dependent on preload, afterload, and myocardial contractility.

Preload

- Volume of blood in the ventricle at end-diastole (LVEDV)

Afterload

- Resistance to ejection of blood from the ventricle
- SVR accounts for 95% of the impedance to ejection
- $SVR = [(MAP - CVP) \times 80] \div CO$

Contractility

- The force and velocity of ventricular contraction when preload and afterload are held *constant*.
- Ejection fraction (EF) is one of the most clinically useful indices of contractility (normal EF is ~60%).

Components of Blood Pressure

Systolic Blood Pressure (SBP)

- Highest arterial pressure in the cardiac cycle.
- Dicrotic notch = a small notch in the invasive arterial pressure curve that represents closure of the aortic valve, producing a brief period of retrograde flow.

Diastolic Blood Pressure (DBP)

- Lowest arterial pressure in the cardiac cycle

Mean Arterial Pressure (MAP)

- $MAP = 2/3 DBP + 1/3 SBP$, or $(2 \times DBP + SBP) \div 3$

Components of Blood Pressure

Pulse Pressure

- **PP = SBP - DBP**
- Normal PP is ~40 mm Hg at rest, and up to ~100 mm Hg with strenuous exercise.
- Narrow PP (e.g. < 25 mm Hg) = may represent aortic stenosis, coarctation of the aorta, tension pneumothorax, myocardial failure, shock, or damping of the system.
- Wide PP (e.g. > 40 mm Hg) = aortic regurgitation, atherosclerotic vessels, PDA, high output state (e.g. thyrotoxicosis, AVM, pregnancy, anxiety)

Blood Pressure Measurement

Non-Invasive Blood Pressure (NIBP)

- Oscillometric BP determination: oscillations in pressure are detected through the cuff as it deflates.
- MAP is measured as the largest oscillation; it is the most accurate number produced by NIBP.
- SBP and DBP are calculated by proprietary algorithms in the machine.

Invasive Arterial Blood Pressure (IABP)

- Most accurate method of measuring BP.
- If system is zeroed, leveled, and properly damped, SBP, DBP, and MAP are very accurate.

Intraoperative Hypertension

- “Light” anesthesia
- Pain
- Chronic hypertension
- Illicit drug use (e.g. cocaine, amphetamines)
- Hypermetabolic state (e.g. MH, thyrotoxicosis, NMS)
- Elevated ICP (Cushing’s triad: HTN, bradycardia, irregular respirations)
- Autonomic hyperreflexia (spinal cord lesion > T5 = severe; < T10 = mild)
- Endocrine disorders (e.g. pheochromocytoma, hyperaldosteronism)
- Hypervolemia
- Drug contamination - intentional (e.g. local anesthetic + Epi) or unintentional (e.g. “Roc-inephine”)

Treatment of Hypertension

- Temporize with fast-onset, short-acting drugs, but ultimately diagnose and treat the underlying cause.
- Pharmacologic Interventions:
 - Volatile anesthetics (cause vasodilation while deepening anesthetic)
 - Opioids (treat pain and deepen the anesthetic)
 - Propofol (quickly sedates the “light” patient; also a vasodilator)
 - Beta-blockers (e.g. esmolol, labetalol, metoprolol)
 - Vasodilators (e.g. hydralazine, NTG, SNP)

Intraoperative Hypotension

- Excessive depth of anesthesia
 - Overdose of induction agent, volatile, or narcotic.
- Inadequate Preload (“the tank is empty”)
 - Hypovolemic shock (hypovolemia, anemia)
 - Increased intrathoracic pressure (e.g. excessive PEEP, I:E ratio, PTX, caval compression, chronic HTN)
- Reduced Afterload
 - Vasodilated states (e.g. liver failure, sepsis/SIRS/shock, anaphylaxis)
 - Depleted catecholamine states (e.g. adrenal suppression from chronic steroid use, methamphetamines, cocaine)
- Diminished Afterload
 - Acute MI, non-perfusing arrhythmia, cardiomyopathies, valvulopathies
 - Pulmonary HTN (decreases LVEDV)

Treatment of Hypotension

- Temporize with fast-onset, short-acting drugs, but ultimately diagnose and treat the underlying cause.
- Turn down the anesthetic (2 MAC? Too much!)
- Volume
 - Reevaluate EBL; replace with crystalloid, colloid, or blood, as needed.
 - Reevaluate patient’s fluid status (deficit, maintenance, and ongoing losses; urine output?).
 - Consider CVP, PAC, or TEE
- Ventilation
 - Reduce PEEP to improve venous return.
 - Decrease I:E ratio to shorten inspiratory time.
 - Rule out PTX
- Metabolic
 - Treat acidosis and/or hypocalcemia

Treatment of Hypotension

- Drugs
 - **Phenylephrine** (Neosynephrine) = α_1 agonist
 - Direct vasoconstrictor
 - Use in vasodilated state with tachycardia
 - Can cause reflex bradycardia
 - **Ephedrine** = α_1 , β_1 , and β_2 (less so) agonist
 - Direct and indirect adrenergic stimulation via NE release
 - Use in vasodilated, bradycardic, low CO states
 - **Epinephrine** = β_1 , α_1 , α_2 , and β_2 agonist
 - Endogenous catecholamine
 - Causes vasoconstriction and increased CO.
 - **Inotropes** (in low CO states)
 - Dopamine, Epinephrine, Milrinone, Dobutamine
 - **Stress-dose steroids** - consider 100 mg hydrocortisone if steroids taken in past 6 months.

References

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