

Cardiovascular Therapy of the Neurosurgical Patient

INASNACC

Basic consideration

- Autoregulation
- Intracranial pressure
- $CPP = MAP - ICP$

Cerebral oxygen delivery

- Cardiac output : preload, contractility, heart rate, after load.
- Cardiac failure: hypovolemia (blood loss, osmotic diuretic, fluid restriction, diabetes insipidus) resulting in insufficient preload, CNS cause (spinal cord and brain stem injury, neurogenic cardiomyopathy), drugs induced (calcium antagonist, beta blocker).
- Blood pressure : $MAP = CO \times SVR$.

Drugs used in cardiovascular therapy

- Guidelines for administration vasoactive drugs.
- Adrenergic agonist
- Adrenergic antagonist
- Phosphodiesterase inhibitors.
- Vasodilators
- Vasoconstrictors

Guidelines for administration vasoactive drugs.

- Use a calibrated pump for all infusions.
- Inject into the intravenous line as close to vein insertion site as possible.
- Avoid using SBP as the goal of therapy because this value is prone to fluctuate from a number of cases, not all which are related to the patients cardiovascular condition. MAP is more reliable variable,
- Potent vasoactive drugs should be administered in a grade fashion starting with a dose below one that is thought to be therapeutic.

Adrenergic agonist

- Norepinephrine
- Epinephrine
- Dobutamine
- Dopamine
- Phenylephrine
- Isoproterenol

Adrenergic antagonist

- Esmolol
- Metoprolol
- Labetalol
- Propranolol
- Phentolamine

Phosphodiesterase inhibitors

- Milrinone is a non adrenergic that enhances contractility and promotes pulmonary and arterial vasodilatation

vasodilators

- Nicardipine
- Sodium nitroprusside (SNP)
- Nitroglycerin (NTG)
- Adenosine
- Hydralazine

vasoconstrictors

- Vasopressin (antidiuretic hormone) is a peptide with potent vasoconstricting properties, which can be useful in increasing arterial tone.

Goal-directed pharmacologic intervention

- Increasing MAP is indicated in a number of clinical setting : decreased SVR, increasing CPP, anaphylacting reaction, CPR.
- Increasing CO is indicated in a number of clinical setting : low CO owing to cardiomyopathy, cerebral vasopasm.

Goal-directed pharmacologic intervention.....

- Lowering MAP can be indicated in a number of clinical settings : hypertension, surgical exposure.
- Lowering HR can be indicated in a number of clinical settings: reduced myocardial oxygen consumption, atrial fibrillation, hypertension.

Management of cardiovascular complication of neurologic disorder

- Cerebral vasospasm (Triple-H therapy).
- Autonomic hypotension (neurogenic shock).
- Carotid endarterectomy (post operative hypotension).
- Carotid endarterectomy (post operative hypertension).
- Acute TBI with hypotension or hypertension
- Post operative hypertension

Triple-H therapy

- Vasospasm is a potentially devastating complication that occurs after SAH and acute TBI.
- Rationale: Triple-H therapy seeks to increase blood flow through narrowed arteries by decreasing viscosity (hemodilution), increasing the driving pressure (hypertension), and expanding the blood volume (hypervolemia).
- Needed invasive monitoring: CVP, PA, arterial line.
- Have risk of pulmonary edema

Acute TBI with hypertension

- Secrete excessive catecholamine.
- Cushing reflex as protective mechanism to maintain CPP.
- treatment: all maneuver to decrease ICP.

Postoperative hypertension

- In absence of intracranial hypertension, post craniotomy hypertension should be treated to reduce risk of cerebral swelling and bleeding in operative site.
- However, if cause is elevated ICP, target therapy is to decrease ICP.
- Analgesic without risk of bleeding (choice is NSAID without risk of bleeding).