2007b(9): Briefly describe the cardiovascular responses to central neural blockade

Central neural blockade achieved by subarachnoid (spinal) or epidural
- LA (bupivacaine, ropivacaine) &/or opioid (fentanyl, morphine)
- Effects will be more pronounced in:
  - Elderly (↓physiological reserve)
  - Fixed cardiac output states → risk of critical cardiac event
  - ↓blood vol (hypovolaemic)

**CVS Response** → ↓MAP
- 2° blockade of β sympathetic chain fibres
  - Run in thoracolumbar region → therefore level of central block will affect degree of ↓MAP
- MOA: removes tonic SNS activity on vascular smooth muscle
  - Blocks α1 adrenoceptors (GPCR → ↑PKC → ↑IP3/DAG → ↑Ca → constriction) and β2 receptors (GPCR → ↑cAMP → ↓Ca → dilation)
- Result:
  - vasodilation (arteriolar) → ↓afterload
  - venodilation (venous) → ↑venous capacitance
  - veno- > vasodilation → ~75% blood vol pools in venous circulation

**Level of block**
- Brainstem block → inhibition of vasomotor centre → unable to activate SNS response → profound ↓MAP → life threatening
- ‘High block’ → T1-T4 cardio-acceleratory centre → blockade → unable to ↑HR/contractility with SNS stimulation → profound ↓MAP
- Mid-thoracic / Renal level block → ↓GFR → activation of RAA system by ↓afferent arteriolar stretch
- Sacral block → nil sympathetic chain blockade (only parasympathetic fibres) → minimal effect on peripheral vascular tone

**Detector Systems**
1. High pressure baroreceptors (carotid sinus, aortic arch) → ↓stretch → ↓inhibitory input to SNS → stimulation of vasomotor centre

Result:
- ↑SNS → Heart (↑HR, ↑contractility); vasoconstriction (unable to below level of blockade); lesser degree venoconstriction to mobilise blood reservoirs (liver, lungs, skin)
  - Unable to compensate for large loss blood vol to capacitance system
- Activation of renin-angiotensin system (direct, β1R stimulation)
  - ↑renin – cleave angiotensinogen → ATI →ACE → ATII
  - ATII → direct vasoconstrict → min effect on venous capacitance
- ↑ADH release from post pituitary
  - ↑H2O reabsorption DCT (↑urea transport into renal medulla)
  - Vasoconstriction 2° V1R stimulation in vessels
2. Low pressure baroreceptors (right atrium, great vessels) → ↓stretch → ↓ANP secretion

Result: ↓inhibition RAA / ADH system, ADH; ↓Na/H2O release

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