By Amanda Diaz

2003b(10)/2000b(1): Describe the role of baroreceptors in the control of systemic arterial pressure

General: Arterial pressure is maintained within narrow limits during normal functioning.

\[ \text{MAP} = \text{CO} \times \text{SVR} \]

Achieved by:
- Detection system (baroreceptors)
- Central control (medulla, hypothalamus, higher centres)
  - Baseline: SNS tone present, PNS quiescent
- Effectors (SNS/PNS efferents)

**Baroreceptors**
- Detect changes in blood pressure
- 2 types of baroreceptor:
  - High pressure
  - Low pressure (volume)

**High pressure Baroreceptors**
- Detect changes in blood pressure by degree of stretch
  - ↑rate of firing with ↑MAP (graph) → project inhibitory neurones to SNS
- Present in carotid sinus and aortic arch
- Rapid response system operating via negative feedback to maintain constant MAP via changes in SVR and CO

**Mechanism of Action:**
- ↑MAP → stretches baroreceptors
  - Inhibitory afferents to RVLM → ↓SNS activity → ↓rate of firing of efferents to heart and peripheral vessels via interomediolateral columns
    - Heart: ↓HR, ↓contractility, ↓SV
    - Vessels: ↓SVR → vasodilatation ↓tendency for venous return → venodilatation
  - Stimulatory afferents through CNIX and CNX (nucleus tractus solitaries) to medulla (vagal nucleus/nucleus ambiguous) → ↑PNS activity → ↑efferent activity to heart via vagus nn
    - Heart: ↓HR, ↓contractility, ↓SV
  - Firing rate set-point of high pressure baroreceptors can be reset to a higher level in response to chronically elevated MAP (HT)

  Clinically, response of high pressure receptors can be tested via Valsalva manoeuvre

**Low Pressure (volume) Baroreceptors**
- Detect changes in volume which cause stretching
- Present in right atrium, great veins
- Respond to ↑stretch (mechanoceptors)
- In general, provide a slow-response feedback system to exert effects on blood vol over extended periods of time
- ↑vol (~10%) → afferents via CN X to medulla → Overall inhibitory effect on heart (stimulation PNS, inhibition SNS)
  - Heart: ↓HR, ↓contractility, ↓SV, ↓CO
  - Vessels: venodilatation, vasodilatation
  - Also efferents from hypothalamus → ↓ADH, ↓thirst
  - Production of ANP from atrium
Overall effect is to ↓effective blood vol by ↑reservoir capacity, ↑salt and H₂O excretion via kidneys

**Valsalva Maneouvre**
- Clinically measure the responsiveness of high pressure baroreceptors (carotid sinus, aortic arch)
- Inspiration held against a closed glottis/nose & mouth for 10s
- Then released

**Phase 1**: Inspiration held → ↑intrathoracic pressure → slight ↑CO from emptying of pulmonary venous reservoir → ↑MAP

**Phase 2**: Continued ↑intrathoracic pressure → ↓VR (↓CO) → ↓firing of high pressure baroreceptors → ↑SNS/↓PNS → ↑HR, ↑TPR (vasoconstriction), ↓venous capacitance (venoconstriction)
  - Unable to compensate completely → MAP decreases gradually

**Phase 3**: Release of Valsalva → sudden ↓intrathoracic pressure → sudden ↑capacitance of pulmonary vasculature → ↑pulmonary blood vol → ↓CO → ↓MAP

**Phase 4**: Return of CO to normal in presence of ↑TPR → overshoot of MAP → detected by high pressure baroreceptors → ↓SNS stimulation (↓TPR, venodilatation), ↑PNS (vagal stimulation)
  - ↓HR

**Valsalva Ratio**: ratio of highest phase 2 HR: lowest phase 4 HR
- Normal = 1.5
- <1.5 in autonomic dysfunction