Advanced Cardiovascular Physiology (1st Year University notes)

End-diastolic volume:

- Volume of blood in ventricles at beginning of systole
- Depends on venous system return to the heart

Venus return:

- Skeletal muscle pump
- Respiratory pump – Pressure changes in abdomen and thorax during breathing
- Sympathetic innervations of veins/venoconstriction – increases Venous return

Increased VR → Increased EDV → Increased preload → Increased SV

Stroke volume = End diastolic volume (EDV) – End systolic volume (esv)

Ejection Fraction (EF):

- Ejection fraction = (SV/EDV) * 100
- Ejection fraction is normally above 55%
- Heart failure deduced by ejection fraction below 45%

Contractility (Inotropy):

- Intrinsic ability of heart muscle fibre to contract any given fibre length
- Independent of preload
- Depends on interaction between Actin-Myosin
- Calcium ions interacting with contractile filaments

Positive Inotropes:

- Circulating of adrenaline/NA
- Sympathetic stimulation
- Drugs – Digitalis (Cardiac glycosides) beta receptor agonists

Negative inotropes:

- Beta receptor antagonists
- Calcium channel blockers
- Heart disease, ischaemia can reduce inotropy

Afterload:

- Load against which left ventricle contracts to eject blood
- Depends on amount of blood in LV and resistance of arteries
- Greater the aortic pressure, greater LV afterload
- Afterload is increase in; hypertension, arterial constriction and valvular stenosis
- Increase in afterload results in decreased Stroke volume
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- Epinephrine (b2), PGI2 is released cAMP to the MLCK simultaneously with the calcium ions
- MLC along with ATP, Calcium ions and cAMP causes phosphorylation when contracting and vice versa
- When relaxing, MLCP is produced when NO enters the cell to cGMP and Rhokinase

Control of Arteriolar tone:

- In Neural Controls, Vasconstrictors are Sympathetic nerves and Vasodilators are Neurons that release NO
- In Hormonal controls, Vasconstrictor examples are Epinephrine, Angiotensin II and Vasopressin whereas Vasodilators are Epinephrine and Atrial Natriuretic Peptide
- In Local Controls, Vasconstrictors are Internal BP (myogenic response) whereas Vasodilators are decrease in oxygen, Osmolarity, Adenosine, Eicosanoids, Bradykinin, Substances released from injury and Nitric Oxide
- All 3 types of controls result in Arteriolar smooth muscle

Other determinants of Blood Pressure:

- Veins acting as volume reservoir (60% blood volume)
- Reduced BP leads to increased sympathetic nerve activity and constricting veins for increased venous return and increased cardiac output for BP rising
- Blood volume being inversely proportional to BP

Baroreceptor Reflex:

- Controls movement-to-movement fluctuations in MAP
- Rapid response
- Tonicly active
- Negative feedback
- Stretch sensitive mechanoreceptors found in Aortic Arch and Carotic Sinus

Baroreceptor Reflex process:

- Increase in BP
- Increase in firing of Baroreceptors in Carotid Arteries and Aorta
- Sensory neurons passed through to CV control centre in Medulla Oblongata
- Lead to either Sympathetic and Parasympathetic output
- For sympathetic, less NE released whereas Parasympathetic, more ACh on Muscarinic receptor
- Either a-receptor or B1-receptor released
- A-receptor causes Arteriolar Smooth Muscle
- B1-receptor causes either Ventricular Myocardium or SA node
- Arteriolar Smooth Muscle causes Vasodilation which causes decrease in Peripheral resistance
- Ventricular myocardium causes decrease in force of contraction, SA Node leads to lower heart rate; both leading to lower Cardiac Output
- Both decreases result in decrease in Blood Pressure
- Negative feedback cycle

Hormonal Control of Arteriolar Tone and BP:

- Renin-Angiotensin-Aldosterone system
- Anti-diuretic hormone
- Natriuretic peptides