(a) To describe the responses to change in posture.

Supine

- all blood vessels at the level of the heart
- Pressures (mmHg):
  - aorta - 100
  - arteries - 95
  - veins - 5
  - RA - 1

Standing

- pressure = egh

  e = density of blood
  g = acceleration due to gravity
  h = height

- for each cmH2O of blood above or below the heart, gravity causes the MAP to decrease or increase respectively by 0.77mmHg.

- decreased hydrostatic pressure -> 500mL of venous pooling from thorax.
- immediate fall in RA pressure to 0
- increase in hydrostatic pressure in capillaries -> more ultrafiltration + local tissue oedema.
  - decrease in VR
  - decrease in Q (by 20%)
  - decrease in MAP

Compensatory mechanisms:

Mechanical

(1) Thoracic pump

- negative intrathoracic pressure on inspiration -> 'sucks' blood from abdomen into thorax

(2) Muscular pump

- muscle contraction exerts pressure on deep veins.

(3) Venous valves
- interrupt vertical column of blood -> reduce the effective hydrostatic pressure -> reduce venous pressure in feet from 105 to 20mmHg.

**Autonomic**

- decreased MAP sensed by carotid & aortic baroreceptors
- decreased discharge through CNIX & CNX
- sensed by A2 area in tractus solitarius in posterolateral portions of medullar & lower pons.
- uninhibited sympathetic tone
- discharge of norad from adrenal medulla producers:
  1. increased HR & contractility
  2. vasoconstriction
     - sk muscle
     - skin
     - gut
     - kidneys
  3. venoconstriction

**Hormonal**

- decreased atrial stretch -> increase in ADH & decrease in ANP
- decreased blood flow -> release of AG II -> Renin -> ALD -> vasoconstriction and increase Na+ reabsorption.

**Autoregulatory**

- cerebral myogenic & metabolic mechanisms work to some extent.

**Summary**

**Mechanical**

Thoracic pump
Muscular pump
Venous valves

**Autonomic** - baroreceptor reflex -> sympathetic tone increased.

HR & contractility
Vasoconstriction arteriolar
Vasoconstriction venous

**Hormonal**

ADH
AG II-Renin-ALD axis
ANP

**Autoregulatory**

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Myogenic
Metabolic

**Trendelenburg Position**

- head down

**CNS**

- increased cerebral arterial & venous pressures -> increased capillary hydrostatic pressure ->
  - cerebral oedema
  - raised ICP
  - chemosis (can't close eyes)
  - raised intraocular pressure
  - facial + airway swelling

**CVS**

- increased VR due to redistribution of blood to central compartment.
- increased Q
- decreased ADH, increased ANP
- increased vagal tone

**RESP**

- decreased FRC
- decreased PVR
- high airway pressures
- increased alveolar deadspace

**Reverse Trendelenburg**

- head up

**CNS**

- CPP reduced
- increased sympathetic tone

**CVS**

- decreased VR
- pooling in lower limbs
- loss of muscle pump
- incompetent venous valves

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- vasoconstriction & tachycardia
- increased ADH, decreased ANP
- increased ATII-renin-ALD

**Lateral position**

- minimal effect on hydrostatic pressure of capillaries in systemic circulation.
- better blood flow to lower lung
- changes V/Q profile
- IPPV perferentially distributes ventilation to the non-dependent lung.
- right lateral decubitus produces the highest vagal tone, and lowest sympathetic tone -> protective in AMI.

(b) To account for the cardiovascular changes seen in haemorrhage & hypovolaemia.

See Table 2 - ATLS classification of shock

Shock = inadequate blood flow to maintain normal organ function.

**Symptoms & signs of haemorrhagic shock:**

- pallor
- collapse of superficial veins
- tachycardia
- decreased u/o
- hypothermia
- hypotension

**Post shock:**

- decreased SV, Q & MAP

**Seconds: - defense of blood pressure**

- sensed by atrial stretch receptors -> increased sympathetic tone -> adrenaline release from adrenal medulla.
- increased secretion of AGII-renin-ALD

**Minutes: - defense of blood volume**

- increase in plasma volume (by 500mL] from transfer of interstitial fluid into capillaries
- blood transferred from peripheral (portal & hepatic) to central compartment.

**Hours:**

- increased Na+ and H2O retension in kidneys.
- quenching of thirst

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Days: - defence of blood constituents

- synthesis of plasma proteins by hepatic synthesis.

Weeks:

- synthesis of RBC's

See graph - Q & VR curves - show affects of haemorrhage and then what happens with correction of blood volume.

(c) To explain the cardiovascular effects and responses in different forms of shock.

Types & Causes of Shock:

**Hypovolaemic**
- Haemorrhage
- Burns
- Fluid loss - V & D

**Distributive**
- Neurogenic
- Anaphylaxis
- Sepsis

**Cardiogenic**
- MI
- CHF
- Arrhythmias

**Obstructive**
- TP
- PE
- Cardiac tamponade

Cardiovascular effects:

**Hypovolaemic** - see above

**Distributive**
- vasodilation
- 'warm shock'

**Cardiogenic**
- congestion in lungs & viscera

Obstructive

- same as cardiogenic

(d) Explain the cardiovascular responses in pregnancy, exercise, cardiac failure, and during IPPV, anaesthesia, PEEP and the Valsalva manoeuvre.

Pregnancy

1st trimester

2nd trimester

3rd trimester

- Q still increased 33% but in supine position decreased secondary to hypotension from obstruction of IVC & aorta -> fetal asphyxia & distress.
- blood volume increased by 33%
- red cell volume increase by 33%
- plasma volume increases by 50%
- relative anaemia
- oxy-Hb dissociation curve shifts to right from increase in 2,3-DPG
- decrease in TPR c/o placenta acting like a AV shunt + vasodilation in kidney, GIT, heart, breasts & skin.

Labor

- pain & apprehension increase Q & SV 45% over prelabor values
- each uterine contraction increases BP, central blood volume & Q by 20%
- normal blood loss (1) NVD - 500mL (2) C/S - 1L

Post-partum

- central blood volume & SV increases owing to emptying of uterus -> relieving obstruction of IVC & aorta.

Exercise

- anticipation of exercise sends cortical signals to vasomotor centre -> decreases parasympathetic tone + increases sympathetic tone
- increase in myocardial contractility & HR -> Q

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Changes in regional blood flow:

(1) sympathetically mediated -> vasoconstriction

(2) local mechanisms (fall in PO2, increase in metabolites, rise in PCO2, accumulation of K+, increased T...) -> vasodilation

- Sk muscle O2 consumption increases 100 fold
- Renal blood flow (normal 1260mL/min -> 250)
- Liver & GI blood flow (normal 1700mL/min -> 300)
- Brain unchanged
- Heart increases (250mL/min -> 1000)
- Skin initially decreases with sympathetic vasoconstriction -> increases as T rises -> decreases as O2 consumption nears maximum.

- HR can increase to 180/min
- SV increases from 70 to 140mL
- diastolic filling time decreases.
- Q can increase to over 20-30L/min
- myocardial O2 consumption 250 to 3500mL/min
- once exercise starts, blood flow is maintained by local mechanisms (arteriolar vasodilation) - see C4
- decreased pH, increased 2,3 DPG and increased T shifts O2 dissociation to the right -> favoring O2 off loading

- VR increases from 5 to 20L/min from

(1) decreases resistance in arterioles
(2) increase activity of thoracic & muscular pump
(3) sympathetically mediated diversion of blood from splanchnic & renal areas to central blood volume, &
(4) norad mediated vasoconstriction.

- all these increase MSFP
- decrease in SVR from metabolic vasodilation -> allows heart to pump more efficiently
- the baroreceptor reflex helps to buffer changes in BP

Isometric exercise

- muscle contraction continual -> compresses vessels
- tonically contracted muscles with increased SVR (to overcome restriction to flow)
- vasodilator substances build up in muscles -> stimulate sensory nerves and vasomotor centre to increase BP
- increase in HR

Isotonic exercise

- usual exercise where there is changing muscle length.
- contractions cause increase in HR
- also increase in SV & net fall in SVR due to vasodilation of exercising muscles.
Post exercise:

- when exercise stop -> abrupt fall in HR & Q
- SVR remains low while body is removing vasoactive metabolites.
- repayment of O2 debt from anaerobic metabolism needed to help maintain O2 levels.
- lactate + O2 -> CO2 + H2O
- restoration of O2 stores

  - myoglobin
  - FRC
  - blood (Hb + dissolved)
- + ATP & phosphocreatine levels.

Cardiac failure

= primary abnormality of the heart which leads to an inability to pump blood at a rate required to meet metabolic need.

**Systolic failure** = impaired contractility -> reduced SV -> inadequate ventricular emptying -> cardiac dilatation -> elevated ventricular diastolic pressure.

Causes:

- cardiomyopathy
- hypertension (chronic, excessive work)
- valve disease
- IHD (loss of contracting cells)

**Diastolic failure** = increased resistance to ventricular inflow & decreased ventricular diastolic capacity.

Causes:

- constrictive pericarditis
- restrictive cardiomyopathy
- hypertensive cardiomyopathy
- hypertrophic cardiomyopathy

- can be diagnosed by 2D echo with doppler mitral inflow analysis.
- shows normal EF & abnormal LV filling -> pulmonary congestion.

Compensation mechanisms:

**Short term**

- myocardial contractility falls
- Q & MAP fall
- end-diastolic & atrial pressure increase

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- Q curve flattens but VR curve doesn’t shift.
- baroreceptors sense low MAP
- increase in sympathetic tone opposing atrial stretch reflexes.
- tachycardia, increase in contractility & vasoconstriction.
- steepens Q curve.
- ANP & BNP release -> decrease norad, increase natriuresis, increase vasodilatation.

Long term

- diminished blood flow and vasoconstriction.
- decreased GFR -> Na+ & H2O retention
- increased ATII -> renin secretion -> ALD
- increased ADH release via baroreceptors
- increased fluid retention -> increased capillary hydrostatic pressure -> increase ultrafiltration -> interstitial oedema.
- increased circulating blood volume
- endothelin 1 release (vasoconstrictor & hypertrophy of myocardium)

Mechanisms of Treatment

Digoxin - inhibit sarcolemma Na-K ATPase so Na gradient falls -> Na-Ca2+ counter transport decreases -> allows Ca2+ to build up in cells -> increased contractility.

Diuretics - reduce fluid retention

Vasodilators - decrease afterload

Venodilators - increase venous capacitance

B blockers - help with myocardial remodelling

ACE Inhibitors - help with myocardial remodelling

Ca2+ sensitisers - ie levosimenden increase contractility without increasing intracellular Ca2+.

IPPV

Background:

- mechanical ventilation by intermittent generation of +ve pressure sufficient to overcome lung elastic & airways resistance.
- provided via ETT or tracheostomy or proseal.
- if inspiration fast - time constants determine regional distribution.
- if inspiration slow - regional compliance determines distribution.

Cardiovascular response to IPPV:
(1) Decrease in sympathetic tone

- IPPV in the critically unwell relieves WOB, hypoxia, hypercapnia & acidaemia -> decreases sympathetic tone -> decrease Q, VR and MAP.
- must fluid load.

(2) Decreased Preload

- initially blood moved from pulmonary circulation to heart -> increases preload.
- subsequently preload decreases because of decreased VR & diastolic filling due to external splinting & septal shift.
- end result = decrease in Q & VR.

(3) Decreased afterload

- increased pressure gradient from thorax to abdomen & intrathoracic - extrathoracic aorta.
- decreased in transmural pressure gradient
- these results in the ventricle having to generate less pressure to achieve ejection.

(4) No effect on contractility

(5) rebound increase in ANP when ceased -> polyuria.

(6) increased O2 flux

(7) decreased MAP from inhibition of cardiovascular regulatory centres.

Anaesthesia

I think this topic is a bit huge!

PEEP

- producers the same effects as IPPV but quantitatively greater.
- positive end-expiratory pressure delivered to a ventilated patient.
- 5-15cmH2O

PEEP 5cmH2O

- improves arterial oxygenation with minimal affect on Q.

PEEP 10

- increases FRC by 500mL in patient with a compliance of 50mL/cmH2O
- raises range of TV above closing capacity
- opens closed alveoli
- reduces airway resistance
- improves ventilation of dependent, overperfused parts of lung.
- may reduce VR to RV.

**PEEP 15**

- decreases VR by constantly increased intrathoracic pressure obstructing filling of RA.
- increased pulmonary vascular resistance -> raises RV afterload -> transmitted to heart -> leftward shift in interventricular septum -> impairs diastolic filling + decreased endocardial blood flow ? impairing contractility.

**Valsalva manoeuvre**

- inspiration to TLC then a forced expiration against a closed airway.
- used to test the integrity of the autonomic reflexes.

**See Graph - Faunce page 91**

**Phase I**

- increase in MAP initially as increased intrathoracic pressure squeezes blood from intrapulmonary vessels to increase LVEDV -> increased SV by Starlings law.
- decrease in HR for a few beats as baroreceptors sense increase in MAP from increased SV.

**Phase II**

- MAP falls as high intrathoracic pressure compresses veins -> decreased SV -> decreased Q.
- baroreceptor sense this -> increased in HR

**Phase III**

- airway opened -> intrathoracic pressure returns to normal.
- blood pools in dilated pulmonary vessels -> decreased VR -> MAP dips for a few beats.
- HR peaks

**Phase IV**

- peripheral vessels remain vasoconstricted.
- VR & MAP rise and overshoots.
- baroreceptor sense -> inhibit vasomotor centre -> decreased sympathetic tone + increased parasympathetic tone -> HR falls
- MAP & HR settle to prior level.

**Clinical uses of Valsalva:**

- reversion of SVT
- testing autonomic function

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- assessment of murmurs
  HOCUM & MVP - increase in phase II
  AS & MR - decrease in phase II
- clear eustacian tube in ear infections
- equalising intracranial pressure during changes in altitude.
- check whether patient can equalise pressure before hyperbaric O2 treatment.
- fluid assessment - if arterial line systolic pressure variation < 7mmHg throughout valsalva = optimal fluid volume status.

(e) To explain the cardiovascular changes accompanying the process of ageing.

Ageing = the normal physiological process of degeneration which begins from 30yr and become symptomatic at 60 and ends in death.

Mechanisms:

- apoptosis of hypothalamic pacemaker cells.
- DNA repair destabilization -> progressive telomere loss.
- autoantigens to cytoplasm or nuclei
- free radical damage
- collagen deposition
- failure of the Na-K ATPase pump

Cardiovascular changes with age:

- increase in ventricular wall thickness
- myocardial fibrosis
- valvular calcification
- loss of elasticity in peripheral circulation -> increased SVR -> myocardial hypertrophy
- coronary atherosclerosis
- decrease in ventricular compliance
- loss of Beta receptor responsiveness

Results:

- 40% decrease in Q due to decrease in SV & HR.
- during stress Q determined more by Frank-Starling mechanism rather than nervous input.