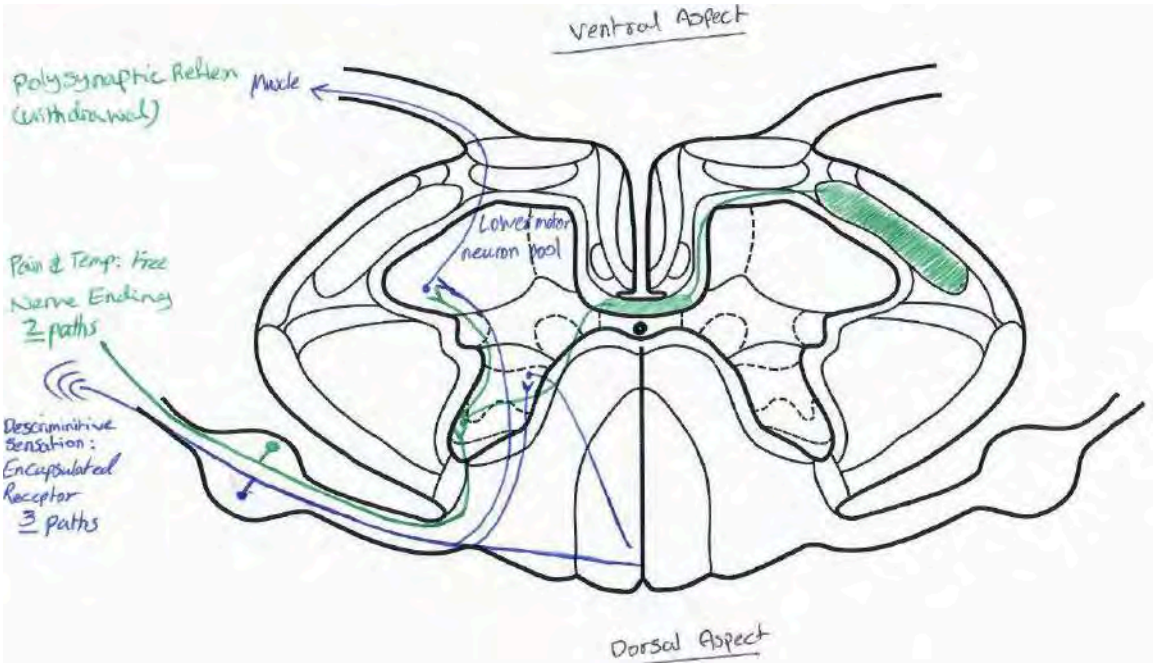
<table>
<tr><th colspan="4">Summary of Course Assessment</th></tr>
<tr><th></th><th>Weighting/format</th><th>Assessment information</th><th></th></tr>
<tr><td rowspan="2">Practical 20%</td><td>End-of-lab quizzes 20%</td><td colspan="2">Test at the end of each lab.4% each lab, all 5 lab marks count</td></tr>
<tr><td colspan="3">Must achieve >10% to pass the course as a whole</td></tr>
<tr><td rowspan="4">Theory 80%</td><td>Test 1 (90min) 20%
~100 secs/Q
46 MCQs only</td><td><u>First 17L:</u>
Nervous system (4), Cardiovascular system (7), Respiratory system (4)
<u>First 2 labs:</u>
Human brain, Sheep heart dissection</td><td>2 MCQs per lecture (i.e. 34 questions assessing the first 17 lectures)
6 MCQs per lab topic (i.e. 12 questions assessing the first 2 lab topics)</td></tr>
<tr><td>Test 2 (90min) 20%
~100 secs/Q
52 MCQs only</td><td><u>Second 17L (L18-L33):</u>
Musculoskeletal system (5), Renal system (3 + 1 online), Autonomic and endocrine (2), Reproductive systems (4), First 2 digestive system lectures (2)
<u>Final 3 labs:</u>
Respiratory, Musculoskeletal and Reproductive systems</td><td>2 MCQs per lecture (i.e. 34 questions assessing the 17 lectures)
6 MCQs per lab topic (i.e. 18 questions assessing the final 3 lab topics)</td></tr>
<tr><td>Final Exam (2h)
30%</td><td><u>All lectures</u>
<u>All labs</u>

Fill in the blank (word/phrase/number)
Labeling Diagrams
Describe (sentence) /Explain(paragraph)
Drawings and flow charts
T/F + Reason</td><td><table><tr><th>Topic</th><th>Sessions</th><th>Marks</th></tr><tr><td>Nervous</td><td>4</td><td>24</td></tr><tr><td>Autonomic & Endocrine</td><td>2</td><td>12</td></tr><tr><td>CVS structure</td><td>3</td><td>18</td></tr><tr><td>CVS function</td><td>4</td><td>24</td></tr><tr><td>Reproduction - Female</td><td>2</td><td>12</td></tr><tr><td>Reproduction - Male</td><td>2</td><td>12</td></tr><tr><td>Respiratory structure</td><td>2</td><td>12</td></tr><tr><td>Respiratory function</td><td>4</td><td>24</td></tr><tr><td>Renal structure & function</td><td>1 online + 3</td><td>24</td></tr><tr><td>Musculoskeletal</td><td>5</td><td>30</td></tr><tr><td>Digestive</td><td>3</td><td>24</td></tr><tr><td>Integrative</td><td>Labs</td><td>12</td></tr><tr><td>Total</td><td>34+5</td><td>228</td></tr></table></td></tr>
<tr><td>Online activities 10%</td><td>Weekly online quizzes</td><td></td></tr>
<tr><td colspan="3">Must achieve >40% to pass the course as a whole</td></tr>
</table>

Comparing Polysynaptic (/withdrawal) Reflex vs Monosynaptic/Myotactic Reflex

Fastest? Monosynaptic/Myotactic Reflexes. Reasons:

1. It's just 1 single synapse
2. And heavily myelinated



Disruption of sensory pathways

Lesion in the brain/brain stem → ***Associative Sensory Loss***

Lesion on the spinal cord → ***Dessociative Sensory Loss (Brow-Sequad Syndrome)***

Study from course guide

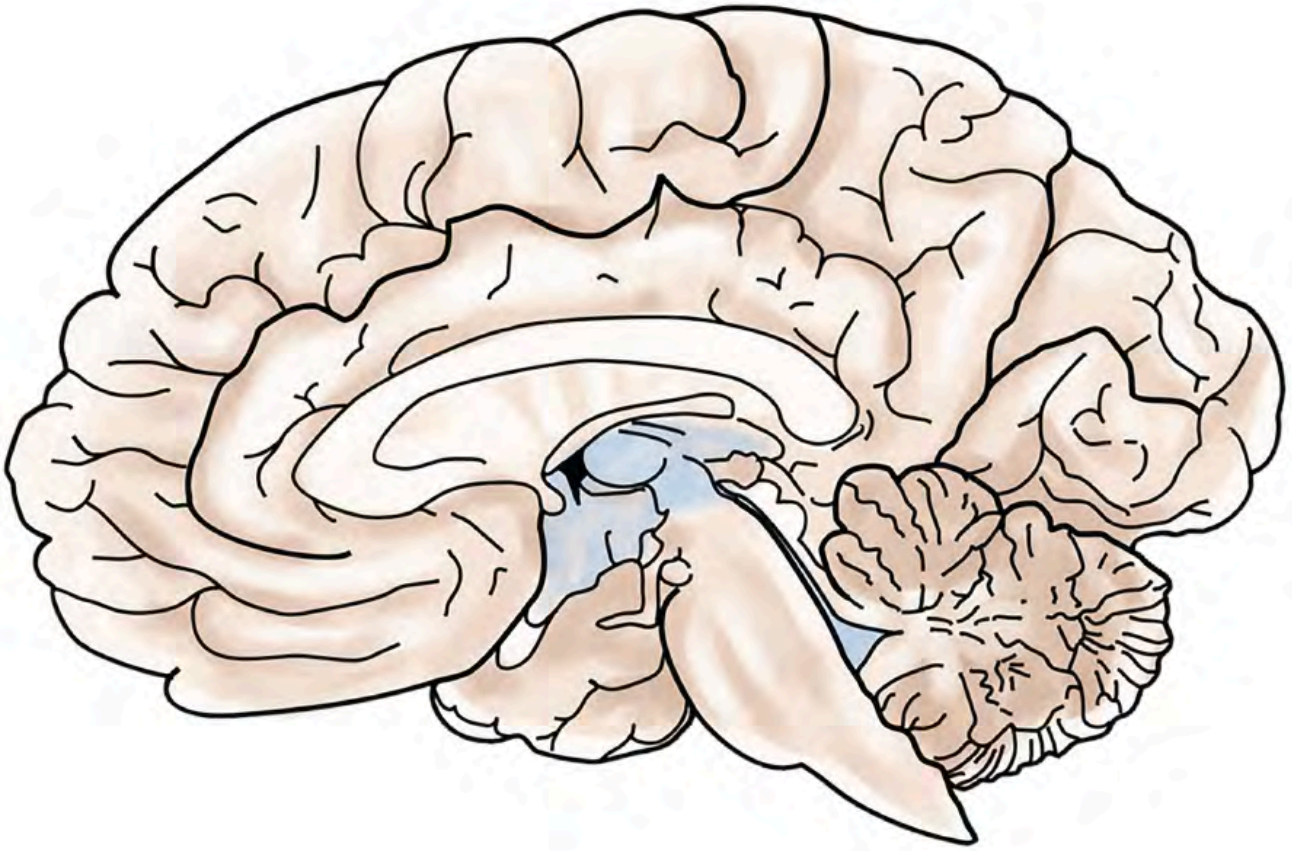
[2015 Exam] Fill in the blanks to complete the paragraph below.

When the pain and temperature pathway and the discriminative touch and pressure pathway are both affected by a lesion on the right side of the spinal cord, we refer to the pattern of sensory loss as being (a/an) dissociative ✔ sensory loss, because touch and pressure are lost on the right ✔ side of the body and pain and temperature are lost on the left ✔ side of the body. When both these pathways are affected on the right side of the brain stem, touch and pressure are lost on the left ✔ side of the body and pain and temperature are lost on the left ✔ side of the body, resulting in (a/an) associative ✔ sensory loss.

Overview

	Discriminatory	Non-Discriminatory
Sensations	Touch and Pressure	Pain and Temperature
Conduction Velocity	50 m/s	1 m/s
Receptor Class	Encapsulated Receptor	Free nerve ending
Enters spinal cord through	Dorsal Root	Dorsal Root
Primary Neuron synapses in	Cuneate/Gracile Nuclei	Dorsal Grey Horn
Secondary Neuron projects through	Medial Lemniscus	Spinothalamic tract
Secondary Neuron Synapses in	Ventro-posterior thalamus	Ventro-posterior thalamus
Tertiary Neuron projects through	Internal Capsule	Internal Capsule
Tertiary Neuron Synapses in	Primary Somatosensory Cortex	Primary Somatosensory Cortex

Identify: Orbital gyrus, Fornix, Lamina terminalis, Optic nerve, Oculomotor nerve, Pituitary gland, Pineal gland, Mamillary body, Interventricular foramen



Fetal heart structures and differences

Foramen Ovale [fetal]/Fossa Ovalis [adult]

In fetal life

Lungs are not fully developed yet → not ventilated — filled with amniotic fluid → very little oxygen in the alveoli (alveolar pO_2 is extremely low) → hypoxia

Pulmonary arterioles sense hypoxia → vasoconstriction

How?

→ Hypoxia → K^+ channels close → depolarization → Ca^{2+} influx → smooth muscle contraction → hypoxic vasoconstriction

→ due to vasoconstriction, pressure and vascular resistance in the pulmonary circuit is quite high

This means that in foetal circulation, unlike in the adult, the pressure is higher in the right side of the heart.

Blood moves from RA → LA through foramen ovale

After birth

Lungs expand with air → alveoli fill with O2 → alveolar PO2 rises

Oxygen acts on pulmonary arteriole smooth muscle → Inhibits hypoxic vasoconstriction → Triggers vasodilation

Vasodilation → pulmonary arterioles open up → pulmonary vascular pressure/resistance falls dramatically.

Blood now flows freely into the lungs → much more blood returns to the left atrium via pulmonary veins.

Left atrial pressure > Right atrial pressure

This pressure gradient presses the septum primum (flap valve)against the interatrial septum, functionally sealing the foramen ovale → Fossa Ovalis

Ductus Arteriosus [feta])/Ligamentum Arteriosum [adult]

During fetal life, a temporary blood vessel, called the ductus arteriosus, shunts blood from the pulmonary trunk into the aorta. Hence, only a small amount of blood enters the nonfunctioning fetal lungs. The ductus arteriosus normally closes shortly after birth, leaving a remnant known as the ligamentum arteriosum, which connects the arch of the aorta and pulmonary trunk

Path of blood in placenta and fetal circulation

Placenta → Umbilical vein --> oxygenated blood through IVC to RA -->

Ox blood from RA goes to:

1. RV -->
    1.1. Pul. trunk --> Ductus arteriosus --> descending Aorta --> systemic circulation --> umbilical artery --> placenta
    1.2. Pul arteries to feed lungs --> deox blood into pul. vein --> comes back into LA

Ox blood from RA goes to:

2. Foramen Ovale --> LA --> LV --> Aorta --> systemic circulation --> umbilical artery --> placenta

Note: there's a little mixing of ox and deox blood from the pul. arteries into the systemic blood but it's minor.

Patent Ductus Arteriosus (PDA)

PDA can result in heart failure.

If the ductus remains open ("patent") after birth the result would be a large shunt of blood from the aorta to the pulmonary artery. In that case the volume of blood reaching the lungs would be larger than normal.

If it stays open (PDA), high-pressure blood from the aorta flows into pulmonary circulation → pulmonary overcirculation

→ ↑ lung blood flow → higher BP in lung → pul. Edema → less O_2 diffusion

Because extra blood cycles through lungs → higher venous return

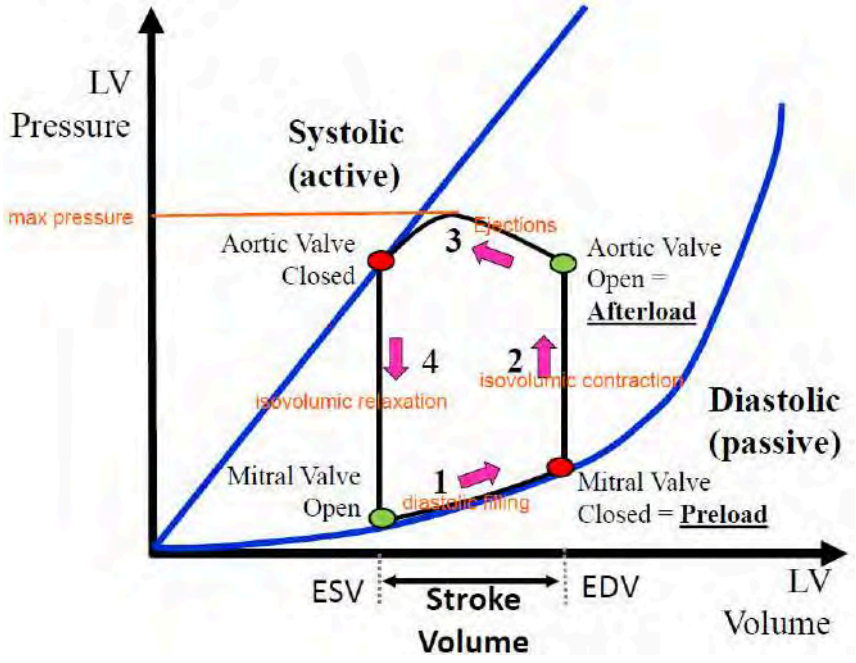
→ heart gets overworked (The LV, in particular, works harder to pump the extra blood.). → heart failure

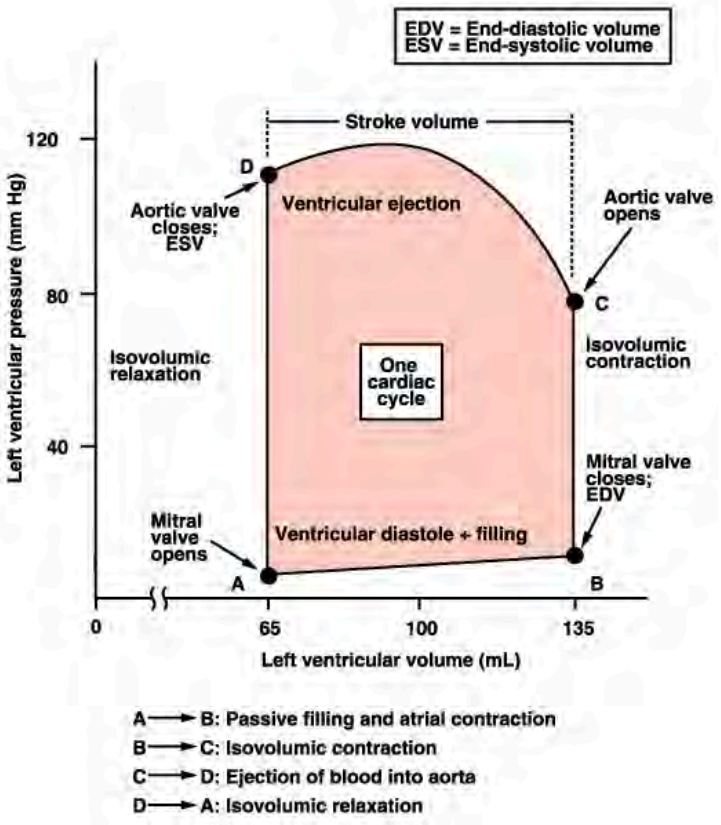
Ventricular Pressure-Volume Relationship: ***Cardiac Cycle – Pressure Volume Loop***

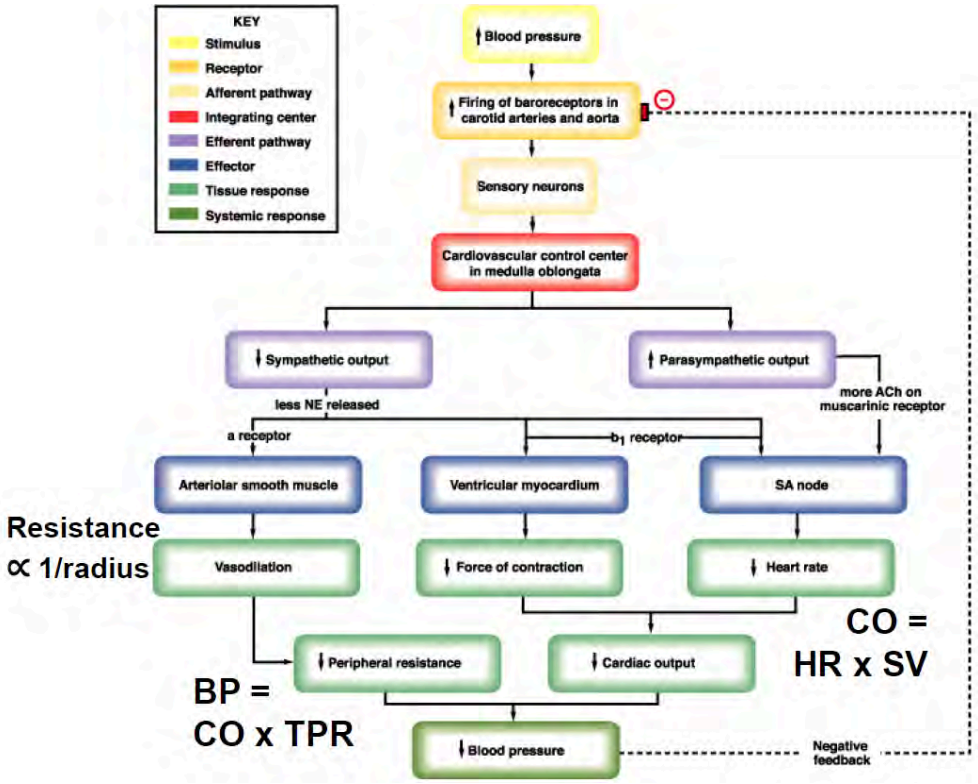
Limits of ventricular performance:

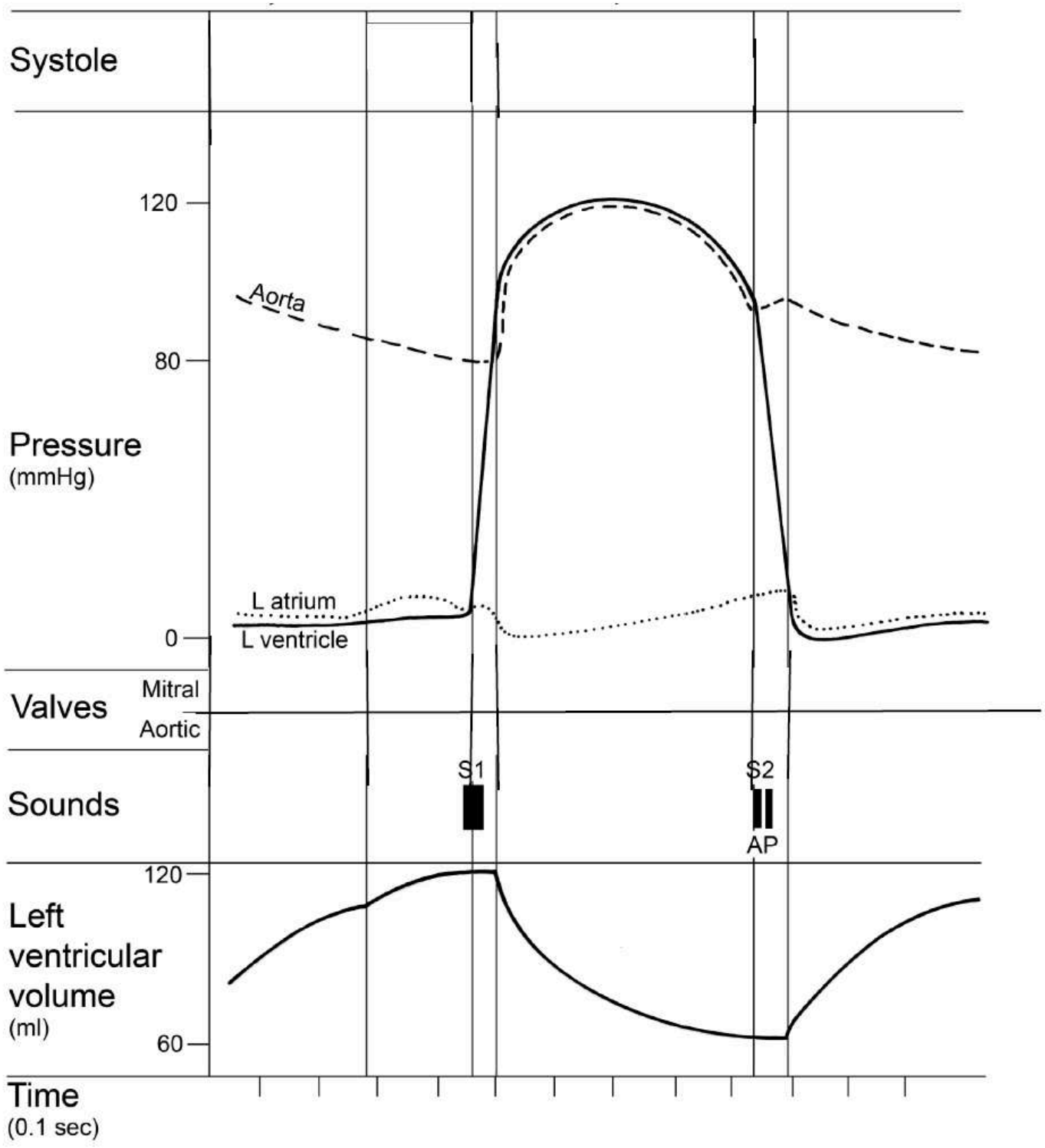
- Passive relationship of the LV wall – Diastolic
  During diastolic filling, there's a small but steady increase in the LV pressure as blood enters. Then as you start to get into the connective tissue in the ventricle, it gets harder (muscle fibers stretch easily at first, but as LV gets filled more, non-muscular components like collagen, start to resist further stretch) for blood to enter as P is increasing.

- Active relationship of the LV wall – Systolic
  During systole when the heart is contracting, the pressure that can be generated by the LV, increases with increased preload/V → Starling's law

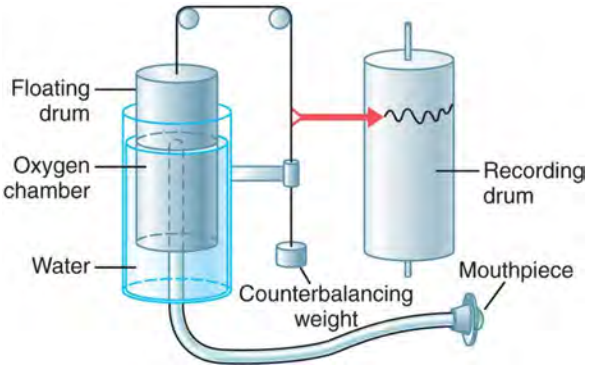





KEY
Stimulus
Receptor
Afferent pathway
Integrating center
Efferent pathway
Effector
Tissue response
Systemic response
↑ Blood pressure
↑ Firing of baroreceptors in carotid arteries and aorta
Sensory neurons
Cardiovascular control center in medulla oblongata
↑ Sympathetic output
↑ Parasympathetic output
less NE released
more ACh on muscarinic receptor
a receptor
b1 receptor
Arteriolar smooth muscle
Ventricular myocardium
SA node
Resistance ∝ 1/radius
Vasodilation
↓ Force of contraction
↓ Heart rate
CO = HR x SV
↑ Peripheral resistance
↑ Cardiac output
BP = CO x TPR
↑ Blood pressure
Negative feedback


Systole
120
80
0
Pressure
(mmHg)
Aorta
L atrium
L ventricle
Valves
Mitral
Aortic
Sounds
S1
S2
AP
120
Left
ventricular
volume
(ml)
60
Time
(0.1 sec)

Spirometry: measuring lung volume with a spirometer

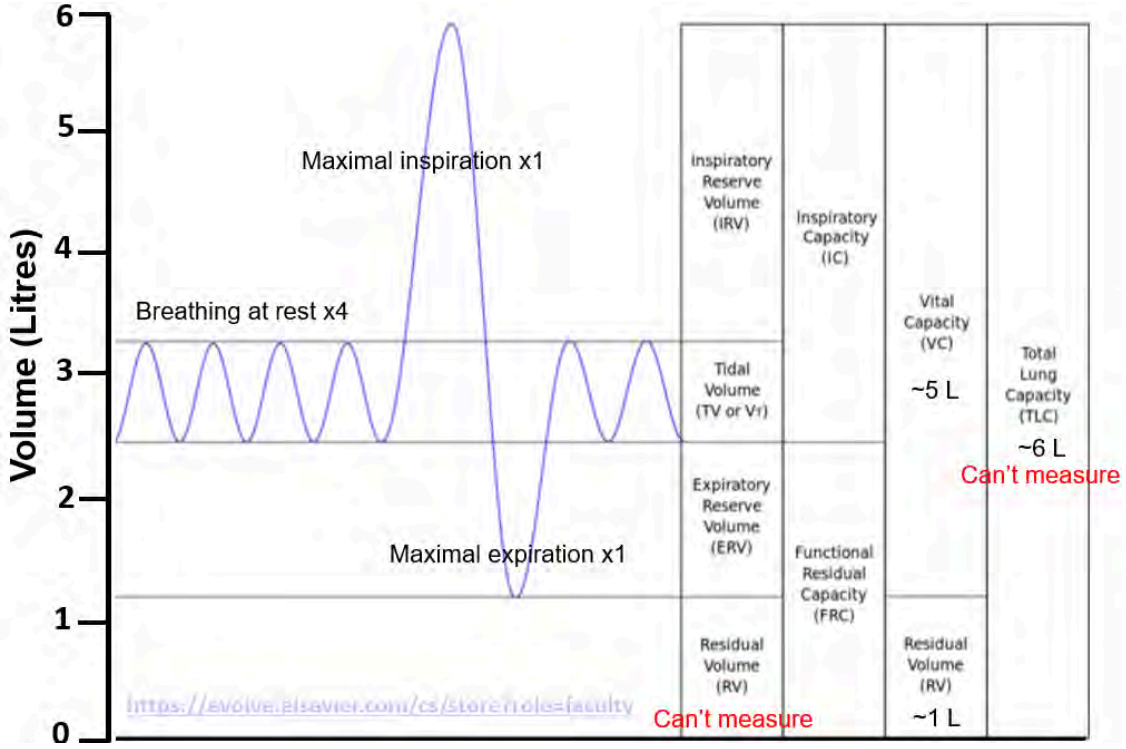


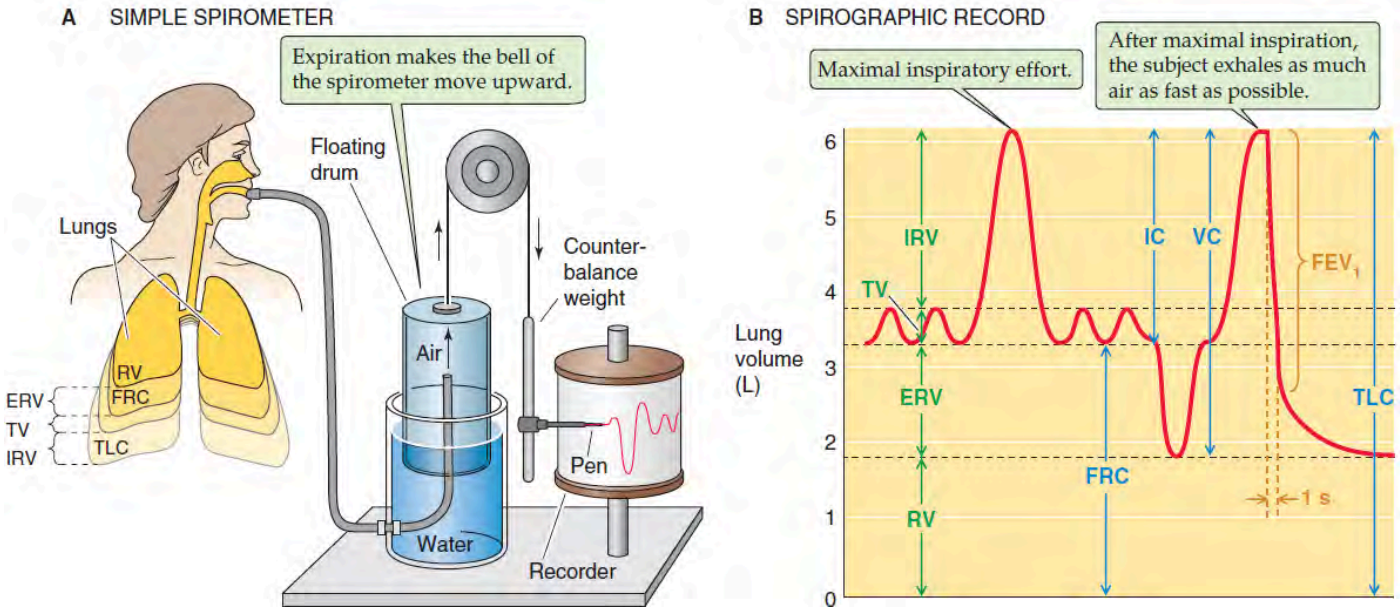
Respiratory Volumes Measured:

- ***Tidal breath*** (V°_{T}): volume of 1 breath at rest (e.g. 0.5 L)
- ***Respiratory frequency (f)***: e.g. 6 breaths in a minute
- ***Minute ventilation*** $V_E = (V_T \times f)$: 12 x 0.5 = 6.0 L/min
- ***Inspiratory reserve volume (IRV)***
- ***Expiratory reserve volume (ERV)***

Residual Volume (RV): the amount of air you cannot breath out – Why?
After inflation, bronchioles start to collapse, trapping air in the alveoli (i.e. part of the lower airway gets sealed off due to the collapsing of the upper airways).
Because we cannot measure RV, we are not able to measure ***Total Lung Capacity (TLC)*** through this approach.

**Volume vs capacity: volume is measured – capacity is calculated



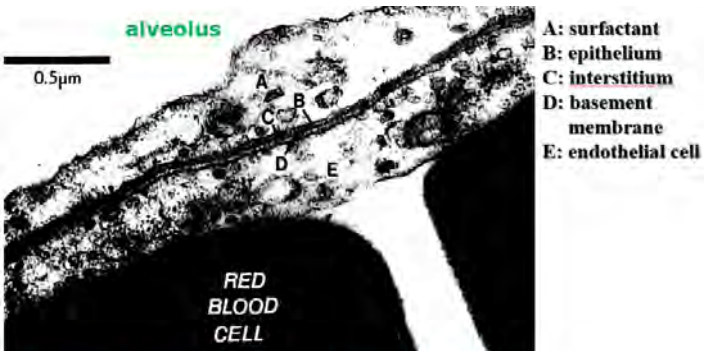


Pulmonary oedema

- **Caused by left heart failure**
  The LV cannot eject blood efficiently. This leads to blood "backing up" into the left atrium and pulmonary veins and capillaries.
  Increased pressure in capillaries - edema
- **Systemic hypoxia**
  Cannot oxygenate properly bc of the edema
- **Breathlessness – 'dyspnoea'**

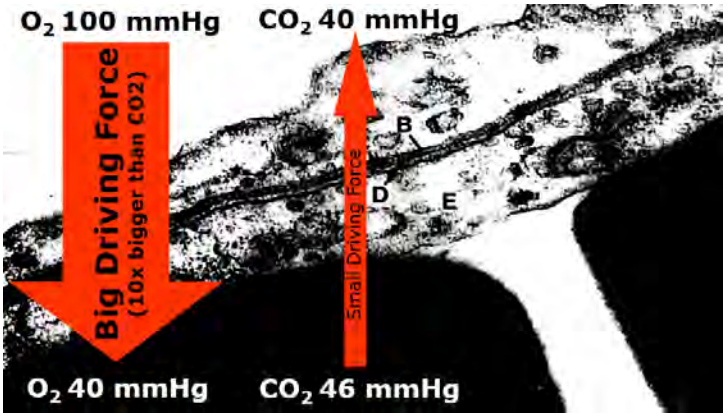
Factors Regulating Movement of Gas Across the Respiratory Surface

1. Area
   - ~300 million alveoli in the human lung
   - 0.3 mm in diameter
   - If spherical, surface area = 50 to 100 m^2 and volume = ~4 L
2. Thickness of tissue
   - Only 0.5 µm between air & blood.



3. Partial pressure differential across tissue

- CO_2 is much more diffusible than O_2, therefore a smaller partial pressure difference still lets it diffuse.



4. Solubility of gas in blood
   - Solubility more important than MWt of gas
   - CO_2 is x25 times more soluble in blood than O_2
   - Movement of both gases across the alveolar membrane are balanced. There's no overall preference for O_2/CO_2. I.e. Both gases move at the same rate.

5. Molecular weight of gas
   - CO_2 & O_2 have very comparable mw, therefore not too important.

Overall diffusion of a gas is a function of all these factors shown by the below equation:

$$\text{Diffusion of Volume of a Gas} = \frac{\text{Lung Area} \times \text{Gas Density} \times (\text{Pressure Differential})}{\text{Thickness of alveolar membrane}}$$

$$\text{Density} = \text{Solubility}/\sqrt{\text{Molec Weight}}$$

Eqn does not need to be memorised

Bone Remodelling

- **In diameter/circumference: Simultaneous Growth & Resorption**
  1. ***Appositional Growth***
  2. ***Bone Resorption***
- **In length (long bone growth)**
  1. ***Endochondral Ossification* at *Growth Plate***

Important points to remember:

1) The tissue bone is too rigid to grow by a process called interstitial growth. Interstitial growth occurs in softer tissues that can deform. This process involves the cells dividing inside the tissue, secreting more extracellular matrix and growing the tissue from within. Because bone is designed to resist deformation it can only grow by adding new bone onto an existing surface (= appositional growth).

Usually bone, but can be cartilage/other tissue

2) The two processes, appositional growth and bone resorption, occur through-out your skeletal system often completely independent of each other. This is known as bone remodelling. The example on the previous page showing a long bone growing in diameter just happens to show the two process occurring at the same time.

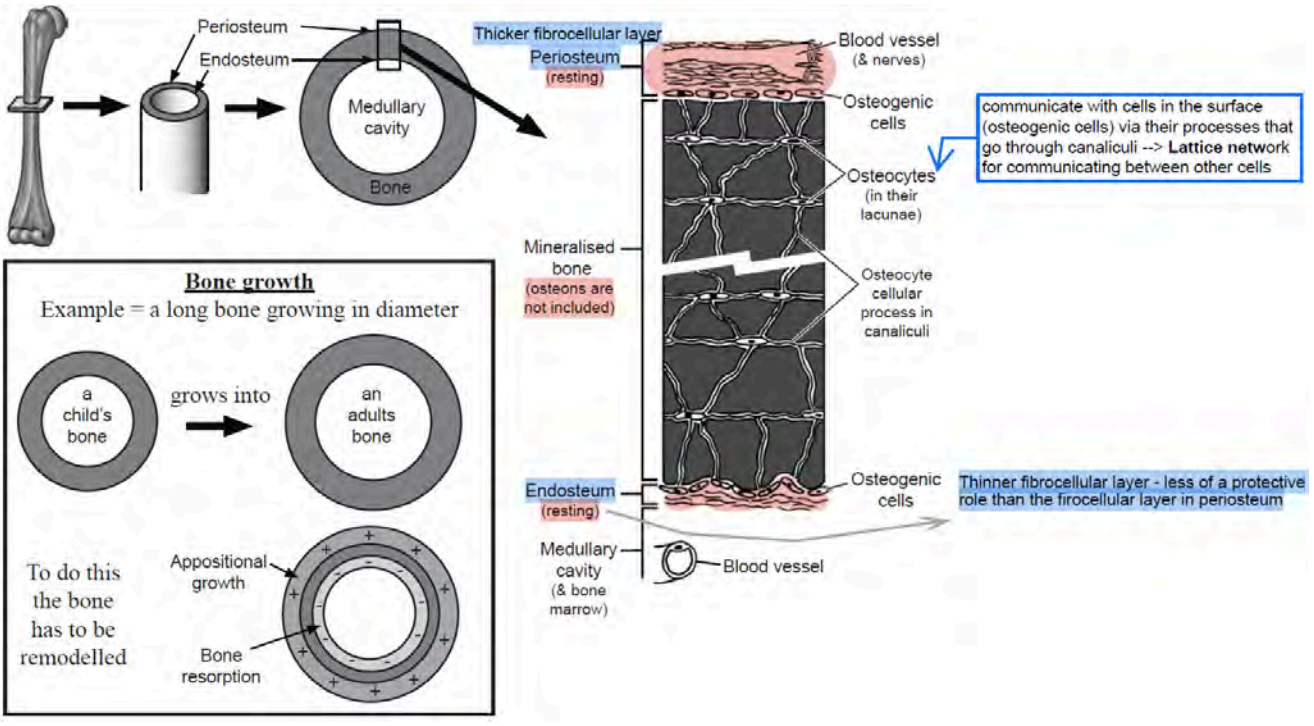
The ratio of these 2 events changes throughout life:

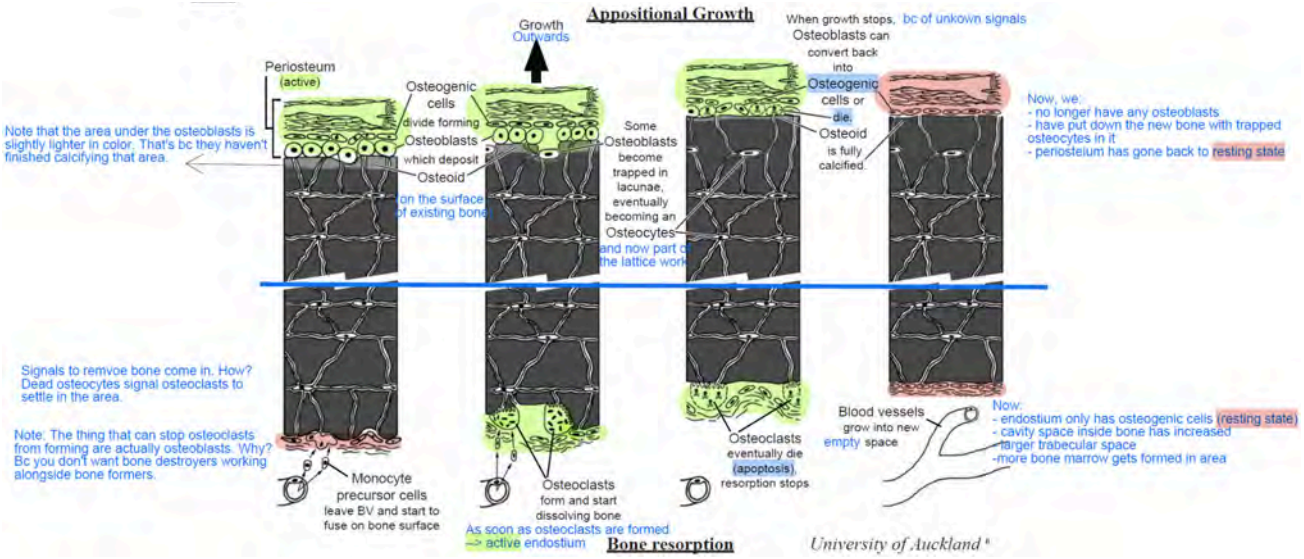
birth - early 20s --> appo. growth > bone reso.

20s - mid 30s --> rates are equal
remodel 10% of skeleton by default /year
if you do weight training --> more appo. growth in space/out of gravity --> bone reso.

mid 30s - end --> bone reso. > appo. growth
elderly lose bone density --> soft/brittle bones

3) Long bones grow in length by a different process called Endochondral ossification. Some of the bones in your skeleton are formed by endochondral ossification. For this course you will not be expected to learn about endochondral ossification in any more detail than what was discussed.

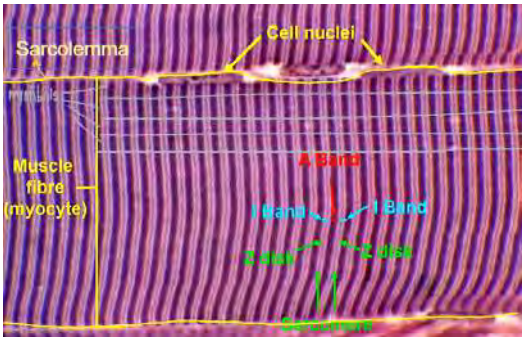




TOPIC 2 : MUSCLE

Biosci 107_Module 7

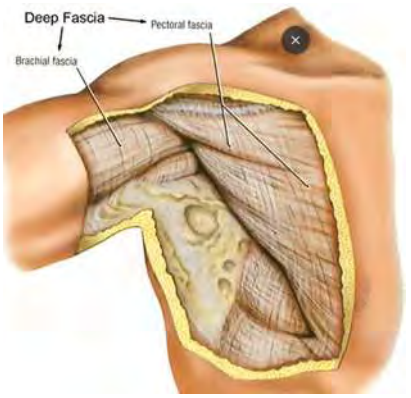
Cardiac & skeletal muscle: ***Striated*** ⇒ All A/I bands of myofibrils perfectly line up



Deep Fascia

Collagenous, sheet-like material covering muscles (not part of muscle).
Diagram shows a cut through skin + a supporting fatty layer ***(Subcutaneous Tissue***/***Supporting Fascia***). E.g.

- ***Pectoral Fascia***
- ***Brachial Fascia***



Venous Return

When muscles shorten, their bellies expand outward. The interosseous membrane they are lying against does not expand (rigid collagen + bone). Blood vessels & veins are also lying along these. Expanding muscles press/compress veins against the membrane.
This compression pushes blood forward in veins (one-way valves prevent backflow).

Compartment Syndrome

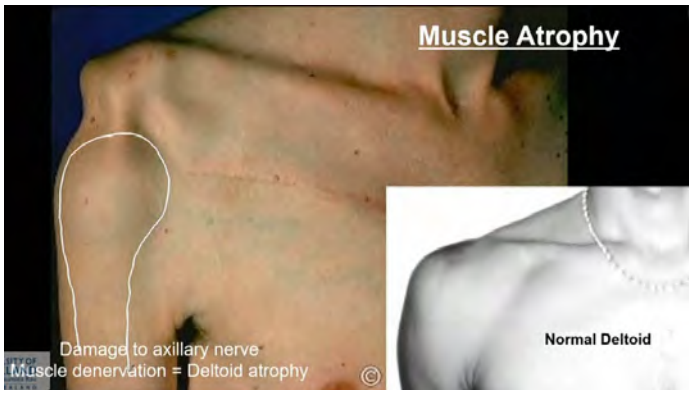
Injury (hemorrhage, infection) of a muscle in one compartment that causes fluid/blood buildup in the compartment compressing nearby soft structures e.g. veins. As arterial supply continues, the absence of outflow results in edema and swelling.
Excessive edema (compression) may:

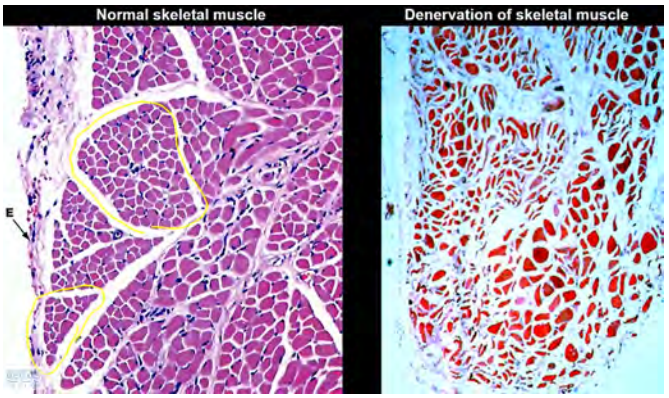
- Close off the artery
- Compress nerves (pain, numbness)

In athletes, where rate of muscle growth > rate of fascia growth:
Muscles tend to grow and get compressed within the slowly growing fascia. Invasive way of dealing with this for athletes: ***Fasciatectomy***:

- Cutting fascia to remove the compressive pressure, fascia heals together bigger?

Muscle Atrophy is easily detectable while steroid hypertrophy is undetectable

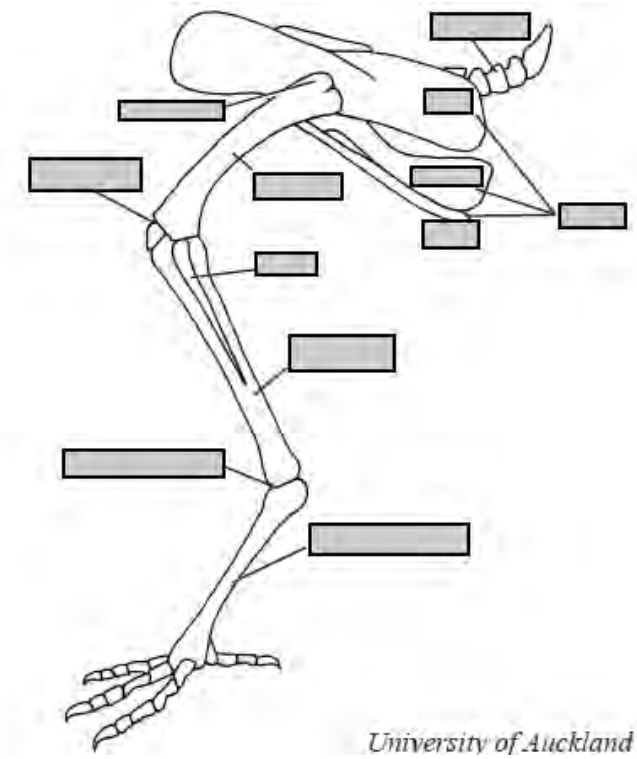


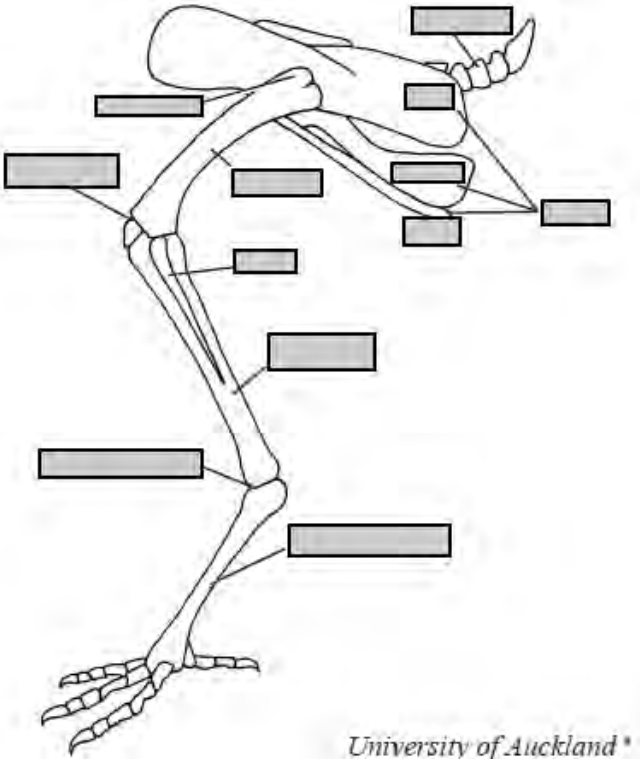


R slide:

- Some muscles have a dual nerve supply. If one is severed the other can still try to keep muscle working. We can see that some of the myocytes in the slide here are quite big bc they still have their nerve supply. Side effect of this is muscle becomes jerky.

Show the movement of the chicken hip and the knee during flexion and extension





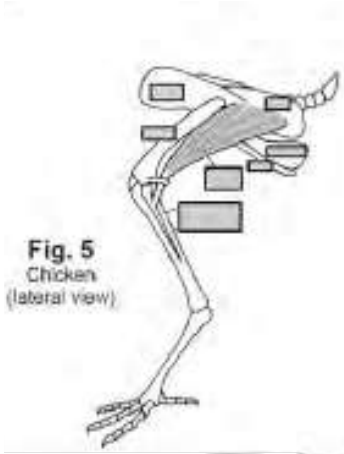


Fig. 5
Chicken
(lateral view)

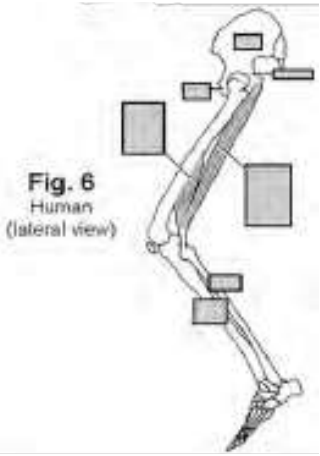


Fig. 6
Human
(lateral view)

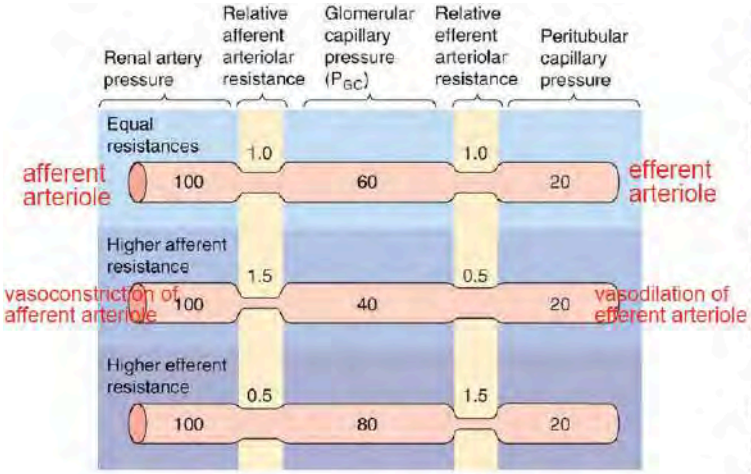
	nearer to the point of attachment of a limb or part.
	further away from the point of attachment of a limb or part.
	nearer to the mid-line of the body.
	further away from the mid-line of the body.
	towards or on the surface of the body.
	away from the surface of the body.
	a muscle's attachment to the bone that moves the least.
	a muscle's attachment to the bone which moves the most.
	to decrease a joint angle.
	to increase a joint angle.

Regulation of Glomerular blood hydrostatic pressure

- ***Vasoconstriction of the Afferent Arteriole – Vasodilation of the Efferent Arteriole***
  Higher afferent resistance means → less flow into the capillary bed → smaller blood volume in the capillary bed → smaller pressure in the capillary bed.
- ***Vasodilation of the Afferent Arteriole – Vasoconstriction of the Efferent Arteriole***
  Higher efferent resistance causes higher volume & pressure in the capillary bed.

This system is used to keep the pressure within the glomerular capillary bed constant, regardless of the incoming pressure.
Changes in the pressure of glomerular capillaries:

- Does not affect pressure elsewhere/systemic pressure as this circuit is <u>parallel</u> to the main one and blood is able to reach all other destinations through multiple other paths.
- Affects the <u>rate of glomerular filtration</u> by changing GBHP.

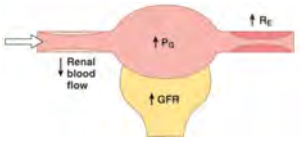


The only capillary bed that has arterioles before and after to it. Allows for tight regulation of pressure gradients to maintain near constant glomerular filtration rate.

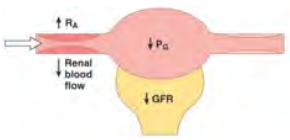
Changing resistance in afferent and efferent arterioles has virtually no effect on systemic pressure (parallel flow).

BUT the potential to have a huge effect of glomerular pressure and thus glomerular filtration rate

An increase in glomerular pressure (i.e. higher GBHP) results in increase in Net Filtration Pressure (NFP), resulting in higher glomerular filtration (↑ GFR). The opposite is true for a pressure drop.

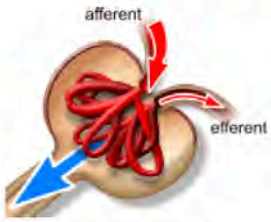


Decreases in pressure can be buffered by vasoconstriction of efferent arteriole



Increases in arterial pressure can be buffered by vasoconstriction of afferent arteriole

Regulation of Glomerular filtration



- Autoregulation: myogenic autoregulation or tubuloglomerular feedback i.e. if you strech a muscle, in tends to constrict in response to that
- Neural: Increased sympathetic nerve activity leads to vasoconstriction of particularly afferent arterioles. e.g. urine production drops during exercise
- Hormonal:
  - Angiotension II via vasoconstriction of afferent and (predominantly) efferent arterioles AT II is usually released when BP is low => helps maintain GFR
  - Atrial Natriuretic Peptide via relaxation of mesangial cells, increasing surface area available for filtration
    Atria stretch as a result of BP overload (indicating high blood volume). Atrial Natriuretic Peptides are released a result of atrial strech to increase GFR (to bring down blood volume). Acts by increasing Na in urine.

i.e.

- Anything that alters the **Glomerular hydrostatic blood pressure** (e.g. the pressure in the afferent artery)
- Anything that alters the **surface area** available for filtration SA aka filtration coefficient

Lecture 3: Hormonal Regulation of the Kidney

Outcome: describe the antidiuretic hormone pathway + Renin angiotensins system and why and how they get triggered.
What happens if we have a change (increase OR decrease) in water, salt, or an isotonic change in the body.

Fluid dynamics

- Water (hypotonic) – rapidly equilibrates throughout ICF (⅔) and ECF (⅓), decreases osmolarity.
- Isotonic solution (/poweraid) – will remain in ECF, has no effect on plasma osmolarity.
- Hypertonic solution – will draw water out of cells – plasma/ECF volume increases, intracellular cell volume decreases.

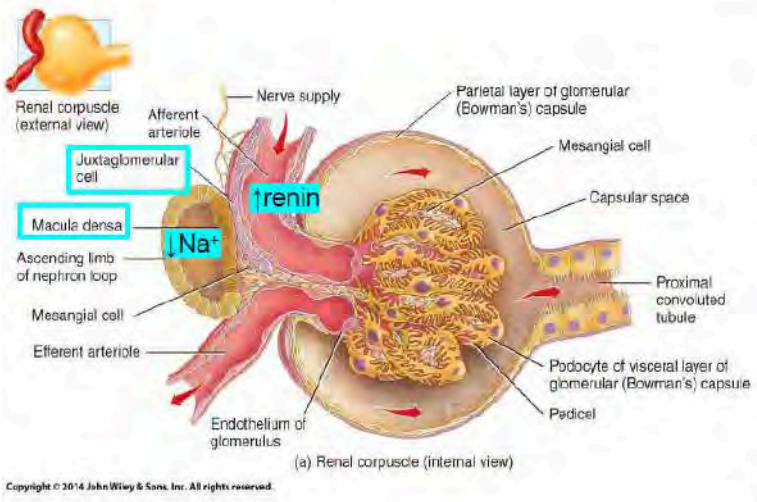
Renin-Angiotensin-Aldosterone system: triggers and actions

- Important in maintaining Na^+ balance (ADH was important in regulating water balance)
- Important in BP regulation (common to both ADH and RAA regulatory pathways)

In the case of a decrease in Na^+, during e.g.
BP decrease ⟶ Net FR drops ⟶ less glomerular filtration (↓ GFR) ⟶ less Na^+ in the distal tubule
Increased sympathetic activity ⟶ vasoconstriction of the afferent arteriole ⟶ Less Na coming in bc of glomerular filtration

Juxtaglomerular apparatus

Distal tubule right up against the afferent arteriole (image below)



Where distal tubule abuts the glomerulus

a. Macula Densa cells respond to a **decrease in NaCl** content by increasing prostaglandins
prostaglandins stimulate JG cells

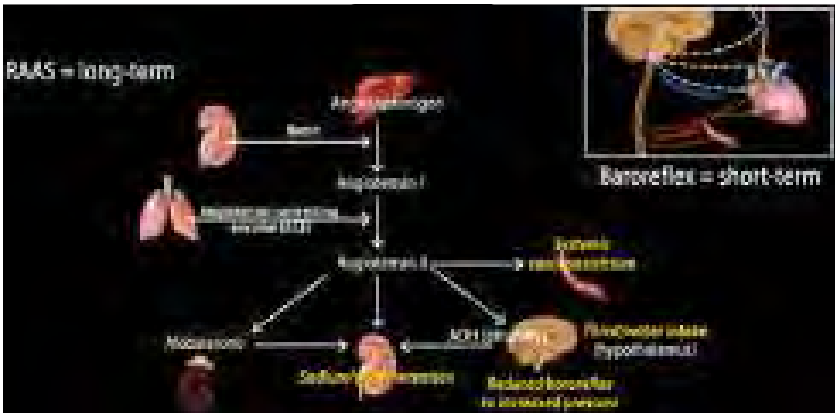
b. Juxtaglomerular (granular) cells in the afferent arteriole release RENIN

c. A **decrease in pressure** (or **increase sympathetic activity**) in afferent arteriole also acts on the juxtaglomerular cells cause the release of renin

Renin release

Triggers for renin release from granular (juxtaglomerular) cells:

- Low [NaCl] in the distal tubule (Na^+ depletion) sensed by Macula densa cells in distal tubule
- Decreased perfusion pressure (i.e. low BP/blood volume) sensed by baroreceptors of granular cells
- Increased sympathetic activity (e.g via baroreflex of the CVS)



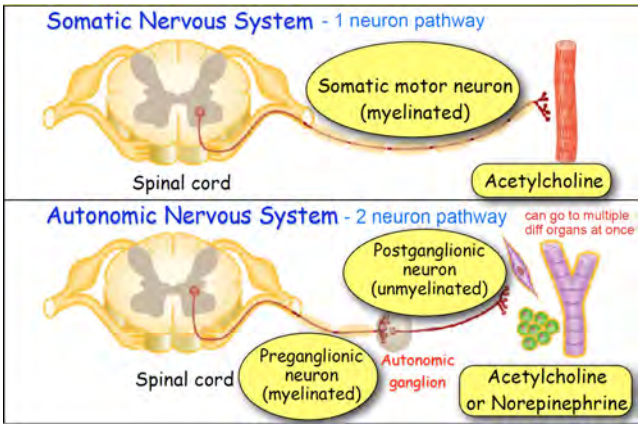
The Relaxation (rest-digest) Response

Autonomic (parasympathetic branch) NS

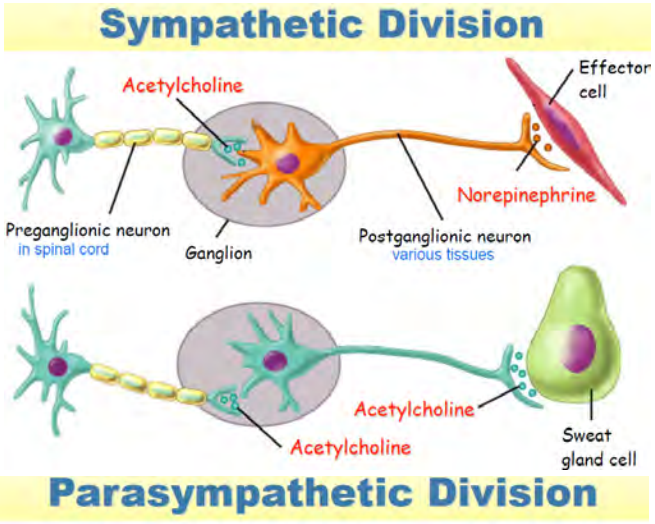
- Heart: Decrease rate and contractility
- Eyes: Contract pupils
- Mouth: Increase saliva
- Lungs: Constrict bronchi, breathe more slowly
- Skin: Dilate peripheral arterioles, hence feel warmer on skin surface
- Gut: Increase digestion

Similarities & difference between the nerves that innervate Somatics NS and the Autonomic NS

	Somatic (Curtis)	Autonomic (Booth)
Sensory input	Somatic senses Special senses	Interoceptors (internal sensing)
Control of output	Voluntary: Cerebral cortex	Involuntary: Limbic system, Hypothalamus, Brain stem, Spinal cord
Effectors	Skeletal muscle	Smooth muscle, Cardiac muscle, Glands



Motor neuron pathway	One-neuron pathway	Two-neuron pathway: Pre- and post-ganglionic
Neurotransmitters	Acetylcholine	Acetylcholine: Preganglionic axons Postganglionic axons: Parasympathetic Sympathetic to sweat glands Norepinephrine: Postganglionic axons Sympathetic to other than sweat glands



In the SNS:

Preganglionic neuron → postganglionic neuron (always Ach).

Postganglionic neuron → target tissues (always norepinephrine)

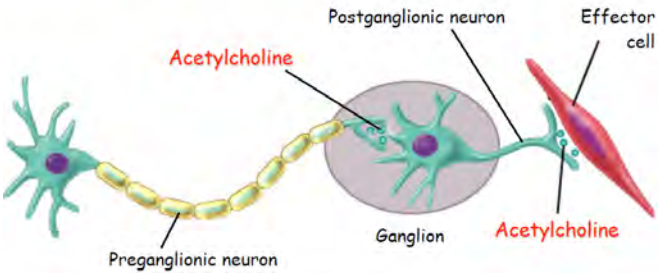
1 exception:

- Post ganglion neurons (from the sympathetic ganglion) → sweat glands (Ach)

In the PSNS:

Preganglionic neuron → postganglionic neuron (always Ach).

Postganglionic neuron → target tissues (always Ach).



Raynaud Disease

Overactivity of sympathetic system

Puberty

GnRH increase from hypothalamus signals ant. pituitary to secrete LH. An increase in plasma LH levels is the first endocrine sign of puberty (result of an increase in GnRH release). Note that GnRH does not appear (hence is not measurable) in blood hence why LH is the first hormone we are able to recognise.

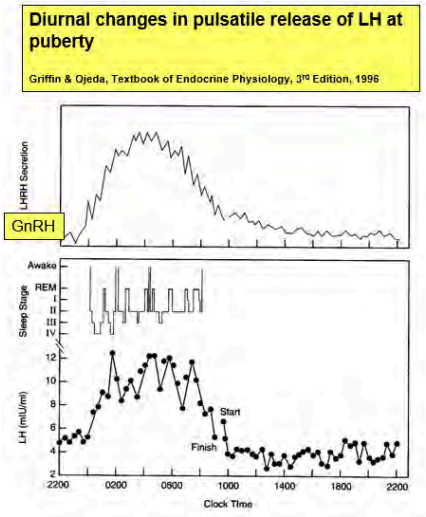
Gonadotrophin secretion (both LH & FSH) occurs in early puberty at night during sleep.

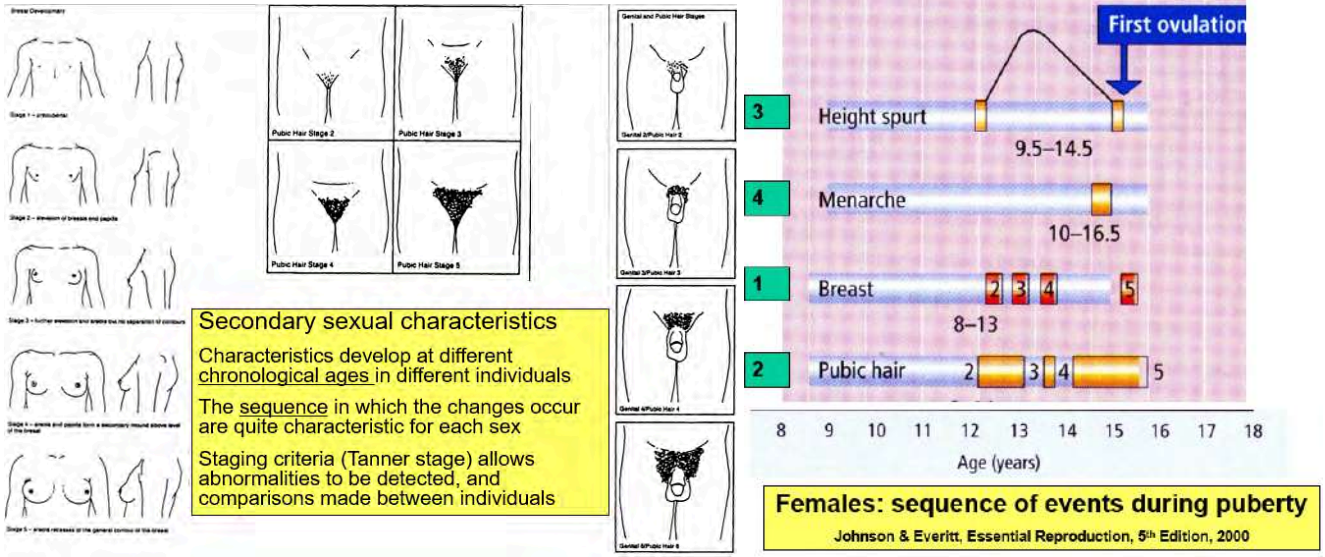
Sex steroids rise in response to the increase in plasma LH:

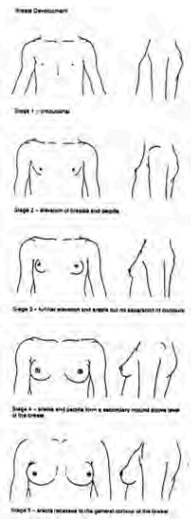
- In females: high LH causes estrogen levels to rise at nighttime
- In males: high LH causes androgen levels to rise at nighttime

In late puberty, daytime LH pulses also increase.

You don't need to know these stages in detail, but just in general, the sequence of events.







Breast development

First physical sign of secondary sexual maturation at age ~10-11

Oestrogen secretion [from developing follicles of the ovary] leads to the appearance of a ***breast bud***, followed by formation of a ***breast mound***

Ovulation, with subsequent progesterone secretion, leads to full breast development

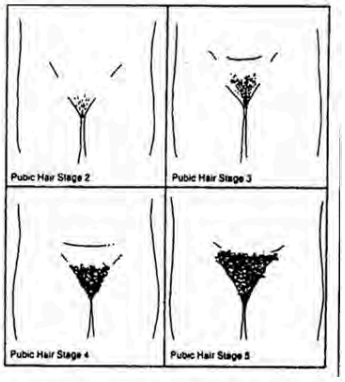
Sexual hair development

Usually within 6 months of the appearance of the breast bud at age ~10-12 (or concurrent)

Due to exposure of hair follicles to androgens

Axillary hair follows ~1 year after pubic hair

If pubic hair arrives earlier than breasts → might be an androgen disorder underlying.

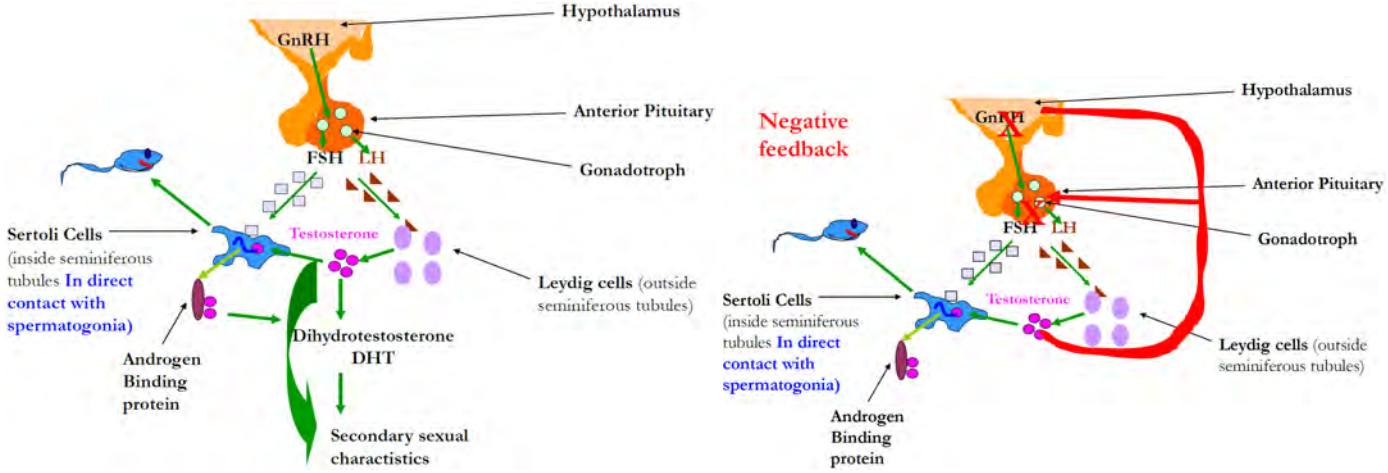


Hormonal Control of Spermatogenesis

DHT: another type of androgen and is twice as potent as T, important in producing 2' sexual chars

Note androgens are steroid hormones (i.e. lipids) hence are hydrophobic and cannot naturally diffuse into blood.
Sertoli cells produce ***Androgen Binding Protein*** which is:

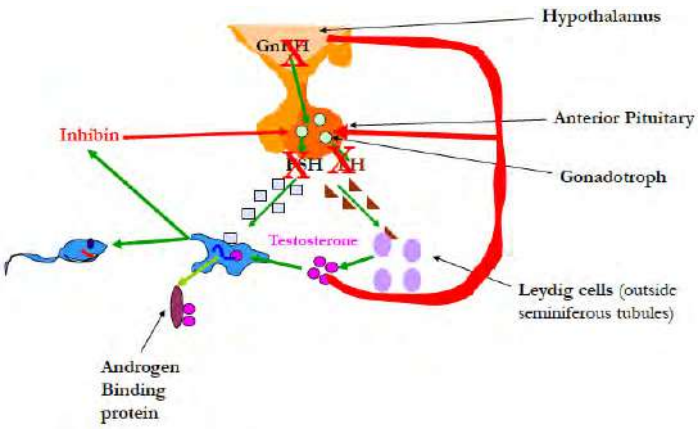
1. The main carrier for DHT and T in blood (majority of ABP has this role) for transport around body
2. Locks T in the seminiferous tubule (as we also need a supply of androgens inside the tubules) - this needs to be carried out by ABPs as steroids are able to freely diffuse across membranes
   → this promotes the final stages of spermatogenesis (note that spermatogenesis starts but is not completed in the absence of androgens (most specifically T))

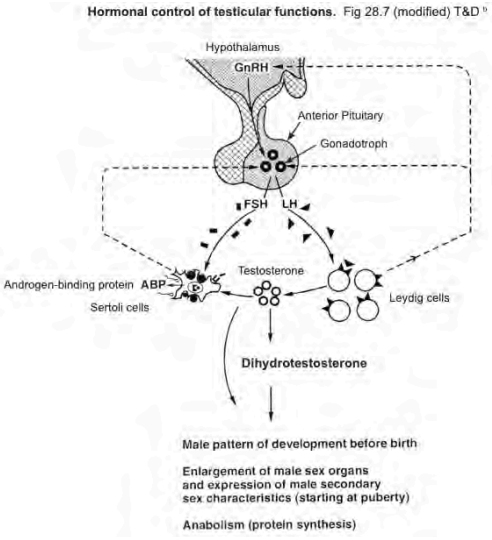


Androgens cause: Aggressiveness, libido, hair growth, baldness

–ve feedback control:

- Testosterone increase causes GnRH levels to decrease.
- T also feeds directly into the gonadotrophs in the ant. pituitary downregulating the production of FSH and LH.
- To inhibit LH and FSH separately (bc they have diff functions), when sertoli cells are stimulated, they produce (alongside ABP) a protein hormone called ***Inhibin*** which feeds back to the FSH producing gonadotrophs in the pituitary.



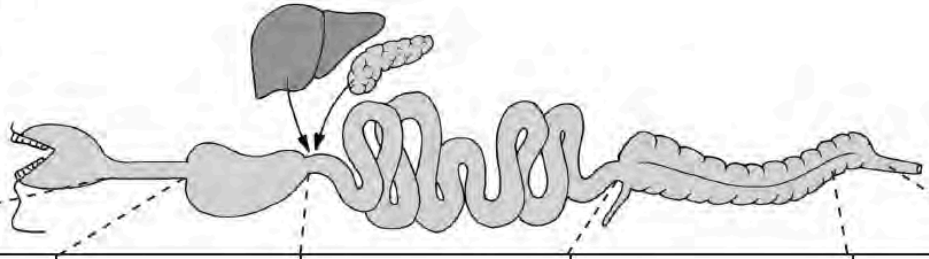


We know that a peptide hormone, ***kisspeptin*** , sits above GnRH in the hypothalamus and the cascade of control of reproduction, but we don't know what controls the production of kisspeptin.

Male Infertility

- Male infertility can have many causes.
- A common feature of infertile men is a reduced sperm count <20 million/ml
  - ***Oligospermia***
  - Or even no sperm ***azoospermia***
  - Or the sperm may be immotile –can't swim.

Summary: Regional variations



Mucosa Epithelium Lamina Propria Muscularis mucosae					
Submucosa					
Muscularis externa					
Serosa / adventitia					
Microvilli					
Modifications involving the mucosa					
Modifications involving the submucosa					
Gross convolutions					